EXHIBIT H

Inquiry into the convictions of Kathleen Megan Folbigg

Forensic Pathology Tender Bundle

TAB	DOCUMENT	DATE
	CALEB	
1.	Neonatal record	5/02/89
	POST-DEATH DOCUMENTS	
2.	Death certificate	20/02/89
3.	Report of death to Coroner	20/02/89
4.	Ambulance report Q006 by Allen Reed	20/02/89
5.	Interim post-mortem report by Dr Royal Cummings	20/02/89
6.	Autopsy report by Dr Royal Cummings	9/05/89
7.	Statement of Allen Reed, ambulance officer	1/09/99
8.	Statement of David Hopkins, paramedic, including: • Ambulance report Q005 dated 29/02/89	1/10/99
9.	Statement of Richard Baines, ambulance officer	29/10/99
10.	Microscopic examination of tissues by Dr Allan Cala	25/11/99
11.	Statement by Dr Barry Springthorpe, including: • Doctor's notes dated 2/02/89 – 5/02/89; • Letter dated 17/02/89; and • Letter dated 21/03/89	6/12/99

TAB	DOCUMENT	DATE
	PATRICK	
12.	Birth certificate	3/06/90
13.	Neonatal record	7/06/90
14.	Letter from Dr Ian Wilkinson to Dr Robert Morris	30/10/90
15.	Letter from Dr Ian Wilkinson to Dr Christopher Marley	30/11/90
	POST-DEATH DOCUMENTS	
16.	Death certificate	13/02/91
17.	Autopsy report by Dr Jan Bishop and Dr Gurpreet Singh-Khaira	14/02/91
18.	Letter from Dr Ian Wilkinson to Dr Christopher Marley	21/02/91
19.	Letter from Dr Alex Kan to Dr Jan Bishop and Dr Gurpreet Singh-Khaira	24/06/91
20.	Final autopsy report by Dr Jan Bishop and Dr Gurpreet Singh-Khaira	2/09/91
21.	Statement of Kathleen Coyle, ambulance officer, including: • Sketch plan of 36 Rawson Street, Mayfield; and • 2 x ambulance reports dated 13/02/91	6/09/99
22.	Statement of Murray Hetherington, ambulance officer, including: • 2 x ambulance reports dated 13/02/91	6/09/99
23.	Statement of Anthony Mullins, ambulance officer	1/10/99
24.	Statement of Dr Ian Wilkinson	8/10/99
25.	Statement of Dr Christopher Walker, including: • Contemporaneous notes dated 13/02/91	18/01/00
26.	Statement of Dr Man Kit Lai, radiologist, including: • CT Brain scan dated 23/10/90 • CT Brain scan dated 05/11/90	11/02/00

TAB	DOCUMENT	DATE
27.	Statement of Dr Joseph Dezordi, including: • Interim discharge letter by Dr Christopher Marley dated 29/10/90; and • History, examination and progress notes by Dr David Cooper dated 18/10/90	17/03/00
28.	Statement by Dr Ian Wilkinson, including: • Death certificate	24/03/00
	SARAH	
29.	Birth certificate	14/10/92
30.	Perinatal database	17/10/92
	POST-DEATH DOCUMENTS	
31.	Death certificate	30/08/93
32.	Report of death to Coroner	30/08/93
33.	Ambulance report Q001 by Louise Bishop, Robert Foxford and Rodney Avery	30/08/93
33A.	Microbiology report	13/09/93
33B.	Microbiology report	21/09/93
34.	Final autopsy report by Dr John Hilton, including: • Microscopic and macroscopic examination by Dr Roger Pamphlett	25/11/93
35.	Statement of Dr Christopher Marley	9/03/99
36.	Statement of Deborah Ann Martin, ambulance officer, including: • Ambulance report Q604 dated 30/08/93	8/10/99
	LAURA	
37.	ECG tracing	Undated
38.	ECG tracing	Undated
39.	EEG tracing	Undated

TAB	DOCUMENT	DATE
40.	ECG tracing	Undated
41.	Birth certificate	7/08/97
42.	NSW midwives data collection	11/08/97
	POST-DEATH DOCUMENTS	
43.	SIDS death scene investigation checklist	Undated
44.	Immunisation notes	3/02/98
45.	Pathologist's data sheet of Dr Allan Cala	1/03/99
46.	Report of death to Coroner	1/03/99
47.	Interim autopsy report by Dr Allan Cala	1/03/99
47A.	Microbiology report	3/03/99
47B.	Microbiology report	4/03/99
47C.	Microbiology report	9/03/99
47D.	Microbiology report	9/03/99
48.	Statement of Dr John Cash, medical practitioner	9/03/99
49.	Statement of Dr Paul Innis, general medical practitioner	15/03/99
50.	Statement by Louise Alderson, ambulance officer	1/09/99
51.	Statement of Brian Wadsworth, ambulance officer, including: • Ambulance Report V70724 dated 1/03/99 • ECG report dated 1/03/99	15/09/99
52.	Statement of Harold Picton, ambulance officer	15/09/99
53.	Statement of Dr Christopher Seton, paediatrician, including: • Sleep study report by Dr Christopher Seton conducted on 2/10/19, dated 7/10/97; • Sleep study report by Dr Christopher Seton conducted on 3/02/98, dated 17/02/98;	23/11/99

TAB	DOCUMENT	DATE
	• Typed letter from Craig Folbigg to Kathleen Folbigg dated 18/03/98; and • Letter from Dr Christopher Seton to DSC Bernard Ryan dated 1/02/00	
54.	Final autopsy report by Dr Allan Cala	13/12/99
55.	Neuropathology report by Dr Michael Rodriguez	13/12/99
56.	Statement of Dr Allan Cala	28/03/03
MULTIPLE CHILDREN		
57.	Statement of Dr Ian Wilkinson	12/03/99
58.	Letter from Dr Allan Cala to DSC Bernard Ryan	29/06/99
59.	Statement of Dr David Cooper, paediatrician, including: • Sleep study dated 15/06/90; • 2 x EEG reports by Dr J T Holland dated 18/10/90 and 5/11/90; and • Sleep study dated 5/11/92	6/12/99
60.	Statement of Dr Bridget Wilcken, including: • NSW Biochemical Genetic Service Report re Patrick dated 13 February 1991; • Letter from Dr Alison Colley to Dr Bridget Wilcken re Patrick and Caleb Folbigg dated 4 December 1991; • Letter from Dr Bridget Wilcken to Dr Alison Colley re Patrick and Caleb Folbigg dated 10 December 1991; and • Newborn Screening Programme Reports re Caleb, Patrick, Sarah and Laura Folbigg dated 13 January 2001	14/01/00
61.	Letter from Dr Allan Cala to DSC Bernard Ryan	19/06/01
EXPERT REPORTS		
62.	Statement of Dr Susan Beal	8/12/99
63.	Statement of Dr Janice Ophoven	6/10/00
64.	Report of Professor Peter Berry	11/00

TAB	DOCUMENT	DATE
65.	Report of Dr Janice Ophoven	1/12/01
66.	Statement of Professor Peter Herdson	17/01/02
67.	Report of Professor Roger Byard	18/10/02
68.	Statement of Dr Robert Ouvrier, including: • J E Constantinou et al, 'Hypoxic-Ischaemic Encephalopathy after Near Miss Sudden Infant Death Syndrome' (1989) 64 *Archives of Disease in Childhood* 703 • R Meadow, 'Suffocation, Recurrent Apnea, and Sudden Infant Death' (1990) 117 *Journal of Pediatrics* 351	28/10/02
69.	Report of Professor Anthony Busuttil	6/11/02
70.	Report of Dr John Christodoulou	18/02/03
71.	Statement of Dr Richard Hawker	6/03/03
72.	Report of Dr Janice Ophoven	27/03/03
73.	Report of Professor Roger Byard	14/04/03
74.	Report of Dr Owen Jones	15/04/03
75.	Supplementary Report of Professor Peter Berry	29/04/03

GREATER NEWCASTLE AREA HEALTH SERVICE
NEWCASTLE WESTERN SUBURBS HOSPITAL

NEONATAL RECORD

PLEASE USE GUMMED LABEL IF AVAILABLE 39999

SURNAME	UNIT NUMB 16
OTHER NAMES	HEET N
ADDRESS FOLEGG	PT'S ADD
DATE OF BIRTH	M.O.

Obstetrician: Dr Davidson

Family Doctor:

Ward: LAYFIELD 2336 DAVIDSON

16-05-07

36 GAISON ST

"Caleb"

DELIVERY DETAILS:

Parity: 6 P0 40/40

Maternal blood group: O p o s Antibodies: Nil

Liquor: mec → pink Duration of ruptured membranes: 10hrs

Duration of labour - 1st stage: 10hrs 10mins 2nd stage: 1 hr - 35m

Time of delivery: 2315 am/pm Date of delivery: 1-2-89

Pain relief and time - ☐ narcotics ....; ☑ epidural 1430; ☐ caudal ....; ☐ G.A. ....

F.H.S. - ☐ normal; ☑ tachycardia during second stage; ☐ decel. - type Variable; ☐ other ....

Delivery complications - ☐ cord; ☐ shoulder dystocia; ☐ other ....

Placental weight: 780 gm. Abnormalities: slight infarction

Complications during labour: Keilania Rotation + delivery.

Maternal temp 37.5

NEONATAL HISTORY:

Sex - ☑ male; ☐ female; ☐ indeterminate

Birth weight: 3220 gm. Length: 42.2 cm. H.C.: 33 cm.

Resuscitation - ☑ aspiration ☐ oxygen ☐ bag and mask ☐ tracheal intubation ☐ mec. asp. from trachea ☐ drugs: ....

Time of initiation of normal respiration: ....

Apgar:

	Heart rate	Resp. effort	Muscle tone	Reflex irritability	Colour	
1 min.:	2	2	2	2	1	Total: 9
5 mins.:	2	2	2	2	1	Total: 9

Cord blood collected - ☑ No; ☐ Yes - tested for: ....

Cord vessels: (number) 3

Konakion 1mg. given by: KRobertson Checked by: Mchurin.

Feeding intention - ☑ breast; ☐ artificial formula: ....

Comments: Developed nasal flaring → slight grunting resp. Transferred to nursery planned in cot 2 Hx nasal mec

Signature: KRobertson (Labour Ward Sister)

DISCHARGE SUMMARY:

Feeding - ☐ fully breast fed ☐ breast fed and comp. ☑ artificial feeding

Cord - ☑ on; ☐ off

Weight: 3180 gm

Guthrie Test - date: 5-2-89

H.C.: 33 cm.

Date of discharge: 5.2.89

Comments: ....

Signature: N. Urquhart (Sister)

36.

1-1-87

NEONATAL RECORD
INITIAL MEDICAL EXAMINATION:

Date of birth ..... Time of birth ..... Assessment of gestational age ..... wks

Age at examination .....hours/days Small for dates: Yes ☐ No ☐

Activity: ..... Moulding/Caput: Yes ☐ No ☐

NEWBORN HEALTH EXAMINATION

SURNAME: FOLBIGG FIRST NAME Caleb SEX: Male/Female

DOB: 1-2-89 MEDICAL RECORD No: .....

NEWBORN BLOOD SCREENING TESTS: Guthrie TAKEN: 5-1-89 (Date)

APGAR AT 1 MIN 9 5 MINS 9 TYPE OF DELIVERY: NORMAL ☐ BREECH ☐

FORCEPS ✓ CAESAREAN ☐

VACUUM EXTRACTION ☐ OTHER ☐

NOTE: Write in figures for weight, length and head circumference.
For other items:

✓ for Normal ? for Recheck X Requires or Receiving Treatment

	1/2	FINDINGS 5/2		FINDINGS	
WEIGHT (kg)	3.580	3180	SKIN	✓ bruise R temple	
LENGTH (cm)	42.5		ABDOMEN	✓	
HEAD CIRCUMFERENCE (cm)	35	34.7	UMBILICUS	✓	
EYES	R –	L –	ANUS	✓	
EARS	R –	L ✓	GENITALIA	NO	
MOUTH	✓		TESTES	R ✓	L ✓
FONTANELLES	–		HIPS	R –	L –
COLOUR	– ++ jaundice				
CVS: HEART	✓		REFLEXES – MORO	✓ ✓	
FEMORAL PULSES	R ✓	L ✓	SPINE	✓	
CHEST	✓		LIMBS – UPPER	R ✓	L ✓
			LOWER	R ✓	L ✓

EVALUATION: FT @ infant, Kolland forceps, initial RDS associated c transient tachypnea. mucus plug jaundiced - Home 5/2 Review 7/52 please

EXAMINER'S SIGNATURE: [signature]

DESIGNATION: Paediatrician

DATE OF EXAMINATION: 5/V 89

*IF A HOSPITAL COPY IS REQUIRED, PLEASE PHOTOSTAT

5

Abnormalities: .....

Consultation: Yes ☐ No ☐ Paediatrician: ..... Other: .....

Comment: .....

DISCHARGE EXAMINATION:

Date: ..... Age at examination ..... days/weeks

Infection	Jaundice	Hernia	Normal	Abnormal
☐ Eyes	☐ No	☐ No	☐	☐ Head & Fontanelle
☐ Nails	☐ Yes	☐ Yes - Specify	☐	☐ Respiratory System
☐ Skin		.....	☐	☐ Heart sounds & murmurs
☐ Mouth			☐	☐ Femoral pulses
☐ Cord	Head Circumference: ..... cm		☐	☐ Abdomen
☐ Other - Specify			☐	☐ Hips

Summary: .....

.....

M.O. Signature: .....

NEW SOUTH WALES

BIRTHS, DEATHS AND MARRIAGES REGISTRATION ACT 1995

DEATH CERTIFICATE

REGISTRATION NUMBER
101757/1989

1 DECEASED	Family Name	FOLBIGG
	Christian or Given Name(s)	Caleb Gibson
	Date of Death	20 February 1989
	Place of Death	Mayfield (36 Rawson St.)
	Sex and Age	Male 2 weeks
	Place of Birth	Waratah, NSW
	Period of Residence in Australia	Life
	Place of Residence	36 Rawson Street Mayfield
	Usual Occupation	-
	Marital Status at Date of Death	-
2 MARRIAGE(S)	Place of Marriage	
	Age when Married	
	Full Name of Spouse	
3 CHILDREN	In order of birth names and ages	
4 PARENTS	Father's Name	Craig Gibson FOLBIGG
	Mother's Name	Kathleen Meagan
	Mother's Maiden Family Name	MARLBOROUGH
5 MEDICAL	Cause of Death and Duration of last illness	Sudden Infant Death Syndrome Inquest dispensed with at Newcastle
	Name of Certifying Medical Practitioner or Coroner	C.A. Elliott, Coroner
6 BURIAL or CREMATION	Date	22 February 1989
	Place	Newcastle Crematorium
7 INFORMANT	Name	C.G. Folbigg
	Address	36 Rawson Street Mayfield
	Relationship to deceased	Father
8 REGISTERING AUTHORITY	Name	V.M. Bennett, Principal Registrar
	Date	01 March 1989
9 ENDORSEMENT(S)		Not any

Before accepting copies, sight unaltered original. The original has a coloured background.

REGISTRY OF BIRTHS
DEATHS AND MARRIAGES

SYDNEY **18 Jan 2000**

I hereby certify that this is a true copy of particulars recorded in a Register in the State of New South Wales, in the Commonwealth of Australia

Registrar

P. 79A 35/89

RECEIVED
20 FEB 1989
Court House
NEWCASTLE

REPORT OF DEATH TO CORONER

Mayfield Police Station
20 February, 1989.

The Coroner,
NEWCASTLE

SUBJECT: Death of FOLBIGG, Caleb Gibson Age 19days.
Marital state N/A Address 36 Rawson Street, Mayfield.
Time and date of death: 2.53am, 20.2.89.
Place of death: 36 Rawson Street, Mayfield.
By whom found: Mother Address: 36 Rawson Street, Mayfield.
By whom reported to Police: Mother Address: 36 Rawson Street, Mayfield.
By whom last seen alive: Mother Address: 36 Rawson Street, Mayfield.
When last seen alive: 1am, 20.2.89
Deceased a native of (County and District): Australia. NSW.
Occupation N/A
(If pensioner state type and include whether appropriate authorities informed)
If Military or Invalid pensioner, state disability:
Name and address of nearest relative and relationship: Mother and Father 36 Rawson Street, Mayfiel
Name and address of identifying person: Craig GIBSON, Father, 36 Rawson Street, Mayfield.
Police present when deceased identified: Sen Const BRYANT and Sgt Ryan.
Did deceased leave a will? N/A
By whom burial or cremation is being arranged: Not Known.
Property and clothing found on and with the deceased. (Attach inventory if space insufficient):
Yellow Jump Suit and white blanket
Miscellaneous Property Book Reference:
How property and clothing disposed of and on whose authority: Property (clothing) handed to Father on Identification.
Circumstances under which death took place. (If any previous illness, and deceased seen by doctor, particulars should be given. Where treated by a doctor a note should be obtained giving particulars of treatment from such doctor):

About 1am on Monday the 20 February, 1989, the child was fed by his mother. He was then put to bed in adjoining bedroom. About 2.53am (20.2.89) the mother awaoke and checked the child and found him to be cold and apparently dead, she found a small amount of blood and thrôth around the childs mouth. The father was alerted and began CPR, till Ambulance Officers arrived, but to no avail. The body was conveyed to the Newcastle Hospitaâ where life was pronounced extinct at 4am, by Dr Sandy CHAPMAN.

The child had been taken to a Dr Springthorpe, of Watt street, Newcastle for treatment for a ' Laxy Larynx'. No medication was given.

Signature: K.J.Bryant
Rank: Sen Const
Annual leave from April, March 1989. to

(Continued overleaf)

NOTE:
(1) This form should be prepared in quadruplicate in all cases where a death is reported to the Coroner. The original and two copies should be forwarded to the Coroner. All statements in duplicate should be lodged with the Coroner at least 7 days before the date of the inquest.
(2) The full name and address of all persons and the registered number of all motor vehicles concerned should be indicated.

DEPARTMENT OF HEALTH, N.S.W.
AMBULANCE SERVICE

GRADE 1 2 3 4 5
REGION HR

AMBULANCE REPORT

DATE	JULIAN	CASE No.	PATIENT	SEX	AGE
20/2/89	051	Q [illegible] 6	1 OF 1	M	20/5

CALL TYPE	AUTHORITY	STN No.	RADIO No.	RESPONSE	CATEGORY
MS	PUBLIC CALL	Q91	A102	2	C1

TIME BOOKED	TIME OUT	LOCATION	DEPART	DESTINATION	CLEAR
	2555	303	3		3440

PATIENT DETAILS

CODE	TITLE	SURNAME	FIRST NAME	D.O.B.
		FOLBIGG.		

ADDRESS | CODE

STRETCHER S
WALKING W
W CHAIR C ☐

☐ W COMP ☐ 3rd PARTY **DEBTOR DETAILS**

CHG CODE	AMOUNT	CHG. KM	CODE	TITLE	SURNAME/BUSINESS	INITIALS
2		10	E899			

PENSION No. / WARRANT No | ADDRESS | CODE

OCCUPATION | EMPLOYED BY

		FROM	CODE	TO	CODE
ODO IN	87 938	36 RAWSON			
ODO OUT	87 928	MAYFIELD.	2304		E899
TRIP KM	10				

HISTORY (State chief complaint first)

CODE H – Infant 20 days old found deceased in cot. ECG monitor showing asystole.

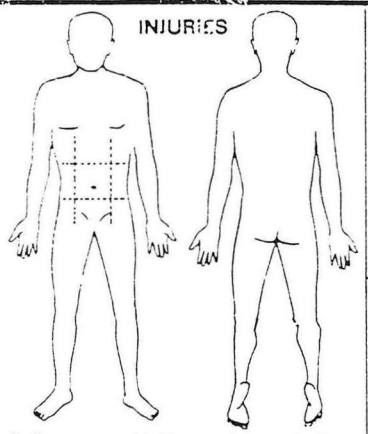
PRONOUNCED

EXAMINATION

AIRWAY	☑ Clear	☐ Obstructed	
BREATHING	☐ Present	☑ Absent	
	☐ Shallow	☐ Deep	
CIRCULATION	☐ Present	☑ Absent	
Buccal Mucosa	☐ Pink	☑ Blue	☐ Pale
Skin temp.	☐ Normal	☑ Cold	☐ Hot
Sweating	☑ Nil	☐ Mod.	☐ Profuse
Vomiting	☑ Nil	☐ Small	☐ Large
Fitting	☑ Nil	☐ Yes	Number
Burns	☑ Nil	☐ Superficial	%
		☐ Deep	%
Blood Loss (External)	☑ Nil	☐ Under 500ml	☐ Over 500ml

Site .....

INJURIES



F Fracture H Haemorrhage B Burn
L Laceration T Tenderness P Pain
A Abrasion C Contusion
S Swelling D Dislocation

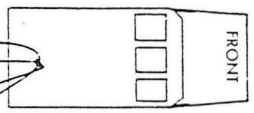
Specific observations ☐ Nil

PREPARED BY: JH

CHECKED BY: BS

Breath Sounds .....

× Position of Patient
→ Point of Impact
▨ Damaged Area



Sent Belt Not / Worn

Estimated Impact Speed High Medium Low

ROAD USER: 1 Driver 2 Passenger 3 Motorcyclist & Pillions 4 Pedal Cyclist 5 Pedestrian 6 Others

	Time	Pulse	Blood Pressure	Capillary Filling	Respiration Rate	Respiration Effort	L Level of Cons.	Total Score

Pupils	Size T	Size R	Size L	React. R	React. L

GLASGOW COMA SCALE	Eye Opening	Motor Response	Verbal Response	S Score

Vital Signs Points			Add Vital Sign Points
	First		+ + + + =
	Final		+ + + + =
			Difference Between Total Score =

Eye Opening:	Best Motor Response	Verbal Response:
4 Spontaneous	6 Obeys	5 Orientated
3 To Speech	5 Localises	4 Confused Conversation
2 To Pain	4 Withdraws	3 Inappropriate Words
1 NIL	3 Abnormal Flexion	2 Incomprehensible Sounds
	2 Extends	1 NIL
	1 NIL	

TABLE: 14=15=5 | 11=13=4 | 8=10=3 | 5=7=2 | 3=4=1

9 FEB 1989

OFFICE COP

TREATMENT BEFORE ARRIVAL
0 Nil 1 CPR within 3 mins.
2 First Aid 3 Medical

Patient's state prior to arrival
A Conscious C Unknown
b Unconscious Mins. .....

PREVIOUS ILLNESS
☐ Nil ☐ Renal
☐ Unknown ☐ Stroke
☐ Hypertension ☐ Epilepsy
☐ Cardiac ☐ Psychiatric
☐ Respiratory ☐ G.I.T.
☐ Diabetes
☐ Other .....

Allergies ☐ Nil ☐ Unknown

Present Medication ☐ Nil ☐ Unknown

Comments .....

TREATMENT

AIRWAY 1 Suction
1 Oral Airway
2 Nasal Airway
☐ ETT

BREATHING 1 RM
2 RS
3 ..... (other)
1 IPPV

CIRCULATION 1 Cardiac Compression

POSTURE 1 Lateral
2 Supine
3 Sitting
4 Legs Elevated

O_2 THERAPY 1 High Conc. Mask
2 Nasal Prongs
3 28% Ventimask
1 14L 2 8L 3 4L
1 Demand Valve

HAEMORRHAGE CONTROL ☐ Dressing
☐ Elevation
☐ Pressure

IV CANNULATION 1 Type .....
2 Unsuccessful
3 In Situ
Local given ☐ No ☐ Yes

INTUBATION 1 Type .....
2 Unsuccessful
3 In Situ

ANALGESIA
E Entonox 1 Not Effective
N Narcotic 2 Part Effective
3 Effective

SPLINTS
1 Hare 5 KED
2 Thomas 6 Other
3 Air 7 Donway
4 Cervical Collar

MAST Suit - Inflated
1 Under 500ml 2 Over 500ml
(External blood loss)

TOURNIQUETS
1 Rotating
2 Arterial
3 Lymphatic
Time On .....

STRETCHER
1 Carry Chair 4 Carry Sheet
2 Back Board 5 Other
3 Jordon Frame

KITS
1 Poison
2 Burns
3 Maternity
4 Other

BLOOD SAMPLE TAKEN
Glucose Est. .....
Blood Type &
Cross Match .....

TYPENEX No.

ATTACH ECG STRIPS HERE

TRANSTAT	
	BYSTANDER
	Effect
	ROAD USER
	Severity
	TREATMENT before arrival
	Conscious / Unconsc.
	SUCTION
	AIRWAY
	BREATHING
	IPPV
	CIRCULATION
	POSTURE
	O_2 THERAPY
	Litre
	DEMAND VALVE
	CANNULATION
	INTUBATION
	ANALGESIA
	Effects
	SPLINTS
	SPLINTS
	MAST SUIT
	TOURNIQUETS
	STRETCHER
	KITS
	ECG
	ECG
	ECG
	D.C. SHOCK
	FLUID / DRUG
	Units
	FLUID / DRUG
	Units
	FLUID / DRUG
	Units
	FLUID / DRUG
	Units
	FLUID / DRUG
	Units
	PROTOCOL
	PROTOCOL
	PROTOCOL
	PROTOCOL
	RESCUE
	COMPLICATIONS
	HAZARDS
	TENS PNEUM RELIEF
	COMMENTS
	MEDICAL REVIEW

RHYTHMS MONITORED
(Abbreviations in heavy print)

1 Sinus Rhythm
2 Sinus Bradycardia
3 Sinus Tachycardia
4 Prem. Atrial Cont.
5 SupraVent. Tachy
6 Atrial Fib / Flut.
7 Idio Vent. Rhythm
8 Asystole
9 Pacemaker Rhythm
10 Prem. Vent. Cont.
Frequency .....
Runs ☐
R on T ☐
Multifocal ☐
11 Vent. Tachycardia
12 Vent. Fibriilation
13 1° Heart Block
14 2° Heart Block
15 3° Heart Block
16 Junctional
17 Multiple Arrhythmia
18 Elect. Mech. Dissn.

TIME	ECG	TREATMENT	DOSE	ROUTE	SHOCK	RESULT	AUTH.

COMMENTS, MEDICAL ASSISTANCE, PROBLEMS, INSTRUCTIONS, ETC.

See Case 005

Dr's Signature .....

Condition at Destination ☐ Improved ☐ Unchanged ☐ Deteriorated

Officer 1 Driving	PRINT NAMES	Officer 2
K. DOHERTY		A REED
	SIGNATURES & EMPLOYEE No's	
	2009	

Copy

DIVISION OF FORENSIC MEDICINE
Newcastle, N.S.W.

INTERIM POST MORTEM REPORT

To: Coroner, Court House, NEWCASTLE

From: Dr. R. CUMMINGS

Name: CALEB GIBSON FOLBIGG

PM No: 89037

Address: 36 Rawson Street,
MAYFIELD

Provisional Cause of Death

Ia SUDDEN INFANT DEATH SYNDROME

Ib

Ic

II

Note: This is a provisional report only and subject to alteration in final report.

Signed _____
Forensic Pathologist

Date: February 20, 1989

NAME FOLBIGG, Caleb Gibson AGE 19 DAYS SEX M PM NO. 89037

PM Date 20.2.89 Time 11.45 Died 20.2.89 Approx. TOD 2am

Build avg. Height 55 cm Weight 3970 kg

Eyes blue Hair Brown Scars Tattoos

Wounds

Evidence of Medical Treatment

ETT ☐ NGT ☐ IDC ☐ ECG PADS ☐ IV ☐

Surgical Wounds Surgical Drains

Heart (325) Sac RV LV

Valves TV PV AV MV

Chambers

Myocardium

Great vessels

Coronary Arteries RCA LAD LCA

Chest R.Cav L.Cav Med.

Sternum Ribs (R) (L)

Lungs (L 34 R 53) Airways

L Lung R Lung

Moist

Mouth Teeth/Dentures Oesophagus

Stomach Appendix

Bowel Peritoneum

Liver (178)

Gallbladder Bile Ducts Pancreas

Spleen (15) Lymphnodes Thymus (19)

Kidneys (L 21 R 21) Capsule Ureters Bladder

Uterus Tubes Ovaries

Prostate Testes

Skull ✓ Neck

Brain (465)

B/A ☐ TOX ☑ Stomach, liver CO ☐ MICRO ☐ CHEM ☐

HISTO ☑ NEURO ☐ POLAROID ☐ SLIDES ☐ POLICE ☐

SUMMARY SIDS.

PERSONS PRESENT

P Lucey

D Kemp

COD

CORONERS ACT, 1980

Medical report upon the examination of the dead body of:—

Name: Caleb Gibson FOLBIGG ..... 89037 sg

I, Royal CUMMINGS, a legally qualified medical practitioner, carrying on my profession at the Regional Forensic Pathology (City Morgue), Newcastle, in the state of New South Wales, do hereby certify as follows:—

1. At 11.45 in the fore noon, on the 20th day of February, 19 89 at Newcastle in the said State, I made an internal examination of the dead body of a male infant identified to my by Peter DUCEY of C/- City Morgue, Newcastle in the State aforesaid, as that of Caleb Gibson FOLBIGG aged about 19 days years.

2. I opened the three cavities of the body.

3. Upon such examination, I found:

EXTERNAL EXAMINATION

The body was that of a well nourished and normally developed male child appearing the stated age of 19 days. Height 55cm (crown/heel), weight 3970g. Hair was brown, eyes were blue. Rigor mortis was present and post mortem staining was seen over the back. There were no external signs of injury and the infant appeared to have been well cared for.

INTERNAL EXAMINATION

Cardio-vascular System

Pericardial sac was normal. The heart (25g) was of normal size and was normally developed. Valves and chambers were normal. Myocardium showed no evidence of disease. Great vessels were normal.

Respiratory System

Pleural cavities were normal. Larynx, trachea and bronchi were unremarkable. The lungs (L:34g R:53g) were somewhat mottled on their pleural surfaces. Cut surfaces were somewhat moist but otherwise unremarkable.

(For continuation — see over)

4. In my opinion, death had taken place about less than one day previously and the cause of death was:

i DIRECT CAUSE:— Disease or condition directly leading to death ..... (a) SUDDEN INFANT DEATH SYNDROME (due to or following)

ANTECEDENT CAUSES:— Morbid conditions, if any, giving rise to the above cause, stating the underlying condition last ..... (b) ..... (due to or following) (c) .....

ii Other significant conditions contributing to the death but not relating to the disease or condition causing it .....

TO THE CORONER

NEWCASTLE

(Signature) .....

(Date) May 9, 1989

Post-mortem carried out on Caleb Gibson FOLBIGG aged 19 days
At 11.45 in the forenoon on the 20th day of February, 1989.

Alimentary System
Tongue, pharynx and oesophagus were normal. The stomach contained a large quantity of curdled milk. Small and large bowel and appendix were normal. Peritoneal cavity was normal. The liver (178g) was healthy. Gallbladder, bile ducts and pancreas were normal.

Haemopoietic System
The spleen (15g) was healthy. There were no enlarged lymph nodes. The thymus (19g) showed no unusual features.

Genito-urinary System
The kidneys (21g) each were normal. Ureters and bladder were normal.

Endocrine System
Pituitary , thyroid and adrenal glands were normal.

Central Nervous System
The skull, cranial cavity and meninges were normal. The brain (465g) showed no unusual features on its meningeal and sectioned surfaces.

HISTOLOGY

HEART	Normal
LUNGS	Are congested and in places show incomplete aeration, in other sections their alveoli contain extravasated red blood cells and a small amount of eosinophillic exudate.
LIVER	Normal.
KIDNEY	Normal.
SPLEEN	Normal.
THYMUS	Normal.
ADRENAL GLAND	Normal.
LYMPH NODES	Normal.

SUMMARY OF AUTOPSY FINDINGS
Both lungs were moderately moist
No other unusual features were seen

R. CUMMINGS MD, FRCPA
Regional Forensic Pathologist
May 9, 1989

... contd. page 3

Post-mortem carried out on Caleb Gibson FOLBIGG aged 19 days years.
At 11.45 in the forenoon on the 20th day of February, 1989.

Samples of liver and stomach and contents have been forwarded to the Division of Analytical Laboratories for toxicological examination.

TOXICOLOGY REPORT

Routine screening tests for poisons were negative.

COMMENT
The gross and microscopic findings are consistent with a "cot death".

R. CUMMINGS MD, FRCPA
Regional Forensic Pathologist
May 9, 1989

STATEMENT in the matter of:
Death of FOLBIGG children

Place: Morisset Police Station
Date : 1 September 1999

Name: Allen Albert REED
Address: Lot 95 Off Main Road, Mulbring **Tel. No.:** 02 49380107
Occupation: Retired **STATES:-**

1. This statement made by me accurately sets out the evidence which I would be prepared, if necessary, to give in court as a witness. The statement is true to the best of my knowledge and belief and I make it knowing that, if it is tendered in evidence, I shall be liable for prosecution if I have wilfully stated in it anything which I know to be false, or do not believe to be true.

2. I am 64 years of age.

3. I am currently living with my wife at our Mulbring home. I have been retired for approximately eighteen months to two years. Prior to that, I was a paramedic with the Ambulance Service of New South Wales. I held the qualifications of a Paramedic for the final nineteen years of my career, performing duty in the Newcastle area both on the road and with the Rescue Helicopter.

4. On Wednesday the 1st of September, 1999 I spoke with Detective RYAN at the Morisset Police Station. Detective RYAN showed me copies of two Ambulance Report Sheets, numbers 005 and 006 completed on the 20th of February, 1989. I read these two documents and recognised that the writing on case number 006 was my handwriting. The incident referred to was one that I attended when I was working as a paramedic at Hamilton Ambulance Station.

5. I do not recall that incident because it was back in 1989, but from reading the document I can state the following: at 3.03am

Witness: _____ **Signature:** A Reed

on the 20th of February, 1989 Paramedic Ron DOHERTY and I attended a premises at 36 Rawson Street, Mayfield to assist Ambulance Officers Richard BAINES and Dave HOPKINS who were in the process of treating a baby who was not breathing. On arrival, either Ron or I applied an ECG monitor to the baby which recorded Asystole - meaning that baby was deceased. The external examination showed that the baby had blue Buccal Mucosa which was a blue/purple colouring around the outside of the mouth. I recorded the skin temperature as being cold to touch, the airway was clear and obviously no breathing and no circulation were present. Ron and I did not find any sweating, vomiting, fitting, burns or blood loss.

Witness: _____ **Signature:** _____

B. Rowe
D/S/C.
1.9.99

New South Wales Police P.190
v2.8

STATEMENT in the matter of:
Death of FOLBIGG children

Place: Hamilton
Police Station
Date : 1 October 1999

Name: David William HOPKINS
Address: 16 Terrence St, Adamstown Heights **Tel. No.:** 02 49573641
Occupation: Paramedic **STATES:-**

1. This statement made by me accurately sets out the evidence which I would be prepared, if necessary, to give in court as a witness. The statement is true to the best of my knowledge and belief and I make it knowing that, if it is tendered in evidence, I shall be liable to prosecution if I have wilfully stated anything which I know to be false or do not believe to be true.

2. I am 42 years of age.

3. I am a qualified Ambulance Paramedic (Level 5) with the New South Wales Ambulance Service. I have been a paramedic for the past seven years and an ambulance officer for the past fourteen years.

4. At 9.30am on Friday the 1st of October 1999 I spoke with Detective RYAN at the Hamilton Police Station. He showed me an ambulance report case number Q005. I read this document and recognised that I had completed it back in 1989 and it related to an incident with I attended with Ambulance Officer Richard BAINES. I refreshed my memory from the document however I still remember attending that job.

5. In the morning of 20 February 1989 I was working as a Level 2 Ambulance Officer at Hamilton Ambulance Station. I was rostered on with Dick BAINES who was the Paramedic. Ron DOHERTY

Witness: _____ **Signature:** _____

and Alan REED who are also Paramedics were working at Hamilton that morning in another vehicle.

6. At 2.55am that morning Dick and I responded to a call to attend 36 Rawson Street, Mayfield for a baby who was not breathing. We arrived at the house at 2.59am that morning. I can't remember what the house looked like or what exactly happened when we got there. I remember walking inside the house and seeing a young man and young woman who appeared upset. Dick either went into a room and gathered the baby or the woman brought the baby to Dick and I in a room which appeared to be a loungeroom. I saw a very young baby dressed in light clothing. It was laid down on the loungeroom floor in the supine position near the entrance to the house. I removed the baby's upper clothing and established that the patient was in a state of cardiac arrest, ie that is unconscious, not breathing and pulseless. I noted that the patient was warm to touch, was pale around the mouth and lips. Dick cleared the patient's airway. I have noted in my report that the patient's airway was obstructed and I cannot remember exactly what obstructed the airway. I assume that there may have been saliva or fluid in the airway which needed to be cleared prior Dick inserting a Guedel's airway. I cannot categorically state what obstructed the airway, maybe Dick can clarify this.

7. A short time later, REED and DOHERTY arrived at the scene and assisted Dick and myself. I was performing ECM - External Cardiac Massage on the patient. REED or DOHERTY attached the ECG monitor to the patient and obtained an ASYSTOLE reading which meant that the patient was deceased. At this time the young man and woman appeared totally distraught, they were both crying. I remember seeing an older woman there but I don't know

who she was.

8. We continued resuscitation attempts until it became clear that a successful outcome was not possible. Dick and I then attempted to placate the parents of the deceased baby. I can't remember the parents telling me anything about the circumstances surrounding the baby's death but I would have asked how the baby was found.

9. I don't remember the police arriving at the house that morning. I do remember placing the baby back in a white bassinette in a bedroom of the house. I also encouraged the young woman who I presumed was the mother of the baby to hold the baby. Dick and I walked outside the house and I completed the ambulance report. I have indicated Sudden Infant Death Syndrome on the report, however this was something I presumed at the time.

EXHIBIT: I NOW SEEK TO PRODUCE REPORT Q005.

I did not see or hear anything that night that raised any suspicion towards the parents of the baby. At 3.38am that morning Dick and I left the house and continued other duties.

10. On 18 October 1990 I was performing duty as a Level 3 Ambulance Officer at the Hamilton Ambulance Station with Ambulance Officer Lance YORKE. (Lance is now deceased) At 4.31am that day, Lance and I responded to a call to attend 36 Rawson Street, Mayfield for a child suffering respiratory distress. Lance and I arrived at the house 4.41am that day. I remember walking to the side entrance of the house where we were met by the same woman I saw at that same house in 1989. She was

holding a small baby in her arms. The baby was wrapped in a blanket and the woman was crying. She said words to the effect of, "My baby is having trouble breathing and won't wake up properly."

11. We asked the lady to step back into the house so we could quickly examine the baby. I remember the baby's upper clothing was moved to expose the chest and I saw the baby appeared to be having respiratory difficulties. It was pale around the face and listless. The patient exhibited tracheal tug and intercostal recession. Lance and I decided to immediately transport the baby to the Mater Hospital which we did. We left the house with the baby being nursed by the mother on the stretcher. I applied oxygen therapy to the baby en route to the hospital. We arrived at the hospital at 4.52am that morning and the baby was treated by hospital staff.

12. At the hospital that morning Lance commenced the Ambulance Report case number Q007. I completed the remainder and majority of the report.

EXHIBIT: I NOW SEEK TO PRODUCE REPORT Q007.

Witness: _____ **Signature:** _____

B. Ryan
D/S/C
1-10-99.

DEPARTMENT OF HEALTH, N.S.W.
AMBULANCE SERVICE
GRADE 1 2 3 4 5
REGION H/R

AMBULANCE REPORT

DATE	JULIAN	CASE No.	PATIENT	SEX	AGE
20/2/89	0.5/9	0095	1 of 1	M	19/52

CALL TYPE	AUTHORITY	STN. No.	RADIO No.	RESPONSE	CATEGORY
A.	CONTROL	0911	A28	1	1

TIME BOOKED	TIME OUT	LOCATION	DEPART	DESTINATION	CLEAR
	0255	0259	0338	0348	0434

3c

PATIENT DETAILS

CODE	TITLE	SURNAME	FIRST NAME	D.O.B.
1	LATE	FOLBIGG.	CALEB.	

ADDRESS: 36 RAWSON ST. MAYFIELD. CODE: 2304

STRETCHER S / WALKING W / W/CHAIR C: S

☐ W/COMP ☐ 3rd PARTY **DEBTOR DETAILS**

CHG. CODE	AMOUNT	CHG. KM	CODE	TITLE	SURNAME/BUSINESS	INITIALS
A		13		1	Folbigg	

PENSION No. / WARRANT No.:
ADDRESS: 36 Rawson St, Mayfield CODE: 2304
OCCUPATION:
EMPLOYED BY:

ODO IN	23054	FROM: RES.	TO: ROYAL NEWCASTLE Hospital
ODO OUT	23035	CODE: 2304	CODE: 0215
TRIP KM	19		

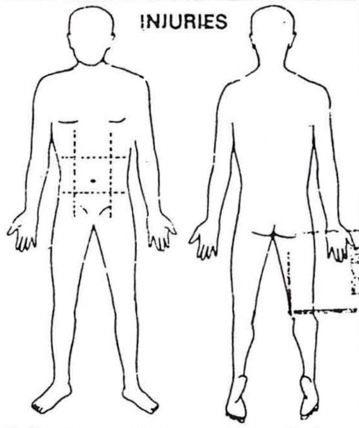
HISTORY (State chief complaint first) SUDDEN INFANT DEATH SYNDROME
O/A. O/E. PT PALE, WARM TO TOUCH BUT NOT BREATHING
RESUSITATION WAS COMMENCED – BUT TO NO AVAIL
PARAMEDICS IN ATTENDANCE

EXAMINATION

AIRWAY	☐ Clear	☑ Obstructed	
BREATHING	☐ Present	☑ Absent	
	☐ Shallow	☐ Deep	
CIRCULATION	☐ Present	☑ Absent	
Buccal Mucosa	☐ Pink	☐ Blue	☑ Pale
Skin temp.	☑ Normal	☐ Cold	☐ Hot
Sweating	☑ Nil	☐ Mod.	☐ Profuse
Vomiting	☑ Nil	☐ Small	☐ Large
Fitting	☑ Nil	☐ Yes	Number
Burns	☑ Nil	☐ Superficial %	
		☐ Deep %	
Blood Loss (External)	☑ Nil	☐ Under 500ml	☐ Over 500ml

Site ....

INJURIES



F Fracture H Haemorrhage B Burn
L Laceration T Tenderness P Pain
A Abrasion C Contusion
S Swelling D Dislocation

Specific observations ☐ Nil
PREPARED BY: DM
CHECKED BY: BS
Breath Sounds

X Position of Patient
→ Point of Impact
▨ Damaged Area
FRONT
Seat Belt Not / Worn
Estimated Impact Speed High Medium Low
ROAD USER: 1 Driver 2 Passenger 3 Motorcyclist & Pillions 4 Pedal Cyclist 5 Pedestrian 6 Others

Time	Pulse	Blood Pressure	Capillary Filling	Respiration Rate	Respiration Effort	Level of Cons.	Total Score	Pupils Size T	R	L	React. R	L	GCS Eye Opening	Motor Response	Verbal Response	Score
0259	0	0	0	0	0	1							1	1	1	3
0308	0	0	0	0	0	1							1	1	1	3

Vital Signs Points		Add Vital Sign Points
First	0	0 + 0 + 0 + 0 + 1 = 1
Final	0	0 + 0 + 0 + 0 + 1 = 1
Difference Between Total Score		± 0

Eye Opening: 4 Spontaneous 3 To Speech 2 To Pain 1 NIL
Best Motor Response: 6 Obeys 5 Localises 4 Withdraws 3 Abnormal Flexion 2 Extends 1 NIL
Verbal Response: 5 Orientated 4 Confused Conversation 3 Inappropriate Words 2 Incomprehensible Sounds 1 NIL

TABLE: 14⇒15 = 5 | 11⇒13 = 4 | 8⇒10 = 3 | 5⇒7 = 2 | 3⇒4 = 1

OFFICE COPY

20 FEB 1989

TREATMENT BEFORE ARRIVAL
0 Nil 1 CPR within 3 mins.
2 First Aid 3 Medical

Patient's state prior to arrival
A Conscious C Unknown
B Unconscious Mins. ....

PREVIOUS ILLNESS
☐ Nil ☐ Renal
☐ Unknown ☐ Stroke
☐ Hypertension ☐ Epilepsy
☐ Cardiac ☐ Psychiatric
☐ Respiratory ☐ G.I.T.
☐ Diabetes
☐ Other ...

Allergies ☐ Nil ☐ Unknown

Present Medication ☐ Nil ☐ Unknown

Comments ....

TREATMENT

AIRWAY 1 Suction
1 Oral Airway
2 Nasal Airway
☐ ETT

BREATHING 1 RM
2 RS
3 EAR (other)
1 IPPV

CIRCULATION 1 Cardiac Compression

POSTURE 1 Lateral
2 Supine
3 Sitting
4 Legs Elevated

O_2 THERAPY 1 High Conc. Mask
2 Nasal Prongs
3 28% Ventimask
1 14L 2 8L 3 4L
1 Demand Valve

HAEMORRHAGE CONTROL ☐ Dressing
☐ Elevation
☐ Pressure

IV CANNULATION 1 Type ....
2 Unsuccessful
3 In Situ
Local given ☐ No ☐ Yes

INTUBATION 1 Type ....
2 Unsuccessful
3 In Situ

ANALGESIA
E Entonox 1 Not Effective
N Narcotic 2 Part Effective
3 Effective

SPLINTS
1 Hare 5 KED
2 Thomas 6 Other
3 Air 7 Donway
4 Cervical Collar

MAST Suit - Inflated
1 Under 500ml 2 Over 500ml
(External blood loss)

TOURNIQUETS
1 Rotating
2 Arterial
3 Lymphatic
Time On ....

STRETCHER
1 Carry Chair 4 Carry Sheet
2 Back Board 5 Other
3 Jordon Frame

KITS
1 Poison
2 Burns
3 Maternity
4 Other

BLOOD SAMPLE TAKEN
Glucose Est. ....
Blood Type & Cross Match ....

TYPENEX No.

ATTACH ECG STRIPS HERE

TRANSTAT

4	BYSTANDER
5	Effect
0	ROAD USER
0	Severity
2	TREATMENT before arrival
6	Conscious/Unconsc.
1	SUCTION
1	AIRWAY
3	BREATHING
1	IPPV
1	CIRCULATION
2	POSTURE
1	O_2 THERAPY
3	Litre
0	DEMAND VALVE
	CANNULATION
	INTUBATION
	ANALGESIA
	Effects
	SPLINTS
	SPLINTS
0	MAST SUIT
0	TOURNIQUETS
5	STRETCHER
0	KITS
	ECG
	ECG
	ECG
	D.C. SHOCK
	FLUID/DRUG
	Units
	FLUID/DRUG
	Units
	FLUID/DRUG
	Units
	FLUID/DRUG
	Units
	FLUID/DRUG
	Units
	PROTOCOL
	PROTOCOL
	PROTOCOL
	PROTOCOL
	RESCUE
	COMPLICATIONS
	HAZARDS
0	TENS PNEUM RELIEF
1	COMMENTS
0	MEDICAL REVIEW

RHYTHMS MONITORED
(Abbreviations in heavy print)
1 Sinus Rhythm
2 Sinus Bradycardia
3 Sinus Tachycardia
4 Prem. Atrial Cont.
5 SupraVent. Tachy
6 Atrial Fib/Flut.
7 IdioVent. Rhythm
8 Asystole
9 Pacemaker Rhythm ☐
10 Prem. Vent. Cont.
Frequency ....
Runs ☐
R on T ☐
Multifocal ☐
11 Vent. Tachycardia
12 Vent. Fibrillation
13 1° Heart Block
14 2° Heart Block
15 3° Heart Block
16 Junctional
17 Multiple Arrhythmia
18 Elect. Mech. Dissn. ☐

TIME	ECG	TREATMENT	DOSE	ROUTE	SHOCK	RESULT	AUTH.
	1						

CHECKED
CASE SHEET
DETAIL CHECKED & FOUND CORRECT
O.I.C. — STATION OFFICER

COMMENTS, MEDICAL ASSISTANCE, PROBLEMS, INSTRUCTIONS, ETC.

Dr's Signature ....

Condition at Destination ☐ Improved ☐ Unchanged ☐ Deteriorated

Officer 1 Driving PRINT NAMES Officer 2
...AINES D. HOPKINS
SIGNATURES & EMPLOYEE No.'s

MOORE ... print carbonless paper (patented) JR 179766 8

New South Wales Police

STATEMENT in matter of: Caleb Gibson FOLBIGG

Place: Newcastle Police Station

Date: 29 October, 1999

Name: BAINES, Richard Anthony **Tel No.:** 49754530

Address: 14 Rothley Gardens, Balmoral

Occupation: Businessman

States:-

1. This statement made by me accurately sets out the evidence which I would be prepared, if necessary, to give in court as a witness. The statement is true to the best of my knowledge and belief and I make it knowing that, if it is tendered in evidence, I shall be liable to prosecution if I have wilfully stated in it anything which I know to be false or do not believe to be true.

2. My age is 51 years. (Born 7.10.48)

3. I was employed by the New South Wales Ambulance Services between 1974 and 1997. I was employed as a paramedic ambulance officer. My training included specialist medical training, including rescue duties. My duties included the emergency pre hospital responses so therefore I usually did not perform general ambulance duties though could be called on to do so at any time. I would often assist various general duty units at various times.

4. At this time because of the amount of cases that I have attended in the years I worked as an ambulance officer I am unable to recall many of those incidents. On Friday 29 October I attended Newcastle Police Station where I spoke to Detective senior Constable Bradley who told me something. He showed me some case report forms dated 29.2.89. I have reviewed those documents.

5. From reviewing those records I am able to say a case report

Witness: _____ Signature: _____

has been completed by Dave HOPKINS. He has listed my name on the bottom of that case report. He was therefore the treatment person and filled out the case report and I would have been the driver at that time. We were manning a general duties unit at that time. I was not working as a paramedic at that time though I was qualified to do so. I resumed those duties some time later. I am able to say another case report dated 20.2.89 relates to another ambulance unit which was despatched some three minutes later from the same station. Both units were despatched from Hamilton or the code Q911 which is written on both forms. The crew of the other unit was Ron DOHERTY, who was the paramedic station officer and driver at that time and also Allan REED who was the treatment person.

6. I am able to say that we left the station at Hamilton at 2.53am and arrived at 36 Rawson Street, Mayfield at 2.59am. I am also able to say the backup unit arrived at address at 3.03am. I am able to say that the patient was a baby named Caleb FOLBIGG. I am also able to say that the baby was pale, not breathing and warm to touch upon examination. resuscitation was commenced but was unsuccessful. I am also able to say that an airway was cleared with oral suction using the oxygen apparatus. A Guedels airway was placed in the babies mouth. At some stage mouth to mouth resuscitation was commenced and that would have been when we initially attended prior to getting the oxygen equipment on line. When the oxy viva was on line or prepared for use it was used with a Robert Shaw valve and mask. We started IPPB or intermittent positive pressure ventilation. We have then commenced cardiac compression with the baby lying on it's back. The resuscitation was continued until the paramedic arrived. An E.C.G or electro cardiograph monitor was used by the second unit and the patient was found to be suffering in asystole or the electro cardiograph was in straight line. Therefore the patients heart had ceased to function.

Witness: _____ Signature: _____

7. I am unable to say the length of time that the resuscitation was undertaken from reviewing those records but relying on the fact that we departed the scene at 3.38am I would estimate that the efforts to resuscitate the child were reasonable prolonged. I am also able to say there was no pulse, that the blood pressure was nil. That indicated that the baby was in cardiac and respiratory arrest.

8. It would appear that we had found a lifeless baby and obviously the child did not respond to basic life support. I am also able to say that it would appear the infant had suffered no apparent observable external injury. At that time I had attended many hundred of cases and possibly thirty of those cases involved infants.

Witness: _____ Signature: _____
R. BAINES

Witness: _____ Signature: _____

COPY

NSW INSTITUTE OF
FORENSIC MEDICINE

42-50 PARRAMATTA ROAD
PO BOX 90
GLEBE NSW 2037
PHONE (02) 9660 5977
FAX (02) 9552 1613

MICROSCOPIC EXAMINATION OF TISSUES:

Name: Caleb Gibson FOLBIGG

Institute Case No: 99/11208 (89/037)

Heart: Normal.

Lungs: Congestive changes are present with focal areas of haemorrhage present within some alveolar spaces.

Spleen: Normal.

Kidney: Normal.

Adrenal: Normal.

Thymus: Normal.

Liver: Normal.

Lymph node: Normal.

A D Cala MBBS Dip RACOG FRCPA

Forensic Pathologist
NSW Institute of Forensic Medicine

25 November, 1999

EXPERT CERTIFICATE in the matter of: Death of Caleb FOLBIGG
Police -v-
Place: 10/28 Watt Street, Newcastle Date: 6.12.99

Name: Barry John SPRINGTHORPE
Address: 10/28 Watt Street, Newcastle Tel.No: 49297844
Occupation: Consultant Paediatrician STATES:-

EXPERT CERTIFICATE
Section 177, Evidence Act 1995 No. 25

1. This statement made by me accurately sets out the evidence which I would be prepared, if necessary, to give in court as a witness. The statement is true to the best of my knowledge and belief and I make it knowing that, if it is tendered in evidence, I shall be liable to prosecution if I have wilfully stated anything which I know to be false or do not believe to be true.

2. I am 62 years of age.

3. I hereby certify:
My full name is **Barry John SPRINGTHORPE**
My contact address is **10/28 Watt Street, Newcastle**
I have a specialised knowledge based on the following training, study and experience:-
I am a Consultant Paediatrician having completed MBBS at Sydney University in 1960. I worked in general practice in Australia, the United Kingdom and Canada until 1972 before returning to the Children's Hospital; in Sydney to pursue the FRACP. I established the Child Development Unit in Newcastle in 1976 - emphasis on developmental problems, SIDS and child abuse. As part of this role I established the SCAN (Suspected Child Abuse and Neglect) Group which coordinated the efforts of the Health, Welfare and Police in suspected child abuse cases. I have published material in relation to child abuse. I have been in private general paediatric practice for the past twenty years.

See Continuation Sheet ...

Witness: _____
LINDA B. FOGGO

Signature: _____

4. About 1.45pm on the 2nd of February 1989 I examined Caleb FOLBIGG at the Western Suburbs Hospital in Newcastle. Caleb was then about fourteen hours old and had developed respiratory distress which required some oxygen through the night. This is quite a common occurrence in new born children. A chest x-ray was performed and was judged to be clear. Caleb's condition improved over the next two days and was discharged apparently well on the 5th of February 1989.

5. I have perused the medical notes in my possession regarding Caleb and in those notes I have recorded that Caleb's father expressed concern at Caleb's noisy breathing during feeding, but I could detect no abnormality to explain those concerns.

EXHIBIT: SEE TYPED DOCTOR'S NOTES

6. I reviewed Caleb on the 17th of February 1989 at two weeks of age and noted that there was still a mild inspiratory stridor with a little recession which was most marked when he was upset or lying flat on his back. This condition is common and usually resolves spontaneously with time. I then generated a letter that date to Doctor DAVIDSON the referring obstetrician and a copy to Dr LEEDER the general practitioner.

EXHIBIT: SEE LETTER DATED 17.2.89.

7. In the morning of the 20th of February 1989, I received a telephone call from Mrs Kathleen FOLBIGG at my surgery. She told me that the baby had been found dead in the cot about 3am that morning having been put down around 1am and failing to respond to efforts at resuscitation. After this conversation I made handwritten notes at the bottom of the letter dated 17.2.99.

8. On the 21st of March 1989 I interviewed Mr and Mrs FOLBIGG at my

rooms in Newcastle after receiving the autopsy report.

EXHIBIT: SEE LETTER DATED 21.3.89.

5. Based wholly or substantially on the above knowledge, I am of the opinion that Caleb had undoubtedly congenital laryngeal stridor (almost certainly on the basis of laryngomalacia) there was nothing abnormal in the way of laryngeal webs or cysts to be found at autopsy and the cause of his demise remains a mystery.

Witness: _____ Signature: _____

LINDA B. FOGGO

WSH: 2.2.89 1.45pm -1-

CALEB FOLBIDD

Of Kathleen. 36 Rawson Street Mayfield. PRIVATE SHARED. Obstet Davidson. Primip. Born at 11.15pm last night by Keillands weighed 3280 grams HC noted as 33 but I make it 35 cms, apgars 9 at 1 and 9 at 5, no resuscitation needed but developed nasal flaring and a grunt, needed oxygen during the night, is now fine, chest x-ray is good , he is an essentially healthy boy and there is no intention of circumcision . Mum intending to breastfeed. 23 grandchildren of her husbands side and he is the 5th on her side.

WSH: 3.2.89 8.45am -1-

CALEB FOLBIGG

Still a touch tachypnoeic but otherwise OK, bruise laterally over his right eye, mum has decided to abandon breastfeeding, he is a touch jaundiced as well but should prove no problems.

WSH: 11am 4.2.89

BABY FOLBIGG

is a touch more jaundiced but tachypnoea settled down and feeding strongly - hopefully home Tues, Wed. next wk.

CALEB FOLBIGG

WSH: 5.2.89

Is ready to go today, weight 3180 grams, down 100 grams no tachypnoea but father is concerned at noisy resps during feeding but he looks fine. Home today F/U 2 weeks.

DR. B.J. SPRINGTHORPE
Consultant Paediatrician
10/28 Watt St, Newcastle 2300
Ph: (02) 4929 7844
Fax: (02) 4926 3078

DR. BARRY J. SPRINGTHORPE CONSULTANT PAEDIATRICIAN
M.B., B.S. (Syd.), D.C.H. (Lond.), F.R.A.C.P. (Paed.)

P/N 118262L

B. J. SPRINGTHORPE PTY. LTD.
SUITE 10, 3rd FLOOR, ATHCOURT,
28 WATT STREET, NEWCASTLE,
N.S.W. 2300.
PHONE: (049) 26-3144

BJS:AG

17th February, 1989

Dr. R. Davidson
Maitland Road
MAYFIELD. 2304

re: Caleb FOLBIGG b.d. 1.2.89
36 Rawson Street, Mayfield
Parents: Kathleen and Craig

Dear Bob,

This 2 week old infant is now doing well after his initial respiratory distress.

As you know he is the first child of a healthy couple. There is no significant family history (Craig has 23 nieces and nephews all of whom are said to be fighting fit!).

He was delivered by Keillands at fullterm, weighed 3280gms, had good Apgars but rapidly developed a grunt and tachypnoea which gradually subsided over 48hrs.

A chest X-ray was essentially normal though there was just a suggestion of minimal pneumomediastinum. He had a touch of jaundice and there remains a mild inspiratory stridor with a little recession which is most marked when he is upset or lying supine.

He is on Enfalac feeds, is growing on or about the 50th centile for all parameters and there has been no associated cyanosis or gagging with his stridor.

The rest of the examination was quite normal. I feel sure that this is just a simple mild laryngomalacia and should resolve with time.

No further investigations are warranted at this stage.

Could I review his progress in another 2 months all being well.

Kind regards

Barry Springthorpe

c.c. Dr. D. Leeder
King Street
Newcastle.

P.S. 20.2.89 Distraught call from Kathleen this am
Baby found dead in cot at 3am
(put down apparently well at 1am)
→ Failed to respond to resuscitation
SIDA (Kim Hill) aware
I'm waiting on call from
Roy Cummings (forensic pathologist)
could I see family
in ~ 3 weeks?
Barry.

DR. BARRY J. SPRINGTHORPE CONSULTANT PAEDIATRICIAN
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PHONE: (049) 26-3144

BJS:AG

21st March, 1989

Dr. D. Leeder
King Street
NEWCASTLE. 2300

re: Caleb FOLBIGG b.d. 1.2.89
36 Rawson Street, Mayfield
Parents: Kathleen and Craig

Dear Dorothy,

I saw Kathleen and Craig in my rooms today as a follow up to their loss of young Caleb with apparent SIDS on 20.2.89.

I was able to tell them that though Caleb had undoubted congenital laryngeal stridor (almost certainly on the basis of laryngomalacia) there was nothing abnormal in the way of laryngeal webs or cysts to be found at autopsy and the cause of his demise remains a mystery.

Craig had I know been anxious about the baby's feeding difficulties (because of his stridor) prior to his death and the night he died Craig had wanted to take him into the bed with him. However, both the parents in retrospect were sure that the stridor did not distress Caleb unduly during sleep. Even had we done sleep study I doubt very much that we would have discovered any abnormality that required attention.

They have been contacted by the SIDA group and are going to follow through that contact.

I would be very happy to see them again when they embark on their next pregnancy or if they have any further concerns and obviously we will need to study the new baby thoroughly in due course.

Kind regards

Barry Springthorpe

c.c. Dr. R. Davidson
Maitland Road
Mayfield

NEW SOUTH WALES

BIRTHS, DEATHS AND MARRIAGES REGISTRATION ACT 1995

BIRTH CERTIFICATE

REGISTRATION NUMBER
109740/1990

23

1 CHILD	
Family Name	**FOLBIGG**
Christian or Given Name(s)	**Patrick Allan David**
Sex	**Male**
Date of Birth	**03 June 1990**
Place of Birth	**Waratah (Western Suburbs Hospital)**
2 MOTHER	
Family Name	**FOLBIGG**
Maiden Family Name	**MARLBOROUGH**
Christian or Given Name(s)	**Kathleen Meagan**
Occupation	**-**
Age	**22 years**
Place of Birth	**Sydney, NSW**
3 FATHER	
Family Name	**FOLBIGG**
Christian or Given Name(s)	**Craig Gibson**
Occupation	**Manager**
Age	**28 years**
Place of Birth	**New Lambton, NSW**
4 MARRIAGE OF PARENTS	
Date of Marriage	**05 September 1987**
Place of Marriage	**Newcastle, NSW**
5 PREVIOUS CHILDREN OF RELATIONSHIP	**Caleb G deceased**
6 INFORMANT(S)	
Name	**C. FOLBIGG**
Address	**36 Rawson St Mayfield Father**
7 REGISTERING AUTHORITY	
Name	**V. M. Bennett, Principal Registrar**
Date	**20 June 1990**

8 ENDORSEMENT(S)

Not any

Before accepting copies, sight unaltered original. The original has a coloured background.

REGISTRY OF BIRTHS
DEATHS AND MARRIAGES

SYDNEY **17 Jan 2000**

I hereby certify that this is a true copy of particulars recorded in a Register in the State of New South Wales, in the Commonwealth of Australia

Registrar

30

HUNTER AREA HEALTH SERVICE
NEWCASTLE WESTERN SUBURBS HOSPITAL

PLEASE USE GUMMED LABEL IF AVAILABLE

HSMR12

UNIT NUMBER 27

FOLDING, Kathleen
36 Rawson Street, WAPPING 2304
302

NEONATAL RECORD

Obstetrician: .....

Family Doctor: ..... Ward: GF 17

17

39/40.

DELIVERY DETAILS:

Parity: G2P1 Prev babe died NND 20 days old (cot death)

Maternal blood group: A pos Antibodies: NIL

Liquor: CLEAR Duration of ruptured membranes: > 1/24

Duration of labour - 1st stage: 4 HR 40' 2nd stage: 26'

Time of delivery: 1106 am/~~pm~~ Date of delivery: 03 JUNE '90

Pain relief and time - ☑ narcotics 0940 ☐ epidural .....; ☐ caudal .....; ☐ G.A. .....

F.H.S. - ☑ normal; ☐ tachycardia; ☐ decel. - type .....; ☐ other .....

Delivery complications - ☑ cord; x1 ☐ shoulder dystocia; ☐ other .....

Placental weight: 920 gm. Abnormalities: NIL

Complications during labour: NIL

SVD.

NEONATAL HISTORY:

Sex - ☑ male; ☐ female; ☐ indeterminate

Birth weight: 3410 gm. Length: 48·3 cm. H.C.: 33·5 cm.

Resuscitation - ☑ aspiration ☐ oxygen ☐ bag and mask

☐ tracheal intubation ☐ mec. asp. from trachea ☑ drugs: NEONATAL NARCAN 0.5ML IMI @ 1114.

Time of initiation of normal respiration: 90 SECS

Apgar:

	Heart rate	Resp. effort	Muscle tone	Reflex irritability	Colour	
1 min.:	2	2	1	1	1	Total: 7 @ 1'
5 mins.:	2	2	1	2	1	Total: 8 @ 5'

Cord blood collected - ☑ No; ☐ Yes - tested for: .....

Cord vessels: (number) 3

Konakion 1mg. given by: D/ Reid Checked by: .....

Feeding intention - ☐ breast; ☑ artificial formula: Enfalac.

Comments: HPU, HNPMEC. NAD

Babe to be s/B Paed.

Signature: D/Reid (Reg. SCN) (Labour Ward Sister)

DISCHARGE SUMMARY:

Feeding - ☐ fully breast fed ☐ breast fed and comp. ☑ artificial feeding

Cord - ☑ on; ☐ off

Weight: 3450 gm

Date of discharge: 8/6/90.

Guthrie Test - date: 7-6-90

H.C.: ..... cm.

Comments: .....

Sleep Study 14/6/90 ✓ ∝ - Appt Dr Morris 3/52

Signature: ..... (Sister)

-1-87

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NEONATAL RECORD
INITIAL MEDICAL EXAMINATION:

Date of birth ..... Time of birth ..... Assessment of gestational age ..... wks

Age at examination .....hours/days Small for dates: Yes ☐ No ☐

Activity: ..... Moulding/Caput: Yes ☐ No ☐

NEWBORN HEALTH EXAMINATION

SURNAME: ~~Lochie~~ GOTHRIE FIRST NAME ..... SEX: Male/~~Female~~

DOB: 3.6.90 MEDICAL RECORD No: 221375

NEWBORN BLOOD SCREENING TESTS: ..... TAKEN: 7.6.90 (Date)

APGAR AT 1 MIN 07 5 MINS 08 TYPE OF DELIVERY: NORMAL ✓ BREECH ☐

FORCEPS ☐ CAESAREAN ☐

VACUUM EXTRACTION ☐ OTHER ☐

NOTE: Write in figures for weight, length and head circumference.
For other items:

✓ for Normal ? for Recheck X Requires or Receiving Treatment

	FINDINGS		FINDINGS
WEIGHT (kg)	3410	SKIN	✓
LENGTH (cm)	48.3	ABDOMEN	✓
HEAD CIRCUMFERENCE (cm)	33.5	UMBILICUS	✓
EYES	R ✓ L ✓	ANUS	✓
EARS	R ✓ L ✓	GENITALIA	✓
MOUTH	✓	TESTES	R ✓ L ✓
FONTANELLES	✓	HIPS	R ✓ L ✓
COLOUR	✓		
CVS: HEART	✓	REFLEXES – MORO	✓
FEMORAL PULSES	R ✓ L ✓	SPINE	✓
CHEST	✓	LIMBS – UPPER	R ✓ L ✓
		LOWER	R ✓ L ✓

EVALUATION: Normal Newborn

EXAMINER'S SIGNATURE: [signature]

DESIGNATION: Paediatrician

DATE OF EXAMINATION: 4 / 6 / 90

*IF A HOSPITAL COPY IS REQUIRED, PLEASE PHOTOSTAT

5

Referred to Special Care Nursery: Yes ☐ No ☐ Reason: .....

Abnormalities: .....

Consultation: Yes ☐ No ☐ Paediatrician: ..... Other: .....

Comment: .....

DISCHARGE EXAMINATION:

Date: ..... Age at examination ..... days/weeks

Infection	Jaundice	Hernia	Normal	Abnormal
☐ Eyes	☐ No	☐ No	☐	☐ Head & Fontanelle
☐ Nails	☐ Yes	☐ Yes - Specify	☐	☐ Respiratory System
☐ Skin		.....	☐	☐ Heart sounds & murmurs
☐ Mouth			☐	☐ Femoral pulses
☐ Cord	Head Circumference: ..... cm		☐	☐ Abdomen
☐ Other - Specify			☐	☐ Hips

Summary: .....

.....

M.O. Signature: .....

IW/bw
October 30, 1990.

Dr. R. Morris,
61 Dennison Street,
HAMILTON. 2303

c.c. Dr. G. Cooper
Dr. C. Marley
Dr. W. Carey, Adelaide Children's Hospital

Dear Rob,

Re: Patrick FOLBIGG
20 Rawson Street, MAYFIELD.

Thanks for asking me to see this young man.

The story is someone who presented with what sounded initially like apnoea, but who subsequently in the ward demonstrated that he was clearly having seizures, mainly right sided.

On examination, I could not find any neurological problem.

His tone and deep tendon reflexes are normal, and he appears active and interested. There was some suggestion of the right side not functioning as well as the left, but the signs are not marked.

The history was of great interest, in that he had a male sibling, who died at 20 days of unexplained causes, but who previously had a "floppy larynx".

A serum lactate and ammonia were within normal limits, and I understand at this stage that the urinary organic acids are normal, but the urinary amino acid pattern is still being processed.

The most worrying thing is the CT scan, which shows symmetrical areas of hypodensity in occipital regions posteriorally. These changes certain suggest the possibility of a metabolic disorder, although Herpes encephalitis cannot be ruled out absolutely. He had been febrile at various times in his illness, although he had no white cells in his spinal fluid, and only three red cells.

We have chosen to treat as though he might have Herpes Simplex encephalitis, with Acyclovir, but his fits have been quite resistant to treatment, and currently he is on Phenobarb and Dilantin, and we may need to add Rivotril depending up what levels show.

We are sending blood off to the Adelaide Children's Hospital, to look up the lysosimal enzymes, but also the long chain fatty acids. It is a little bit late for him to present with the commatal form ALD, and he does have reflexes, but there may be some other disorder affecting myelination.

I will be away for 16 days in Japan, and I hope that some of these answers will come back by the time I return.

If the Rivotril is not successful, and if the metabolic studies appear normal, then I wonder wether this young man might be a candidate for a course of A.C.T.H., perhaps in a dose of 20 units a day, to try and pull up these fits.

With kind regards.

Sincerely,

Ian Wilkinson.

Dictated by Dr. Wilkinson,
signed in his absence.

I. A. WILKINSON PTY. LIMITED.
DR. IAN WILKINSON
M.B., B.S. (Qld) F.R.A.C.P.
PAEDIATRIC NEUROLOGIST
Provider No.: 108 142Y

115 ELDER STREET,
LAMBTON. N.S.W. 2299
Telephone: (049) 52 6599

IW/mp
November 30, 1990.

Dr. C. Marley,
C/R King & Perkins Streets,
NEWCASTLE. 2300.

c.c. Dr. R. Morris
Dr. C. Challinor

Dear Chris,

Re: Patrick FOLBIGG
36 Rawson St, Mayfield.
DOB: 3/6/90

Rob Morris kindly asked me to take over Patrick's care after I returned from Japan.

He had further seizures during that time, and one of them was a prolonged one, lasting perhaps an hour, which simply consisted of his eyes being deviated upwards and to the side.

The original CT scan was highly suspicious of abnormalities in the occipital lobes, and we repeated it, and it just confirmed further change. There was some concern about the possibility of a degenerative disease, but John Bear felt that this was probably just vascular. We sent them down to Camperdown Children's Hospital to get another opinion just to be certain, and the opinion was the same as John's. Basically it looks as though there has been some impairment of the blood supply in the basilar territories. I believe the parents had some concern that this might have taken place during that prolonged seizure, but in fact he already had changes in that area at the time of the first presentation.

I am not really quite sure what caused this problem, but all of our tests for degenerative disease have come back negative.

Unfortunately his visual performance has clearly dropped off since presentation, and that is consistent with damage to his visual cortex.

He had further seizures whilst in hospital recently with Rotavious gastroenteritis, and currently his anticonvulsants are Phenobarb in a dose of 36mg. twice a day, and after a serum Tegretol level which had fallen again to only 5, I have increased his Tegretol to 5mls. of the syrup twice a day. He has had no seizures now for about a week as I dictate this, but I am very concerned about this young man's future.

We will allow another coupleof months to pass to see if there is any significant recovery, but after Christmas, I think we should be looking at appropriate therapy for him, which may well include the Royal Blind Society.

With kind regards.

Sincerely,

Ian Wilkinson.

NEW SOUTH WALES

BIRTHS, DEATHS AND MARRIAGES REGISTRATION ACT 1995

DEATH CERTIFICATE

REGISTRATION NUMBER
101831/1991

1 DECEASED	Family Name	FOLBIGG
	Christian or Given Name(s)	PATRICK ALAN DAVID
	Date of Death	13 February 1991
	Place of Death	WARATAH (MATER MISERICORDIAE HOSPITAL)
	Sex and Age	Male 8 months
	Place of Birth	WARATAH, NEW SOUTH WALES
	Period of Residence in Australia	Life
	Place of Residence	36 RAWSON STREET, MAYFIELD 2304
	Usual Occupation	-
	Marital Status at Date of Death	-
2 MARRIAGE(S)	Place of Marriage	
	Age when Married	
	Full Name of Spouse	
3 CHILDREN	In order of birth names and ages	
4 PARENTS	Father's Name	CRAIG GIBSON FOLBIGG
	Mother's Name	KATHLEEN MEGAN
	Mother's Maiden Family Name	MARLBOROUGH
5 MEDICAL	Cause of Death and Duration of last illness	(A) ASPHYXIA DUE TO AIRWAY OBSTRUCTION 1 HOUR (B) EPILEPTIC FITS 4 MONTHS
	Name of Certifying Medical Practitioner or Coroner	Dr. I.A. WILKINSON
6 BURIAL or CREMATION	Date	15 February 1991
	Place	NEWCASTLE CREMATORIUM
7 INFORMANT	Name	C.G. FOLBIGG
	Address	36 RAWSON STREET, MAYFIELD 2304
	Relationship to deceased	FATHER
8 REGISTERING AUTHORITY	Name	V. M. Bennett, Principal Registrar
	Date	06 March 1991
9 ENDORSEMENT(S)		Not any

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Before accepting copies, sight unaltered original. The original has a coloured background.

REGISTRY OF BIRTHS
DEATHS AND MARRIAGES

SYDNEY **17 Jan 2000**

I hereby certify that this is a true copy of particulars recorded in a Register in the State of New South Wales, in the Commonwealth of Australia

Registrar

NEWCASTLE MATER HOSPITAL, WARATAH

PATHOLOGY DEPARTMENT

AUTOPSY PART I

SURNAME:	FOLBIGG
CHRISTIAN NAME:	PATRICK
SEX:	MALE
DATE OF BIRTH:	03.06.1990 (8 MONTHS)
MEDICAL RECORD NO.:	36 03 90
TIME AND DATE OF DEATH:	13.02.1991 - 1040 HOURS
TIME AND DATE OF P.M.:	13.02.1991 - 1230 HOURS
P.M. FILE NO.:	91/7
P.M. CONDUCTED BY:	DR. J. BISHOP / DR. G. SINGH-KHAIRA
MEDICAL OFFICER:	DR. I. WILKINSON / DR. R. MORRIS
REFERRED BY:	DR. C. MARLEY, NEWCASTLE.

CLINICAL DIAGNOSIS

1. ENCEPHALOPATHIC DISORDER LEADING TO INTRACTABLE SEIZURES. THE UNDERLYING CAUSE OF ENCEPHALOPATHY NOT DETERMINED ON INVESTIGATION.
2. ASYSTOLIC CARDIAC ARREST AT HOME LEADING TO DEATH.

MACROSCOPIC DIAGNOSIS

1. NORMALLY FORMED MALE INFANT OF APPROXIMATELY EIGHT MONTHS OF AGE.
2. BRAIN AND SPINAL CORD FIXED FOR LATER DISSECTION.
3. HEPATIC CONGESTION.
4. CONGESTED POSTERO-BASAL DEPENDANT SEGMENTS - BOTH LUNGS.
5. ENLARGED THYMUS.

CLINICAL HISTORY:

Presenting complaint: Brought into MMH casualty at 1020 hours by paramedics on 13.02.1991 after an asystolic cardiac arrest at home.

History of presenting illness: The mother put Patrick to bed at 0730 hours. At 0930-1000 hours she found that the baby was not breathing. The baby arrived in MMH casualty at 1020 hours. ECG monitor showed an asystole. Subsequent resuscitation efforts were unsuccessful and the infant was pronounced dead at 1040 hours.

PAST MEDICAL HISTORY:

The mother suffered from a viral illness at twenty six weeks of pregnancy. However Patrick was delivered at full term on 03.06.1990 through a normal vaginal delivery and experienced no problems during the neonatal period.

He was enlisted on the sleep study programme as his elder brother had died in 1989 at nineteen days of SIDS. All his sleep study tests including EEG were normal.

On 18.10.1990 at five months of age he presented to MMH in an apnoeic floppy state. He was resuscitated and he remained well until he developed (while in hospital) generalised and also right sided focal fits which were associated with a low grade fever. The fits were thought to be secondary to Herpes encephalitis and were treated with Acyclovir and large doses of Phenobarbitone and Phenytoin. Cardiac monitoring normal.

INVESTIGATIONS:

CSF: Biochemical and cytology studies were normal. Herpes culture was negative.

Serum herpes IgM: Normal.

US scan of the brain and kidneys: Normal.

Cranial CT scan: Showed hypodense areas in the temporal and occipital lobes secondary to viral encephalitis ? demyelination disorder.

E.E.G.: Showed left frontal lobe epileptogenic foci.

Chest Xray: Showed features consistent with bronchiolitis.

Naso pharyngeal aspirate: Culture for viruses and viral antigens were negative.

Urine metabolic screen: Was negative for methylmalonic acid. Urinary organic amino acid profile, urinary amino acid pattern showed no abnormality. Urinary lactic acid was within normal range.

Serum lactate, ammonia, calcium, magnesium and glucose: Were all normal.

Rectal biopsy: Showed no neuronal inclusion bodies.

Leucocyte inclusions: Were normal.

Blood metabolic screen: was negative for GM1 and GM2 gangliosidoses, and MLD; Gaucher's Krabbe's and Niemann- Pick diseases; Mannosidosis, Fucosidosis, Mucolipidoses II and III, and Mucopolysaccharidosis VII.

Plasma screen for very long chain fatty acids and Phytanic acid was negative for ALD/AMN, Refsum's disease, Zellweger's and other generalised peroxisomopathies. Page 2

Mucopolysaccharide screen was negative.

Plasma carnitine values were normal.

COURSE OF EVENTS:

The fits were stabilised with anticonvulsants and he was discharged from hospital with a diagnosis of intractable seizures, probably viral encephalitis and bronchiolitis.

The following week he again presented on 04.11.1990 with prolonged seizures resembling an oculogyric crisis which resolved spontaneously after 90 minutes. At the time he also had bilateral conjunctivitis and an URTI. Repeat CT scan showed further decrease in the brain substance.

Repeat EEG showed multifocal epileptogenic foci suggesting a progressive encephalopathic disorder.

He was subsequently discharged on 10.11.1991.

He was again admitted on 14.11.1990 for further investigations. Repeat CT scan showed a ? occipital ischaemic area with clinical visual impairment (probably cortical blindness) and developmental regression. All other investigations including echocardiography were negative and he was discharged.

On 23.12.1990 he was admitted again with an oculogyric crisis secondary to past encephalitic basal ganglia problem which was provoked by a viral illness.

MACROSCOPIC REPORT

EXTERNAL APPEARANCE:

The body was that of a normally formed, well nourished male infant weighing 8.57kg with head circumference 44cm, crown rump length 53cm, crown heel length 77cm and foot length 10cm. Peripheral oedema, signs of trauma and jaundice were absent. Externally no abnormality was present.

CENTRAL NERVOUS SYSTEM:

Bones of the skull: The anterior fontanelle was open and of normal size. The posterior fontanelle was closed.

Meninges: No abnormality detected.

Brain: Weighed 750 grams (normal average weight at this age is approximately 714 grams). The brain was fixed for later dissection.

Spinal cord: No abnormality detected. Spinal cord was fixed for later dissection.

RESPIRATORY SYSTEM:

Pleura, diaphragm and the rib cage: No abnormality detected.

Larynx, trachea and bronchi: All these structures were examined and contained frothy mucoid secretion. No foreign bodies were present. The mucosal linings were normal.

Lungs: Right weighed 55 grams (normal average weight at this age is approximately 52 grams). Left weighed 50 grams (normal average weight at this age is approximately 45 grams). Both lungs were congested in their posterior basal dependant segments. No other abnormality was present. Lung tissue was collected for viral and bacterial cultures.

CARDIOVASCULAR SYSTEM:

Pericardium: No abnormality detected.

Heart: Weighed 49 grams (normal average weight at this age is 44 + 8 grams). The atria, ventricles and the valves were examined and showed no abnormality. The origin of blood vessels from the heart was normal. The atrio-ventricular ring from the heart was kept for further histological studies (if required). Heart tissue was collected for EM and metabolic studies.

Aorta and its branches: No abnormality detected.

Venous system: No abnormality detected.

Lymphatic system: No abnormality detected.

HAEMOPOIETIC SYSTEM:

Thymus: Weighed 30 grams (normal average weight at this age is 10 ± 2 grams). It was enlarged. No other abnormality was present.

Spleen: Weighed 27 grams (normal average weight at this age is 20 grams). No abnormality detected.

Bone marrow: No abnormality detected.

HEPATIC-BILIARY SYSTEM AND PANCREAS:

Liver: Weighed 284 grams (normal average weight at this age is 254 grams). On section it was congested. Liver tissue was collected for EM and metabolic studies.

Gall bladder and bile ducts: No abnormality detected.

Pancreas: Weighed 15 grams (normal average weight at this age is 13 ± 2 grams). No abnormality detected.

Peritoneal cavity: No abnormality detected.

GASTROINTESTINAL SYSTEM:

The entire gastrointestinal tract was normal.

URINARY SYSTEM:

Kidneys: Right weighed 32 grams (normal average weight at this age is 31 grams). Left weighed 33 grams (normal average weight at this age is 31 grams). Both kidneys showed no abnormality. Kidney tissue was collected for EM and metabolic studies.

Ureters and urinary bladder: No abnormality detected. Approximately 10ml of urine was collected for metabolic studies.

ENDOCRINE SYSTEM:

Pituitary gland: No abnormality detected.

Thyroid gland: Weighed 4 grams. No abnormality detected.

Adrenal glands: Both together weighed 6 grams. No abnormality detected.

MUSCULOSKELETAL SYSTEM:

No abnormality detected. Skeletal muscle was collected for histology, EM and metabolic studies.

Skin: No abnormality detected. Sections of skin were collected for chromosome studies and fibroblast cultures.

Blood: Blood was collected for chromosomes, culture, Tegretol and Phenobarbitone levels, and FBC.

INVESTIGATIONS ON POST MORTEM TISSUE:

1. Urine, Tissue: Snap frozen were sent to Judith Hammond at the Oliver Latham Laboratory.

2. Dr. Bale, RAHC was questioned concerning investigation of a cardiac conduction defect.

   As previous ECG monitoring showed no abnormality and arrhythmias were never noted clinically. This was thought to be very unlikely. Page 5

Histological confirmation involves embedding and sectioning of the entire atria ventricular ring. Identification of ectopic conduction bundles in this age group is difficult and depends on histological recognition. Special stains are unhelpful. Tissue kept for subsequent dissection if requested.

3. Tissue for viral studies (blood for viral Ab's and lung tissue) have been sent to the JHH Virology laboratory.

4. Tissue collected for EM has been stored at the Mater in case histological or metabolic studies suggest EM would be useful.

5. Fibroblast cultures will be forwarded by Cytogenetics to Dr. Carey at the Adelaide Children's Hospital.

DR. J. BISHOP / DR. G. SINGH-KHAIRA
14.02.1991

I. A. WILKINSON PTY. LIMITED.
DR. IAN WILKINSON
M.B., B.S. (Qld) F.R.A.C.P.
PAEDIATRIC NEUROLOGIST
Provider No.: 108 142Y

115 ELDER STREET,
LAMBTON. N.S.W. 2299
Telephone: (049) 52 6599

IW/mp
February 21, 1991.

Dr. Chris Marley,
C/R King & Perkins Sts,
NEWCASTLE. 2300.

c.c. Dr. C. Challinor
Oliver Latham Lab.
Ms. J. Dwyer, Mater
Dr. W. Carey, Adelaide Children's
Social Work Dept, Mater

Dear Chris,

Re: Patrick FOLBIGG
36 Rawson St, Mayfield.

This is just a formal letter to follow-up our 'phone call. Patrick died at the Mater Hospital on February 13, 1991.

He had a fever the night before, and his parents wondered about the possibility of his having had a seizure at that time. He apparently slept well enough, seemed happy and played with his father early in the morning, and his mother put him down to sleep at about 7.30 a.m., and discovered him a couple of hours later, quite lifeless.

Although he was still warm when found, and when the paramedics came, I am suspicious that he was already dead at that point.

Certainly we were unable to resuscitate him at the Hospital.

An initial post mortem showed some fairly minor petechial haemorrhages, which really could have been agonal, and were not indicative of a cause for his death.

Further investigations are pending. Fibroblasts are being cultured at the Mater, and frozen urine, and also frozen liver specimens have been sent to the Oliver Latham Laboratory.

Clearly with this death at 8 months, coupled with his brother's death at 20 days, we must consider the possibility of some familial disorder, although the cause for this is not clear at the moment.

The only biochemical label we have ever had on a metabolic condition was an arterial lactate, which was elevated on one occasion at 1.6.

He has already had a workup at the Adelaide Children's Hospital, but I have spoken to Judy Hammond, at the Oliver Latham Laboratory, and she will endeavour to look further for some underlying metabolic disorder.

His brain is being fixed at this moment, but we are discussing where it would be best examined when it is ready in a few weeks time.

I have spoken to his parents on February 18, just indicating that we found no particular cause for his death, but that further tests were proceeding. As is my usual practice, I have asked to sit down with them again at any time, but certainly after the post mortem results are available.

.....2/

Patrick Folbigg cont.

More tragically, although we have had a lot of very grave concerns about this young man's future, it appeared as though he was now starting to make some very good gains.

With kind regards.

Sincerely,

Ian Wilkinson.

Royal Alexandra Hospital for Children
HISTOPATHOLOGY DEPARTMENT

61

WARD	*MRN* *P. No.* C71,635
AMO	*NAME* **FOLBIGG, Patrick**
Died : 13/2/91	*DOB* 3/6/90 *SEX* M *AGE* 8m

Specimen & Site P.M. 91/7 Brain sections

Clinical Diagnosis & Data

Date of Biopsy

Drs. J. Bishop & G. Singh-Khaira,
Anatomical Pathology,
Mater Misericordiae Hospital,
Edith Street,
WARATAH. NSW 2298.

Dear Drs. Bishop & Singh-Khaira,

Re: Patrick FOLBIGG : PM 91/7 M 8mths
Convulsive disorder ? since febrile illness and apnoeic attack at 5 months of age.

I have found no convincing evidence of any neuronal storage disease or any leucodystrophy in these sections. The major changes in this extensively sectioned brain are old infarcts and gliosis mostly in the form of old laminar necrosis which, in keeping with the macroscopic finding, is most severe in the parieto-occipital area. The only spongy change is seen in the gliotic cortical scars and the subjacent white matter, in the old infarcts. The cerebellar cortex is unaffected. We can, therefore, rule out Canavan's disease. In the deeper parts of the cerebrum and in the cerebellar and brain stem nuclei there are neurones showing simple atrophy. They could have resulted from this baby's epileptic seizures. In the leptomeninges there appears to be a light lymphoid infiltrate which is in addition to the small amount of residual haemopoiesis normal in this age group. This could be either non-specific and related to the cortical infarcts or related to the treated encephalitis (? assumed or proven).

I believe that the small amount of linear cortical calcification in the occipital region is just part of the laminar cortical necrosis. I can see no suggestive features of toxoplasmosis or cytomegalovirus infection, and the distribution of the lesions is unusual for herpes simplex encephalitis and they certainly appear far more likely to be the result of the episode of cardio-respiratory arrest this baby suffered at about 5 months of age.

24/6/91 A.E. Kan

ROYAL ALEXANDRA HOSPITAL FOR CHILDREN — CAMPERDOWN

WHH 10/90

HAPS

Hunter Area Pathology Service
A UNIT OF HUNTER AREA HEALTH SERVICE
Division of Anatomical Pathology

M.M.H. AUTOPSY REPORT

Dr J. Bishop
Dr S. Braye
Dr V. Chetty
Prof K. Donald
Dr N. Ferguson
Dr R. Murugasu
Dr A. Price

J.H.H. Ph (049) 214443 Fax (049) 214794
M.M.H. Ph (049) 211220 Fax (049) 602136

Hospital/MRN : Q211/0360390	NAME : FOLBIGG, PATRICK
WARD : MEDICAL RECORDS	ADDRESS : 36 RAWSON STREET
REQUESTING Dr: DR I WILKINSON	MAYFIELD 2304
REQUEST DATE : 13/02/91	D.O.B. : 03-Jun-1990
LABORATORY No: MA9100007	SEX : Male

Specimen : POST MORTEM EXAMINATION
Exams : 0000 MPM LW on 13-Feb-1991 at 12:30

c.c. DR. R. MORRIS
c.c. DR. C. MARLEY, NEWCASTLE

CLINICAL DIAGNOSIS:

* ENCEPHALOPATHIC DISORDER LEADING TO INTRACTABLE SEIZURES. THE UNDERLYING CAUSE OF ENCEPHALOPATHY NOT DETERMINED ON INVESTIGATION.
* ASYSTOLIC CARDIAC ARREST AT HOME LEADING TO DEATH.

FINAL DIAGNOSIS:

* NORMALLY FORMED MALE INFANT OF APPROXIMATELY EIGHT MONTHS OF AGE.
* OLD INFARCTS AND GLIOSIS IN THE PARIETO-OCCIPITAL AREA (BOTH CEREBRAL HEMISPHERES), WHICH ARE PROBABLY SECONDARY TO THE CARDIO-RESPIRATORY SUFFERED AT ABOUT FIVE MONTHS OF AGE.

MACROSCOPIC REPORT:

CENTRAL NERVOUS SYSTEM:

Bones of the skull - The anterior fontanella was open and of normal size. The posterior fontanella was closed.
Meninges - No abnormality detected.
Carotids, vertebrals, basilar arteries and Circle of Willis - No abnormality detected.

Brain - Weighed 750 grams. Grossly the gyri of both occipital lobes (visual cortex) were shrunken, thinner and more undulated than normal and the sulci were widened. The frontal, parietal and the temporal lobes externally showed no macroscopic abnormality. On section the cortical grey matter of the visual cortex in both hemispheres was thinner than normal and showed cystic degeneration. The cysts measured 1-2mm in diameter and were present in a linear pattern at the junction of the grey matter and white matter. The underlying white matter was whiter and firmer than normal and appeared to be expanded. The effected areas in the right and left occipital hemispheres measured approximately 40 x 35 x 35mm and 35 x

continued

35 x 30mm respectively. Similar areas of firm white matter were present in left frontal and both parietal lobes.

The remaining cerebral parenchyma showed no macroscopic abnormality.

The mid-brain, pons, medulla oblongata and the cerebellum were macroscopically normal.
Spinal cord - No abnormality detected.

MICROSCOPIC REPORT:

BRAIN:

Please see the attached microscopic report from Dr. Alex Kan (Camperdown Childrens Hospital, Sydney).

LUNGS:

Sections from both lungs were examined. The lung parenchyma showed no significant abnormality apart from small foci of alveolar collapse in the periphery of the lung. The bronchi, bronchioli and the pulmonary blood vessels were normal. The maturation of the lung tissue was consistent with the stated age of the infant.

SECTIONS from the heart, skeletal muscle, liver, spleen, thymus, pancreas, kidneys, thyroid gland, adrenal glands, testes and the gastrointestinal tract were examined, all showed no abnormality apart from mild P.M. autolysis.

The maturation of the abovementioned tissue and organs was consistent with the stated age of the infant.

Post mortem blood cultures grew mixed cocci and bacilli identified as E.coli, Enterococcus faecolis and Enterococcus avium. These findings are not significant and probably reflect contamination. Post mortem lung tissue cultures were negative for organisms. Post mortem lung tissue cultures for viruses and mycoplasma were negative.

TX2020 M49160
TX2020 M54700
TX2030 M49160
TX2030 M54700

Reported 02.09.1991 - DR. J. BISHOP / DR. G. SINGH-KHAIRA

DR J.BISHOP

New South Wales Police

STATEMENT in matter of: Death of Folbigg children

Place: Kurri Ambulance Stn

Date: 6 September, 1999

Name: COYLE, Kathleen **Tel No.:** 02 49694908

Address: 49 Brunker Rd, Broadmeadow

Occupation: Ambulance Officer

States:-

1. This statement made by me accurately sets out the evidence which I would be prepared, if necessary, to give in court as a witness. The statement is true to the best of my knowledge and belief and I make it knowing that, if it is tendered in evidence, I shall be liable to prosecution if I have wilfully stated in it anything which I know to be false or do not believe to be true.

2. My age is 41 years.

3. I am currently the Station Officer Grade 1 of Kurri Ambulance Station. I have been a member of the New South Wales Ambulance Service since 1988 and currently hold a level four classification.

4. On the 13th of February, 1991 I was attached to the Hamilton Ambulance Station and at that time I was a level two ambulance officer. I was rostered on day shift with Ambulance Officer Russell MULLENS on car A68. Russell was rostered as driver and I was treatment officer. Level four Ambulance Officer Murray HETHERINGTON was also working that day 'one out' in car A36.

5. At 10.03am that morning, Russell and I responded to a call of a baby not breathing at a premises at 36 Rawson Street, Mayfield. We arrived at the house at 10.10am that morning at the same time as Murray. I remember the house was an older type (1920's) weatherboard home with gabbles and a verandah at the

Witness: _____ **Signature:** _____

front. I walked in the front door via the verandah with Murray and Russell. We were carrying our equipment and as we walked through the door we entered a large room/loungeroom. I heard the sound of a woman sobbing and I looked to my right. I saw a woman sitting on a lounge and she was hysterical. She had her hands up to her face and she was crying out and sobbing. I walked past her and into another room on my left side. I saw a man kneeling over a small baby. I think this man was attempting CPR on the child.

EXHIBIT: SKETCH PLAN OF 36 RAWSON STREET, MAYFIELD - NOT TO SCALE.

6. My partners and I knelt over the baby and commenced treatment. I have perused an ambulance report number Q037 which I recognise as a document I completed in relation to this incident. This document has allowed me to refresh my memory of what occurred that day. I do remember the incident quite well.

EXHIBIT: I NOW SEEK TO PRODUCE REPORT Q037.

7. I remember performing the heart compression whilst Murray or Russell performed the ventilation on the baby for a very short time. I checked the baby's vital signs and from my notes it appears he was normal/warm to touch and slightly blue around the lips. I have recorded that I noted present shallow breathing in the examination, however this is contradicted in the respiration rate which is recorded as nil. I cannot explain this discrepancy, however I would never have recorded breathing as being present and shallow if I didn't note it that day at some stage.

8. Whilst treating the baby in the first couple of minutes the man in the house identified himself as the father of the child.

Witness: _____ Signature: _____

He said, "His mother just found him like this."
At some stage he also said, "This is our second lost child."

9. One of the ambulance officers picked up the baby. I don't remember what the baby was wearing but he was in some type of clothing. I ran out to the front yard where the ambulance vehicle was parked and I got into the back with the baby. I continued chest compressions while one of the other officers ventilated. The man who identified himself as being the father got into the front of the ambulance and we left en route to the Mater Hospital. I cannot remember anyone else at the house on this day but I am sure someone was left with the mother.

10. In the ambulance we continued CPR as well as asking the father about the baby. He told me that the baby's name was Patrick and he said, "He's had epilepsy, he's blind and has some genetic problems."
We arrived at the Mater Hospital in Newcastle at 10.18am that day and the baby was admitted to the casualty section. At this time I commenced completing the mentioned ambulance report from information supplied to me by Mr Craig FOLBIGG.

Witness: _____ Signature: _____
D/S/C Ryan
6/9.99

Witness: _____ Signature: _____

Tasha
6/8/09
WARDROBE
Rear
Front door
STEPS
Mum on lounge (sitting)

DEPARTMENT OF HEALTH, N.S.W.
AMBULANCE SERVICE

GRADE 1 2 3 4 5
REGION 4 R

AMBULANCE REPORT

DATE	CASE No.	PATIENT	SEX	AGE
13/2/91	0,0,3,7	1 of 1	M	8 mths

CALL TYPE	AUTHORITY	STN. No.	RADIO No.	RESPONSE	CATEGORY
CA	CONTROL	911	A68	1	C1

TIME BOOKED	TIME OUT	LOCATION	DEPART	DESTINATION	CLEAR
1003	1003	1040	1015	1018	1103

PATIENT DETAILS

CODE	TITLE	SURNAME	FIRST NAME	D.O.B.
B	MASTR	FOLDICA	PATRICK	3-6-90

STRETCHER S
WALKING W
W/CHAIR C [S]

ADDRESS: 36 RAWSON ST, MAYFIELD
CODE: 2,3,0,4

☐ W/COMP ☐ 3rd PARTY DEBTOR DETAILS

CHG. CODE	AMOUNT	CHG. KM	CODE	TITLE	SURNAME/BUSINESS	INITIALS
L		16			CRAIG FOLDICA	

PENSION No. / WARRANT No.: 5868328 Medicare CARD
ADDRESS: [illegible] CODE:
OCCUPATION: Per. Phcall to NIB
EMPLOYER: fil 4/2/91 2x12hr

		FROM	CODE	TO	CODE
ODO IN	416	Reo.	2304	MAYF	2311
ODO OUT	16940				
TRIP KM	16				

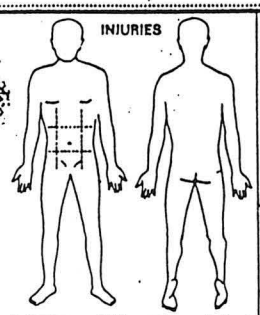
HISTORY (State chief complaint first) CODE 2. DUO) PT IS A 8 MONTH O/O ♂ Fr. HX OF APNOEA EPISODES + EPILEPSY. C/C: DUO CODE 2 TIME? O/A PT ↑ SKIN COLOUR PINK PERIPHERAL CYANOSIS, SKIN TEMP WARM, MIL VITAL SIGNS. PT PH, EPILEPSY. HOD CODE 2 C/O AT HOSPITAL CAR 36, ☒ ON SCENE.

EXAMINATION

AIRWAY	☒ Clear	☐ Obstructed	
BREATHING	☒ Present	☐ Absent	
	☒ Shallow	☐ Deep	
CIRCULATION	☐ Present	☒ Absent	
Buccal Mucosa	☐ Pink	☐ Blue	☒ Pale
Skin temp.	☒ Normal	☐ Cold	☐ Hot
Sweating	☒ Nil	☐ Mod.	☐ Profuse
Vomiting	☒ Nil	☐ Small	☐ Large
Fitting	☒ Nil	☐ Yes	Number
Burns	☒ Nil	☐ Superficial % ☐ Deep %	
Blood Loss (External)	☒ Nil	☐ Under 500ml ☐ Over 500ml	

Site .....

INJURIES



F Fracture L Laceration A Abrasion S Swelling
H Haemorrhage T Tenderness C Contusion D Dislocation
B Burn P Pain

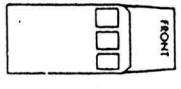
Specific observations ☐ Nil

PREPARED BY KL

CHECKED BY

Breath Sounds .....

× Position of Patient
→ Point of Impact
▨ Damaged Area



Seat Belt Not / Worn
Estimated Impact Speed High Medium Low

ROAD USER: 1 Driver 2 Passenger 3 Motorcyclist & Pillions 4 Pedal Cyclist 5 Pedestrian 6 Others

	Time	Pulse	Blood Pressure	Capillary Filling	Respiration Rate	Respiration Effort	L Level of Cons.	Total Score
	1010	NIL	NIL	NIL	NIL		A	
	1015	NIL	NIL	NIL	NIL		1	
Vital Signs Points	First	NIL	Add Vital Sign Points: NIL + NIL + NIL + NIL + 3 = 3					
	Final	NIL	NIL + NIL + NIL + NIL + 3 = 3					
			Difference Between Total Score ± 3					

Pupils	Size T	Size R	Size L	React. R	React. L	GLASGOW COMA SCALE Eye Opening	Motor Response	Verbal Response	S Score
	0	=	0	2	2	1	1	1	3
	0	=	0	2	2	1	1	1	3

Eye Opening:	Best Motor Response	Verbal Response
4 Spontaneous	6 Obeys	5 Orientated
3 To Speech	5 Localises	4 Confused Conversation
2 To Pain	4 Withdraws	3 Inappropriate Words
1 NIL	3 Abnormal Flexion	2 Incomprehensible Sounds
	2 Extends	1 NIL
	1 NIL	

TABLE: 14-15 = 5 | 11-13 = 4 | 8-10 = 3 | 5-7 = 2 | 3-4 = 1

TREATMENT BEFORE ARRIVAL

0 Nil 1 CPR within 3 mins.
2 First Aid 3 Medical

Patient's state prior to arrival
A Conscious C Unknown
B Unconscious Mins. .....

PREVIOUS ILLNESS
☐ Nil ☐ Renal
☐ Unknown ☐ Stroke
☐ Hypertension ☒ Epilepsy
☐ Cardiac ☐ Psychiatric
☐ Respiratory ☐ G.I.T.
☐ Diabetes
☐ Other .....

Allergies ☐ Nil ☒ Unknown

Present Medication ☐ Nil ☒ Unknown

Comments .....

TREATMENT

AIRWAY 1 Suction, (1) Oral Airway, 2 Nasal Airway, ☐ ETT

BREATHING 1 RM, (2) RS, 3 ..... (other), (4) IPPV

CIRCULATION 1 Cardiac Compression

POSTURE 1 Lateral, (2) Supine, 3 Sitting, 4 Legs Elevated

O_2 THERAPY 1 High Conc. Mask, 2 Nasal Prongs, 3 28% Ventimask, 4 14L 2 8L 3 4L, (1) Demand Valve

HAEMORRHAGE CONTROL ☐ Dressing ☐ Elevation ☐ Pressure

IV CANNULATION 1 Type ..... 2 Unsuccessful 3 In Situ
Local given ☐ No ☐ Yes

INTUBATION 1 Type ..... 2 Unsuccessful 3 In Situ

ANALGESIA E Entonox N Narcotic — 1 Not Effective 2 Part Effective 3 Effective

SPLINTS 1 Hare 2 Thomas 3 Air 4 Cervical Collar 5 KED 6 Other 7 Donway

MAST Suit - Inflated 1 Under 500ml 2 Over 500ml (External blood loss)

TOURNIQUETS 1 Rotating 2 Arterial 3 Lymphatic Time On .....

STRETCHER 1 Carry Chair 2 Back Board 3 Jordon Frame 4 Carry Sheet (5) Other

KITS 1 Poison 2 Burns 3 Maternity 4 Other

BLOOD SAMPLE TAKEN Glucose Est. ..... Blood Type & Cross Match .....

TYPENEX No.

RHYTHMS MONITORED

(Abbreviations in heavy print)

1 Sinus Rhythm
2 Sinus Bradycardia
3 Sinus Tachycardia
4 Prem. Atrial Cont.
5 SupraVent. Tachy
6 Atrial Flb/Flut.
7 IdioVent. Rhythm
8 Asystole
9 Pacemaker Rhythm
10 Prem. Vent. Cont. Frequency ..... Runs ☐ R on T ☐ Multifocal ☐
11 Vent. Tachycardia
12 Vent. Fibrillation
13 1° Heart Block
14 2° Heart Block
15 3° Heart Block
16 Junctional
17 Multiple Arrhythmia
18 Elect. Mech. Diss.

TIME	ECG	TREATMENT	DOSE	ROUTE	SHOCK	RESULT	AUTH.

CHECKED CASE SHEET DETAIL CHECKED & FOUND CORRECT
D.I.C. — STATION OFFICER

COMMENTS, MEDICAL ASSISTANCE, PROBLEMS, INSTRUCTIONS, ETC.

Dr's Signature .....

Condition at Destination ☐ Improved ☐ Unchanged ☐ Deteriorated

PRINT NAMES Officer 1 Driving: R. MILNE Officer 2: R. COLES

SIGNATURES & EMPLOYEE No. 1045

80921-38611

ATTACH ECG STRIPS HE

TRANSTAT

15	BYSTANDER
	Effect
	ROAD USER
	Severity
0	TREATMENT
B	Conscious/Unco
	SUCTION
1	AIRWAY
2	BREATHING
1	IPPV
	CIRCULATION
2	POSTURE
1	O_2 THERAPY
	Litre
0	DEMAND VALVE
	CANNULATION
	INTUBATION
	ANALGESIA
	Effect
	SPLINTS
	SPLINTS
	MAST SUIT
5	TOURNIQUETS
5	STRETCHER
	KITS
	ECG
	ECG
	ECG
	D.C. SHOCK
	FLUID/DRUG
	Units
	FLUID/DRUG
	Units
	FLUID/DRUG
	Units
	FLUID/DRUG
	Units
	FLUID/DRUG
	Units
	PROTOCOL
	PROTOCOL
	PROTOCOL
	PROTOCOL
	RESCUE
	COMPLICATION
0	HAZARDS
0	TENS/PNEUM RE
1	COMMENTS
0	MEDICAL REVIE

OFFICE COPY

52

CASE No. 044 037

CAR No. 68

STATION A

TIME RECEIVED 10 03 13

TIME OUT 10 03 13

ETD ADVISED

SUBURB Maryborough

OFFICERS ON CASE

1

2 o/s. ~~10 10 13~~

3 K. Coyle

4 A. Mullens

5 o/s. 10 15 13

6

7

8

CALLED BY

PHONE

NAME OR LOCATION 36 Rawson St.

CROSS STREET OR LANDMARK

NATURE OF EMERGENCY Baby 8 yrs ? Code 4.

No. OF INJURED

TO Mater Hosp.

	Paramedic response	Yes
	Breathing difficulty	
	Chest pain	
	Severe bleeding	
	Severe injury/burns	
	Fitting	
	Others	

RESCUE VAN

POLICE

RECEIVED BY B.11

CONTROL OFFICER

New South Wales Police

STATEMENT in matter of: Death of FOLBIGG children

Place: Toronto Ambulance Stn

Date: 06 September, 1999

Name: Murray John HETHERINGTON **Tel No.:** 02 49754380

Address: **41 Alkrington Ave, Fishing Point**

Occupation: **Ambulance Officer**

States:-

1. This statement made by me accurately sets out the evidence which I would be prepared, if necessary, to give in court as a witness. The statement is true to the best of my knowledge and belief and I make it knowing that, if it is tendered in evidence, I shall be liable to prosecution if I have wilfully stated in it anything which I know to be false or do not believe to be true.

2. My age is 40 years.

3. I am stationed at Toronto Ambulance Station and I am certified at level four - advanced life support. I have been an ambulance officer for the past sixteen years with about nine of those years at the level four classification.

4. On the 13th of February, 1991 I was stationed at the Hamilton Ambulance Station. On that day, I was rostered on day work to perform single officer ambulance duty from Hamilton Station in car A36. Ambulance Officers Kathleen COYLE and Russell MULLENS were also rostered on day work on car CA68. I was the most senior officer between the three of us.

5. At 10.02am that day I was despatched to 36 Rawson Street, Mayfield in relation to an eight month old baby boy possibly deceased. I arrived at the house at 10.10am that day exactly the same time as officers Coyle and Mullens. The house was a small weatherboard cottage which appeared clean and tidy. I remember that carpet was clean but there is nothing else about

Witness: _____ Signature: _____

the house that I can remember. On arrival Coyle, Mullens and I walked into the house via the front door and into a loungeroom, then a bedroom to the left of the loungeroom. I saw a man kneeling over a small infant who was laying on the floor of what appeared to be a bedroom. The man appeared to be performing mouth to mouth resuscitation on the baby. I knelt down and examined the baby. As a result, I found that the baby had no pulse and was not breathing, thereby being in a cardiac arrest. There was no question of that. I picked the baby up in my arm and commenced CPR. Due to the close proximity of the Mater Hospital and the absence of Paramedics, I decided to transport the baby immediately to the Mater Hospital. I carried the baby in my arm whilst performing CPR and got into the rear of car A68. Officer COYLE accompanied me in the back of the ambulance and officer Mullens drove. I don't remember the father of the baby being present in the ambulance.

6. We drove straight to the Mater Hospital Casualty Section where the baby was immediately treated by Hospital staff. At the hospital I completed an ambulance report Q036. I have a read a copy of this document and certify it as being correct. On the copy which I read the comments section appears to be illegible due to photocopying. From memory I wrote something like, "Paramedics unavailable. Patient transported with CPR en route to Mater Hospital."

EXHIBIT: I NOW SEEK TO PRODUCE REPORT Q036.

7. I remember seeing a woman at 36 Rawson Street, Mayfield that day but I do not recall speaking with her or what she was doing at the house. I remember speaking with a man at the house who I believed was the father of the baby. I don't recall any exact conversation with this man but I remember being told that the baby had some undiagnosed medical history. He said

Witness: _____ Signature: _____

something along the lines of the baby was sick but they were not sure what was wrong with him.

8. I have also read an ambulance report number Q037 which was completed by officer Coyle. This document appears to contain erroneous information in that the examination section shows that the baby was breathing which is then contradicted by the recording of a nil respiratory rate. I can categorically state that the baby was not breathing when I examined him.

Witness: _____ Signature: _____
D/S/C Ryan
6.9.99

Witness: _____ Signature: _____

Ambulance Service of New South Wales

GRADE 1 2 3 4 5
REGION G A

AMBULANCE REPORT

DATE	JULIAN	CASE No.	PATIENT	SEX	AGE
13/3/91	0,4,4,1	Q,0,3,6	1 of 1	A	1

CALL TYPE	AUTHORITY	STN. No.	RADIO No.	RESPONSE	CATEGORY
CA	CALL	Q911	A11	1	C1

TIME BOOKED	TIME OUT	LOCATION	DEPART	DESTINATION	CLEAR
10.02	10.02	10.10	10.15	10.17	11.12

PATIENT DETAILS

CODE	TITLE	SURNAME	FIRST NAME	DOB	STRETCHER S / WALKING W / W CHAIR C
B	BABY	FOLOICA	PATRICK		

ADDRESS: 36 NAWSON ST ANNERLEY — CODE: 4,1,0,4

DEBTOR DETAILS

☐ W.COMP ☐ 3rd PARTY

CHG. CODE	AMOUNT	CHG. KM	CODE	TITLE	SURNAME/BUSINESS	INITIALS
2			E,9,9			

PENSION No. / WARRANT No. — ADDRESS — CODE

OCCUPATION — EMPLOYED BY

		FROM	TO	CODE
ODO IN	70707	ANNERLEY		
ODO OUT	70710			E,9,9,9
TRIP KM	7	CODE 2,1,0,4		

HISTORY (State chief complaint first)

ASSIST A&E & REMOVE TO ANNERLEY

P.T.S.: W.T. / A / B.P. / N / S / W / TOTAL

EXAMINATION

AIRWAY ☐ Clear ☐ Obstructed
BREATHING ☐ Present ☐ Absent ☐ Shallow ☐ Deep
CIRCULATION ☐ Present ☐ Absent
Buccal Mucosa ☐ Pink ☐ Blue ☐ Pale
Skin temp. ☐ Normal ☐ Cold ☐ Hot
Sweating ☐ Nil ☐ Mod. ☐ Profuse
Vomiting ☐ Nil ☐ Small ☐ Large
Fitting ☐ Nil ☐ Yes Number
Burns ☐ Nil ☐ Superficial % ☐ Deep %
Blood Loss (External) ☐ Nil ☐ Under 500ml ☐ Over 500ml
Site

INJURIES

F Fracture L Laceration A Abrasion S Swelling H Haemorrhage T Tenderness C Contusion D Dislocation B Burn P Pain

Specific observations ☐ Nil

PREPARED BY

CHECKED BY

Breath Sounds

× Position of Patient
→ Point of Impact
▨ Damaged Area
FRONT
Seat Belt Not / Worn
Estimated Impact Speed High Medium Low

ROAD USER: 1 Driver 2 Passenger 3 Motorcyclist & Pillion: 4 Pedal Cyclist 5 Pedestrian 6 Others

	Time	Pulse	Blood Pressure	Capillary Filling	Respiration Rate	Respiration Effort	L Level of Cons	Total Score
	1010	0	0	0	0	0	1	
	1015	0	0	0	0	0	1	
	1017	0	0	0	0	0	1	

Vital Signs Points: First 0 / Final 0

Add Vital Sign Points: 0 + 0 + 0 + 0 + 0 = 0

Difference Between Total Score: 0

Pupils — Size T R L; React. R L

GLASGOW COMA SCALE — Eye Opening / Best Motor Response / Verbal Response

TABLE

TREATMENT BEFORE ARRIVAL

0 Nil (1) PR within 3 mins. 2 First Aid 3 Medical

Patient's state prior to arrival: A Conscious (B) Unknown B Unconscious Mins.

PREVIOUS ILLNESS
☐ Nil ☐ Renal
☐ Unknown ☐ Stroke
☐ Hypertension ☐ Epilepsy
☐ Cardiac ☐ Psychiatric
☐ Respiratory ☐ G.I.T.
☐ Diabetes
☐ Other

Allergies ☐ Nil ☑ Unknown

Present Medication ☐ Nil ☐ Unknown
VISION

Comments

TREATMENT

AIRWAY: 1 Suction; (2) Oral Airway; 3 Nasal Airway; ☐ ETT

BREATHING: 1 RM; (2) RS; 3 ..... (other); (1) IPPV

CIRCULATION: (1) Cardiac Compression

POSTURE: 1 Lateral; (2) Supine; 3 Sitting; 4 Legs Elevated

O_2 THERAPY: 1 High Conc. Mask; 2 Nasal Prongs; 3 28% Ventimask; 1 14L 2 8L 3 4L; (1) Demand Valve

HAEMORRHAGE CONTROL ☐ Dressing ☐ Elevation ☐ Pressure

IV CANNULATION: 1 Type ..... 2 Unsuccessful 3 In Situ; Local given ☐ No ☐ Yes

INTUBATION: 1 Type ..... 2 Unsuccessful 3 In Situ

ANALGESIA: E: Entonox N Narcotic; 1 Not Effective 2 Part Effective 3 Effective

SPLINTS: 1 Hare 2 Thomas 3 Air 4 Cervical Collar 5 KED 6 Other 7 Donway

MAST Suit - Inflated: 1 Under 500ml 2 Over 500ml (External blood loss)

TOURNIQUETS: 1 Rotating 2 Arterial 3 Lymphatic Time On .....

STRETCHER: 1 Carry Chair 2 Back Board 3 Jordon Frame 4 Carry Sheet 5 Other

KITS: 1 Poison 2 Burns 3 Maternity 4 Other

BLOOD SAMPLE TAKEN: Glucose Est. ..... Blood Type & Cross Match .....

TYPENEX No.

RHYTHMS MONITORED (Abbreviations in heavy print)

1 Sinus Rhythm
2 Sinus Bradycardia
3 Sinus Tachycardia
4 Prem. Atrial Cont.
5 SupraVent. Tachy
6 Atrial Fib/Flut.
7 IdioVent. Rhythm
8 Asystole
9 Pacemaker Rhythm
10 Prem. Vent. Cont. Frequency ..... Runs ☐ R on T ☐ Multifocal ☐
11 Vent. Tachycardia
12 Vent. Fibrillation
13 1° Heart Block
14 2° Heart Block
15 3° Heart Block
16 Junctional
17 Multiple Arrhythmia
18 Elect. Mech. Dissn.

TIME	ECG	TREATMENT	DOSE	ROUTE	SHOCK	RESULT	AUTH.

COMMENTS, MEDICAL ASSISTANCE, PROBLEMS, INSTRUCTIONS, ETC.

Dr's Signature

Condition at Destination: Improved / Unchanged / Deteriorated

PRINT NAMES: Officer 1 Driving — Officer 2

SIGNATURES & EMPLOYEE No.s

ATTACH COST STICKER HERE

TRANSTAT

BYSTANDER
Effect.
ROAD USER
Severity
1 TREATMENT before arrival
C Conscious/Unconsc
SUCTION
1 AIRWAY
2 BREATHING
1 IPPV
CIRCULATION
2 POSTURE
1 O_2 THERAPY
Litre
DEMAND VALVE
CANNULATION
INTUBATION
ANALGESIA
Effect.
SPLINTS
SPLINTS
MAST SUIT
TOURNIQUETS
STRETCHER
KITS
ECG
ECG
ECG
D.C. SHOCK
FLUID/DRUG
Units
FLUID/DRUG
Units
FLUID/DRUG
Units
FLUID/DRUG
Units
FLUID/DRUG
Units
16 PROTOCOL
01 PROTOCOL
PROTOCOL
PROTOCOL
RESCUE
COMPLICATIO
HAZARDS
TENS/PNEUM.RE
CO
M

OFFICE COPY

12 FEB 1991

MOORE - SPEEDISET - print ... VR 2651 3/8

57

193

OFFICE COPY

DEPARTMENT OF HEALTH, N.S.W.
AMBULANCE SERVICE
GRADE 1 2 3 4 5
REGION 4 R

AMBULANCE REPORT

DATE	JULIAN	CASE No.	PATIENT	SEX	AGE
3/2/91	034 91	B 10 3 7	1 of 1	M	6mo

CALL TYPE	AUTHORITY	STN No.	RADIO No.	RESPONSE	CATEGORY
CA	CONTROL	911	A68	1	C1

TIME BOOKED	TIME OUT	LOCATION	DEPART	DESTINATION	CLEAR
1003	1003	1000	1015	1018	1103

PATIENT DETAILS

CODE	TITLE	SURNAME	FIRST NAME	D O B
B	MASTER	FOLDICG	PATRICK	3-6-90

ADDRESS: 36 RAWSON ST, MAYFIELD — CODE: 2310 4

STRETCHER S / WALKING W / W/CHAIR C: 5

DEBTOR DETAILS ☐ W/COMP ☐ 3rd PARTY

CHG CODE	AMOUNT	CHG KM	CODE	TITLE	SURNAME/BUSINESS	INITIALS
1		16			CRAIG FOLDICG	

PENSION No / WARRANT No: 5868328 — ADDRESS: Coded via N.T.D. — CODE:

OCCUPATION: Per phcall to NIB — EMPLOYED BY: Tel 4/2/91 26/2/91

		FROM	CODE	TO	CODE
ODO IN	416	Res.	2304	MATER	0211
ODO OUT	16940				
TRIP KM	16				

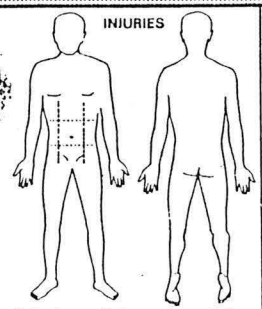
HISTORY (State chief complaint first) CODE 2. (B03) PT IS A 6 MONTH OLD OF C/D PMH OF APNOEA EPISODES + EPILEPSY. O/E. BUD CODE 2 TIME? O/A PT & SKIN COLOUR PINK PERIPHERAL CYANOSIS, SKIN TEMPERATURE, MILK VOMIT. SIGNS. PT PMH EPILEPSY. N.A.D. CODE 2 O/A AT HOSPITAL CAR 36, ~~6~~ ON SCENE.

EXAMINATION

AIRWAY	☑ Clear	☐ Obstructed	
BREATHING	☑ Present	☐ Absent	
	☑ Shallow	☐ Deep	
CIRCULATION	☐ Present	☑ Absent	
Buccal Mucosa	☐ Pink	☐ Blue	☑ Pale
Skin temp.	☑ Normal	☐ Cold	☐ Hot
Sweating	☑ Nil	☐ Mod.	☐ Profuse
Vomiting	☑ Nil	☐ Small	☐ Large
Fitting	☑ Nil	☐ Yes	Number
Burns	☑ Nil	☐ Superficial	%
		☐ Deep	%
Blood Loss (External)	☑ Nil	☐ Under 500ml	☐ Over 500ml

Site .....

INJURIES



F Fracture L Laceration A Abrasion S Swelling
H Haemorrhage T Tenderness C Contusion D Dislocation
B Burn P Pain

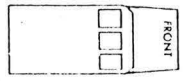
Specific observations ☐ Nil

PREPARED BY KC

CHECKED BY

Breath Sounds .....

× Position of Patient
→ Point of Impact
▨ Damaged Area



Seat Belt Not / Worn
Estimated Impact Speed High Medium Low

ROAD USER: 1 Driver 2 Passenger 3 Motorcyclist & Pillions 4 Pedal Cyclist 5 Pedestrian 6 Others

	Time	Pulse	Blood Pressure	Capillary Filling	Respiration Rate	Respiration Effort	L Level of Cons.	Total Score
	1010	NIL	NIL	NIL	NIL		1	
	1015	NIL	NIL	NIL	NIL		1	
Vital Signs Points	First	NIL						
	Final	NIL						

Add Vital Sign Points

NIL + NIL + NIL + NIL + 3 = 3

NIL + NIL + NIL + NIL + 3 = 3

Difference Between Total Score 0

Pupils

Size T	Size R	Size L	React. R	React. L
0	=	0	2	2
0	=	0	2	2

GLASGOW COMA SCALE

	Eye Opening	Motor Response	Verbal Response	S Score
	1	1	1	3
	1	1	1	3

Eye Opening: 4 Spontaneous; 3 To Speech; 2 To Pain; 1 Nil
Best Motor Response: 6 Obeys; 5 Localises; 4 Withdraws; 3 Abnormal Flexion; 2 Extends; 1 Nil
Verbal Response: 5 Orientated; 4 Confused Conversation; 3 Inappropriate Words; 2 Incomprehensible Sounds; 1 Nil

TABLE | 14-15=5 | 11-13=4 | 8-10=3 | 5-7=2 | 3-4=1

TREATMENT

TREATMENT BEFORE ARRIVAL
0 Nil 1 CPR within 3 mins. 2 First Aid 3 Medical

Patient's state prior to arrival
A Conscious C Unknown
B Unconscious Mins. .....

PREVIOUS ILLNESS
☐ Nil ☐ Renal
☐ Unknown ☐ Stroke
☐ Hypertension ☑ Epilepsy
☐ Cardiac ☐ Psychiatric
☐ Respiratory ☐ G.I.T.
☐ Diabetes
☐ Other .....

Allergies ☐ Nil ☑ Unknown

Present Medication ☐ Nil ☑ Unknown

Comments .....

AIRWAY 1 Suction; (1) Oral Airway; 2 Nasal Airway; ☐ ETT

BREATHING 1 RM; (2) RS; 3 ..... (other); (1) IPPV

CIRCULATION 1 Cardiac Compression

POSTURE 1 Lateral; (2) Supine; 3 Sitting; 4 Legs Elevated

O_2 THERAPY 1 High Conc. Mask; 2 Nasal Prongs; 3 28% Ventimask; 1 14L 2 8L 3 4L; (1) Demand Valve

HAEMORRHAGE CONTROL ☐ Dressing ☐ Elevation ☐ Pressure

IV CANNULATION 1 Type ..... 2 Unsuccessful 3 In Situ
Local given ☐ No ☐ Yes

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ANALGESIA E Entonox N Narcotic; 1 Not Effective 2 Part Effective 3 Effective

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MAST Suit - Inflated 1 Under 500ml 2 Over 500ml (External blood loss)

TOURNIQUETS 1 Rotating 2 Arterial 3 Lymphatic Time On .....

STRETCHER 1 Carry Chair 2 Back Board 3 Jordon Frame 4 Carry Sheet (5) Other

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BLOOD SAMPLE TAKEN Glucose Est. ..... Blood Type & Cross Match .....

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TIME	ECG	TREATMENT	DOSE	ROUTE	SHOCK	RESULT	AUTH.

CHECKED
CASE SHEET
DETAIL CHECKED &
FOUND CORRECT
J.C. — STATION OFFICER

COMMENTS, MEDICAL ASSISTANCE, PROBLEMS, INSTRUCTIONS, ETC.

Dr's Signature .....

Condition at Destination ☐ Improved ☐ Unchanged ☐ Deteriorated

Officer 1 Driving	PRINT NAMES	Officer 2
R MULLINS		R. COYLE
SIGNATURES & EMPLOYEE No		
4045		4045

ATTACH ECG STRIPS HERE

	TRANSTAT
	BYSTANDER
	Effect
	ROAD USER
	Severity
10	TREATMENT before arrival
B	Conscious/Unconsc
	SUCTION
1	AIRWAY
2	BREATHING
1	IPPV
	CIRCULATION
2	POSTURE
1	O_2 THERAPY
	Litre
1	DEMAND VALVE
	CANNULATION
	INTUBATION
	ANALGESIA
	Effects
	SPLINTS
	SPLINTS
	MAST SUIT
	TOURNIQUETS
5	STRETCHER
	KITS
	ECG
	ECG
	ECG
	D.C. SHOCK
	FLUID/DRUG
	Units
	FLUID/DRUG
	Units
	FLUID/DRUG
	Units
	FLUID/DRUG
	Units
	FLUID/DRUG
	Units
	PROTOCOL
	PROTOCOL
	PROTOCOL
	PROTOCOL
	RESCUE
	COMPLICATIONS
4	HAZARDS
0	TENS PNEUM RELI
1	COMMENTS
0	MEDICAL REVIEW

3H

New South Wales Police P.190

v2.9

STATEMENT in the matter of:
Death of Patrick FOLBIGG

Place: Home address of Mr Mullens
Date : 1 October 1999

Name: Anthony Russell MULLENS
Address: 23 Haddington Dr, Cardiff South **Tel. No.:** 02 49565883
Occupation: Ambulance Officer **STATES:-**

1. This statement made by me accurately sets out the evidence which I would be prepared, if necessary, to give in court as a witness. The statement is true to the best of my knowledge and belief and I make it knowing that, if it is tendered in evidence, I shall be liable for prosecution if I have wilfully stated in it anything which I know to be false, or do not believe to be true.

2. I am 44 years of age.

3. I am a level four Ambulance Officer stationed at the Boolaroo Ambulance Station. I have been a member of the New South Wales Ambulance Services for the past thirteen years. I have been qualified at level four for the past seven years.

4. On the 13th of February, 1991 I was a level three ambulance officer stationed at Hamilton. On this day I was working with Ambulance Officer Kathy COYLE on car A68. I was the driver and Kathy was the treatment officer. Ambulance Officer Murray HETHERINGTON was also working out of Hamilton that day as a single unit.

5. At 10.03am that day Kathy and I responded to call of a suspected death of a baby at 36 Rawson Street, Mayfield. We arrived at the house at 10.10am that day at the same time as Murray HETHERINGTON. It was a small cottage with the entrance off a driveway at the right hand side of the house. I walked in the

Witness: _____ **Signature:** _____

front door of the house with Kathy and Murray. I saw a baby inside the house but I can't remember if it was in the loungeroom or a bedroom. I also saw a young man and young woman at the house who I presume were the mother and father of the baby. (One of them said that the baby was blind and had previous other medical problems.) These two people appeared to be upset. Murray commenced CPR on the baby in a room of the house and baby appeared not to be responding. I walked outside the house to the ambulance and prepared for transportation of the baby. I do not remember actually treating the baby.

6. At 10.15am that day Murray and Kathy put the baby into the ambulance and I drove straight to the Mater Hospital. We called off at the Hospital at 10.18am and the baby was treated by hospital staff.

7. At the hospital Kathy completed Ambulance case sheet number Q037. I read this document when it was completed and signed as the driver of the job. On 1 October, 1999 Detective RYAN showed me that case report and I saw that Kathy had recorded that breathing was present and shallow. I did not treat the baby but I believe there was never any breathing noted when we arrived that day. It appears to be an error on the case sheet.

Witness: _____ Signature: _____

B. Ross
D/Sgt
1.10.99

New South Wales Police P.190
v2.9

STATEMENT in the matter of:
Patrick FOLBIGG

Place: John Hunter Hospital
Date : 8 October 1999

Name: Ian Arthur WILKINSON
Address: 115 Elder Street, Lambton Tel. No.: 02 49526599
Occupation: Paediatric Neurologist STATES:-

1. This statement made by me accurately sets out the evidence which I would be prepared, if necessary, to give in court as a witness. The statement is true to the best of my knowledge and belief and I make it knowing that, if it is tendered in evidence, I shall be liable for prosecution if I have wilfully stated in it anything which I know to be false, or do not believe to be true.

2. I am 54 years of age.

3. On 12 March 1999 I completed a statement in relation to my treatment of the late Patrick FOLBIGG. I also arranged for my entire original medical file relating to Patrick FOLBIGG to be forwarded to Detective Bernie RYAN at Singleton, as requested by a Coroner's Order.

4. As a result of a request by Detective RYAN, I am prepared to add further to my original statement. About 10.30am on 13 February 1991 I was working at the Mater Hospital in Newcastle when I was called to the Casualty Department where I saw Hospital Staff performing CPR on a small child who I recognised as Patrick FOLBIGG. During the resuscitation attempts it became clear to me that Patrick had died and there was no point in further attempts at resuscitation. I then discussed with Mr and Mrs FOLBIGG who were in the room watching the attempts, that I felt that it was futile to continue resuscitation. The parents agreed to cessation of resuscitation attempts which took place immediately.

Witness: _____ Signature: _____

5. During the resuscitation attempts I examined Patrick. His appearance at the time was consistent with a patient who had suffered asphyxiation. At that time I knew that Patrick had suffered from epilepsy in the past and felt that on this occasion he could have experienced an epileptic fit which had resulted in obstruction of his airways, asphyxia with consequent cerebral anoxia and subsequent death. At that moment there appeared to be no suspicious circumstances. According to the Hospital records Patrick was pronounced dead at 10.40am that day by Doctor Chris Walker.

6. At this stage I do not recall signing a death certificate relating to Patrick's death. On 8 October 1999 Detective RYAN showed me a copy of a death certificate Reference 101831/... which lists my name as being the Doctor who certified the causes of Patrick's death. The handwriting on this certificate is not my own and there is no signature attached. I am not saying that I did not complete a death certificate in relation to Patrick but I do not recognise the one shown to me. It is possible that this document is a handwritten copy of the original. I see it is annotated by Maree BELL who is the Medical Record Librarian at the Mater Hospital.

7. At the time of Patrick's death I saw no evidence of foul play and it did not appear necessary for the police to be notified. I was satisfied with my diagnosis, however after becoming aware that a further two of Patrick's siblings have died since, I have doubt in my mind. I still believe that Patrick could have been asphyxiated but I have doubts that it was as a result of an epileptic fit. I must stress that I cannot positively rule out that an epileptic fit did cause the asphyxiation. Other causes of asphyxia must now be considered in light of the other deaths in

Witness: _____ Signature: _____

STATEMENT (continued) in the matter of: Patrick FOLBIGG
Name: Ian Arthur WILKINSON

the family. I would not have issued a death certificate if Patrick's death had been preceded by the death of three of his siblings.

Witness: _____ Signature: _____

B. Ryan
DHE.
18-10-99

2I

EXPERT CERTIFICATE in the matter of: Death of Patrick FOLBIGG
Place: John Hunter Hospital Date: 18.1.2000

Name: Christopher WALKER
Address: C/John Hunter Hospital Tel.No: 02 49213000
Occupation: Medical Practitioner STATES:-

EXPERT CERTIFICATE
Section 177, Evidence Act 1995 No. 25

1. This statement made by me accurately sets out the evidence which I would be prepared, if necessary, to give in court as a witness. The statement is true to the best of my knowledge and belief and I make it knowing that, if it is tendered in evidence, I shall be liable to prosecution if I have wilfully stated anything which I know to be false or do not believe to be true.

2. I am 49 years of age.

3. I hereby certify:
My full name is **Christopher WALKER**
My contact address is **C/John Hunter Hospital**
I have a specialised knowledge based on the following training, study and experience:-
I completed the Bachelor of Science (BSC) at Sydney University in 1974, Bachelor of Medicine and Bachelor of Science (MBBS) at the Sydney University in 1978. I was elected a Fellow of the Australasian College for Emergency Medicine (FACEM) in May 1996. I am currently employed as a Specialist Emergency Physician at the John Hunter Hospital.

4. At 10.20am on the 13th of February 1991, I was practicing emergency medicine as Director of the Emergency Department at the Newcastle Mater Hospital. I was on duty when an eight month old male infant was brought to hospital by the New South Wales Ambulance Service. From the pre-hospital ambulance report and speaking with

See Continuation Sheet ...

Witness: _____ Signature: _____

a parent of the child (probably the mother) I obtained the following history:

The child had been found by the mother sometime prior to the ambulance being called that morning. I was told that Mrs FOLBIGG had then called her husband at his work in Kotara and had also called a consultant Paediatric Neurologist, Dr Ian WILKINSON. I was also told that Mr FOLBIGG had driven from his work place in Kotara to their home in Mayfield and had commenced bystander CPR. I was told that this had occurred prior to the arrival of the New South Wales Ambulance Service at the child's home at 10.10am. The ambulance officers reported to me that on arrival at the home they found the child to be pulseless and not breathing. The child was reported by the ambulance officers as peripherally cyanosed. The child was also reported to have warm skin temperature. Basic life support was continued by ambulance officers. Bag mask ventilation with oxygen and external cardiac compression was performed until arrival at the Hospital at 10.18am.

5. I examine the child on arrival at hospital. The child was not breathing and receiving ventilator support with oxygen by hospital emergency staff. The child was placed on an ECG monitor. The monitor showed asystole. Resuscitation continued, a 4.5 millimetre noncuffed endotracheal tube was placed in the child's airway. Through this tube one ml of 1:10,000 solution of adrenalin diluted to 10mls was given. External cardiac compression was continued. Intravenous access was obtained and the child was given a total of three doses of .5 milligrams each of adrenalin intravenously. Following the adrenalin the patient developed a broad complex agonal rythym on the ECG monitor which was not maintained. No cardiac output evidenced by a spontaneous palpable pulse was noted at any stage. Resuscitation ceased after twenty minutes and death was pronounced by me at 10.40am on the 13th of February 1991.

Witness: _____ Signature: _____

6. Following the child's death, I interviewed the child's parents in the presence of medical and nursing staff from the Mater Hospital. I then made contemporaneous medical notes in the child's medical record. I noted that Dr Wilkinson indicated an intention to sign a cause of death certificate. I obtained permission from Mr FOLBIGG for an autopsy to be performed on their child Patrick FOLBIGG.

EXHIBIT: I NOW SEEK TO PRODUCE CONTEMPORANEOUS NOTES.

7. It is my opinion that Patrick FOLBIGG suffered an asystolic cardiac arrest prior to his arrival at Hospital and was not resuscitable. I am unable to state was caused Patrick's cardiac arrest.

Witness: _____ Signature: _____
18-1-2000

5mL 1:10,000 ETT
3×

Sign, Date and Record Designation for all Entries

DATE

AIE
DATE 13 FEB 1991
TIME OF ARRIVAL 1025 ← 1020
TIME SEEN BY R.M.O. 1020
TIME OF DISCHARGE

T - P - R - Wt -

Asystolic arrest of 8/12 old boy

Background Presented 5/12 of age apnoeic & floppy. resuscitated
Subsequently:
? encephalitis / encephalopathy
seizure disorder — difficult to control previously, better control of late.
cortical blindness
developmental regression

Brother died 1989 19/52 old "SIDS" — Caleb.

Last night ? seizure
mild ↑ Temp.
otherwise no problems

This am 0730 — Patrick put back to sleep was 'well'
930 — 10 — Mum found baby.
Rang husband. Rushed home from work. No heart beat or breathing. Commenced CPR, Amb & phoned.
Paramedics arrived: No heart beat. No reflx. CPR continued. Baby's colour "good" and still warm.
1020: Arrived Mater Cas.
Asystole on monitor
ETT & IV cannula inserted.
1020 5mls of 1:10,000 adrenalin given ETT
1022 5mL of 1:10,000 adrenalin IV given.

N.M.M.H.

OUTPATIENT CONTINUATION RECORD

Sign, Date and Record Designation for all Entries.

Please use gummed label if available

SURNAME Foldigg	UNIT NUMBER
OTHER NAMES Patrick	
ADDRESS	
DATE OF BIRTH	WARD
V.M.O.	R.M.O.

5mls ie 1:1 muales. Ca Gluconate

13/2/91 1025 ~~5mg~~ Ca gluconate given IV

1029 2·5mL 1:10,000 adrenalin given IV

1032 2·5mL 1:10,000 adrenalin given IV

Only alteration from asystole was the transient development of an agonal rhythm after the dose of adrenalin.

S/B Dr Dzendolet, Dr I. A. Wilkinson. Resus team led by Dr Walker.

CPR continued until arrival of mother. With both parents at bedside, Dr Wilkinson explained that there would be no benefit from further efforts.

1040 Resuscitation ceased and life pronounced extinct.

Parents seen to "know why."

Parents s/b Dr Wilkinson, pastoral care people, Leanne Ellison (Subst.)

Post mortem desired. — paternal permission ✓

WALKER

8/12 ♂ Child with Pr Epilepsy
2° Cortical Blindness

Mother found child apnoeic + lifeless + called Dr Wilkinson + called her husband from work

Mr Folbigg aware from Kalgoorlie transfer (or Commenced Bystander CPR on arrival. Ambulance (non paramedic) arrived ~1010 — transported + NMMH arrived 1020 hrs. on monitor

On arrival Asystolic Apnoeic. Receiving expired air resuscitation.

Sign, Date and Record Designation for all Entries.

DATE	
	4.5 non cuffed ETT
	Give Adrenaline 1.0ml 1:10,000 dilute to 10ml via ETT.
	Then Adrenaline 5ml 1:10,000 IVI = 0.5mg followed by 2 further doses Adrenaline 0.5mg / IVI
	Continuous ventilate / ECG
	Agonal Rhythm 10 beats/min obtained on ECG monitor after Adrenaline
	No output obtained
	Resuscitation ceased after 20min at 1040
	Pt pronounced deceased. 1040 Hrs
	Viz no BP
	nil pulse
	nil spont. respirations
	Dr Williamson has filled in an intention to sign death certificate + permission for Autopsy obtained.
	[signature]
	Anaesthetist
	[illegible]

EXPERT CERTIFICATE in the matter of: Death of Patrick FOLBIGG
Police -v-
Place: Mater Hospital Date: 11.2.2000

Name: Man Kit LAI
Address: 24 Bershire Ave, Merewether Heights Tel.No: 02 49211211
Occupation: Medical Doctor (Radiologist) STATES:-

EXPERT CERTIFICATE
Section 177, Evidence Act 1995 No. 25

1. This statement made by me accurately sets out the evidence which I would be prepared, if necessary, to give in court as a witness. The statement is true to the best of my knowledge and belief and I make it knowing that, if it is tendered in evidence, I shall be liable to prosecution if I have wilfully stated anything which I know to be false or do not believe to be true.

2. I am 45 years of age.

3. I hereby certify:
My full name is **Man Kit LAI**
My contact address is **Mater Hospital**
I have a specialised knowledge based on the following training, study and experience:-
MBBS (Bachelor of Medicine and Bachelor of Surgery) at the University of Hong Kong in 1980. FRCR (Fellow of Royal College of Radiologists) in the United Kingdom in 1985. I have been employed as a Staff Specialist Radiologist at the Mater Hospital in Newcastle since February 1989.

4. On Friday the 11th of February 2000, I spoke with Detective RYAN at the Mater Hospital in relation to a patient known as Patrick FOLBIGG. Before I go any further I would like to state that I do not remember this patient.

See Continuation Sheet ...

Witness: B. Ryan D/S/C. 11.2.2000

Signature:

5. Detective RYAN showed me two C.T. brain scan reports, dated the 23 October 1990 and the 5 November 1990. I recognised these reports as being completed at the Mater Hospital on those dates after the completion of C.T. scans. My name appears at the foot of those reports and I recognise my signature on the report dated the 5 November 1990. This indicates that I definitely completed that report, however I have not signed the other report. Even so, I assume that I did complete that report also. All the C.T. scans at that time were usually completed by me. (C.T. scan stands for Computerised Axial Tomography and is a radiological procedure utilising x-ray to produce images of the body.)

6. I have read what I have reported in the mentioned reports and can say that at the time of reporting, I believed the patient was suffering from encephalitis, possibly due to herpes simplex. From what is recorded in the report, I believe I was searching for the most likely diagnosis and this should always be considered until excluded. I do this everyday which is my usual work practice.

EXHIBIT: I NOW SEEK TO PRODUCE CT BRAIN SCAN - DATED 23 OCTOBER 1990

EXHIBIT: I NOW SEEK TO PRODUCE CT BRAIN SCAN - DATED 5 NOVEMBER 1990

7. I have conducted a through search of the X-ray department at the Mater Hospital and I cannot find the C.T scan films relating to the patient Patrick FOLBIGG. I assume that these films have been destroyed in accordance with Hospital policy.

Witness: _____ **Signature:** _____

NEWCASTLE MATER MISERICORDIAE
ORGAN IMAGING DEPARTMENT
COMPUTERISED TOMOGRAPHY

M.R.N: 0360390
FOLBIGG, Patrick
36 RAWSON STREET
SEX: Male LD 2304 4M
D.O.B: 03-Jun-1990

R.M.O: DR D COOPER
WARD/CLINIC: 4G

REQ.NO: 89C001317

Exams : CB3 on 23-Oct-1990 at 11:00

CT BRAIN (pre and post contrast scans)

In the pre contrast scant there is a decrease in attenuation seen in both occipital lobes, temporal lobe and left frontal lobe. The grey/white matter differentiation is lost. Ventricular system not dilated. No haemorrhage seen. Minimal widening of the peripheral cerebral sulci is seen in the frontal and the parietal lobes.

Post contrast scan with thin cuts over the posterior cranial fossa and temporal lobe shows the hypodense areas involving both posterior parts of the temporal lobes and occipital lobes. Abnormal enhancement demonstrated. The intra-cranial vessels are well enhanced. No abnormal fluid collection seen.

IMPRESSION

The picture is compatible with encephalitis involving both temporal lobes, occipital lobes and left frontal lobe. Herpes encephalitis has to be considered.

DR J. LAI

Printed 23/10/90 14:58 FINAL REPORT

NEWCASTLE MATER MISERICORDIAE
ORGAN IMAGING DEPARTMENT
COMPUTERISED TOMOGRAPHY

M.R.N: 0360390
FOLBIGG, Patrick
36 RAWSON STREET
SEX: Male LD 2304 5M
D.O.B: 03-Jun-1990

R.M.O: Resident MEDICAL OFFICER
WARD/CLINIC: 4G

REQ.NO: 89C001356

Exams : CB3 on 05-Nov-1990 at 14:30

CT BRAIN (pre and post contrast scans)

NO OLD FILMS FOR COMPARISON

In the pre contrast scan there is mild generalised widening of the subarachnoid space. Ventricular system not dilated. There is some increased density seen in both occipital lobes. The grey/white matter differentiation is intact otherwise.

In the post contrast scan with thin cuts over the posterior cranial fossa, the 4th ventricle is not dilated. Some abnormal enhancement is seen in both occipital lobes, patchy in areas and distributed in both grey and white matter (slice 20 - 23).

IMPRESSION

There is generalised loss in brain substance. The patchy enhancement seen in both occipital lobes could be related to the post inflammatory changes. The high density seen in the pre contrast scan may be due to dystrophic calcification.

DR J. LAI

Printed 06/11/90 09:42 FINAL REPORT

2E

EXPERT CERTIFICATE in the matter of: Death of Patrick FOLBIGG

Place: St Kilda Police Station, Victoria Date: 17 March 2000

Name: Joseph George DEZORDI
Contact Address: 11 Nottage Street, St Kilda Tel.No: 0419 258805
Occupation: Paediatrics Fellow STATES:-

EXPERT CERTIFICATE
Section 177, Evidence Act 1995 No. 25

1. This statement made by me accurately sets out the evidence which I would be prepared, if necessary, to give in court as a witness.

The statement is true to the best of my knowledge and belief and I make it knowing that, if it is tendered in evidence, I shall be liable to prosecution if I have wilfully stated anything which I know to be false or do not believe to be true.

2. I am 38 years of age.

3. I hereby certify:
My full name is Joseph George DEZORDI
My contact address is 11 Nottage Street, St Kilda
I have a specialised knowledge based on the following training, study and experience:-
I trained in Medicine at the University of Melbourne, and subsequently trained in paediatrics in Alice Springs, Newcastle, Sydney, and Melbourne over a period of seven years. I also have worked in general practice for an additional three years. I am currently completing my advanced training in paediatrics at the Latrobe Regional Hospital in Victoria.

4. About 5am on the 18th of October 1990 at the Newcastle Mater Hospital, I was working as the paediatrics night resident. I was

See Continuation Sheet ...

Witness: B. [illegible] D/S/C. 17.3.2000

Signature: Joseph Dezordi

called to an emergency in the casualty department, there I examined Patrick FOLBIGG.

I saw a lethargic, cyanosed infant, who was responsive only to painful stimuli.

I spoke with a woman who identified herself as the child's mother and she informed me that Patrick had been coughing at 3.00am and was therefore seen by her at that time. She was then alerted again at 4.30am because she heard him gasping, and she noted that he was blue around the lips, and that he was lifeless and floppy, and was making minimal respiratory effort.

She stated that cardio pulmonary resuscitation was not performed, and that soon after this Patrick gave a high pitched cry.

She stated that he revived slightly when the paramedic administered oxygen some twenty minutes later.

I proceeded to treat Patrick, and perform tests.

He was treated with oxygen administered by a Hudson mask, and I noted that after about fifteen minutes he became more alert, and remained pink, even when the oxygen in high concentration was not being administered. I therefore concluded that Patrick's condition was not likely to be due to a respiratory problem.

5. My detailed examination was generally unremarkable at this stage:

I noted that Patrick was an appropriately grown male infant who was arching his back. I did note that there were no signs of upper airway obstruction or of aspiration which might conceivably

have accounted for the history given by Mrs FOLBIGG [that he had been gasping and that he was barely breathing].

There were no signs to suggest any other serious illness, in particular there were no signs to suggest meningitis.

Importantly there was no evidence of trauma or any injuries.

At that stage, by 6.00am some preliminary test results were available. There was no abnormality in the blood tests. Interestingly however, there was significant glycosuria in the absence of hyperglycaemia. I concluded that this was a response to an acute asphyxiating event. At that time I was thinking possibly a seizure of some kind.

Also of interest is that during this early period in hospital, Patrick vomited three times, but he had no respiratory difficulty with these.

The Chest X ray did not demonstrate signs of aspiration or pneumonia. [It was later reported officially, to show signs, which could be due to bronchiolitis].
The Virological tests however, did not support a diagnosis of bronchiolitis.

6. My conclusions at that time were:

The glycosuria suggested an acute event, possibly a seizure or an episode of prolonged hypoxia. The back arching at that stage had suggested to me that he may have been cerebrally irritable. Meningitis or other pathology involving the brain might cause this. However, he did not have sufficient signs at that time for me to pursue a diagnosis of meningitis.

I recorded the details of my examination and treatment of Patrick in the Mater Hospital medical notes.

EXHIBIT: I NOW SEEK TO PRODUCE MATER HOSPITAL MEDICAL NOTES.

7. I next encountered Patrick at 6.00am on the 20th of October 1990 in Baby's ward at the Mater Hospital. By then it was well established that he was having frequent seizures in hospital. I noted that he was fitting, and that his eyes were deviated to the right hand side.

I note that my main involvement after this revolved around organising tests and obtaining the results of these tests.

I recorded an entry in the Mater Hospital medical notes regarding an abnormal CT scan result, [24th October 1990], this scan demonstrated some pathological process involving the occipital and temporal lobes of the brain. It was not really clear what the cause of these unusual CT scan findings were.

My next involvement was on the 5th of November 1990, when I organised a repeat CT scan. I noted on the 6th of November that this second CT scan, demonstrated abnormalities already seen on the previous scan, but these seemed to have worsened. The cause of this "loss of brain substance" was not really clear to any of the medical staff. I remember being asked by Doctor Ian WILKINSON to send these scans to an expert radiologist in Sydney, Professor Merl DeSilva at the Children's Hospital at Camperdown. I forwarded these two scans and a letter to Professor DeSilva on the 16th of November 1990.

EXHIBIT: I NOW SEEK TO PRODUCE LETTER DATED 16/11/90.

In the afternoon of Wednesday the 21st of November 1990 I telephoned Professor DeSILVA at the Children's Hospital in

Joseph Dezordi

Camperdown. I made the call from the Baby's Ward at the Mater Hospital. I remember that Professor DeSilva spoke with a slight accent and the conversation was similar to the following:
I said, "Have you received the CT scans?"
He said, "Yes. The changes in the CT scans are not classical of encephalitis."
I said, "Do you think they are due to coning?"
[At that time, Doctor WILKINSON was questioning the possibility that the lumbar puncture that Patrick may have caused him to have coned]
Professor DeSilva said, "I don't think that is likely. Have you considered child abuse?"
I said, "What do you mean?"
He said, "Such as shaking."
I don't remember asking Professor DeSilva to return the CT scans, however it would have been my normal practice to do so. I do not know what happened to the original scan films.

I immediately telephoned Doctor WILKINSON and informed him of what I was told by Professor DeSilva. Doctor WILKINSON said, "We will go and see the parents."
I considered recording the conversation which I had with Professor DeSilva regarding the allegations of child abuse, however I thought that it would not have been accepted by the medical profession as being proper. This information was only relayed by telephone and I was stunned by the possible implications from recording such details in hospital notes.

About 10 am the following morning being Thursday the 22nd of November 1990, Doctor WILKINSON, Doctor Rob SMITH and I went to the baby's ward at the Mater Hospital where we spoke with Patrick FOLBIGG's parents. I remember that Doctor WILKINSON said to them words to effect, "Patrick is blind and you will need to refer him to the blind society."
Doctor WILKINSON then said, "We sent the CT scans to a Professor

Joseph Dezordi

in Sydney and we are not really entirely sure what the problem has been, but the possibility of a non accidental injury or child abuse was raised. Has there been any foul play?"

I remember both parents saying, "No."

I did not record this conversation in the medical notes, however I note that Dr SMITH made an entry immediately after this discussion. I have read that entry dated 22.11.90 which does not mention the allegation of child abuse which was put to Patrick's parents.

Witness: _____ Signature: Joseph Dezordi

NEWCASTLE MATER MISERICORDIAE HOSPITAL

We Care

INTERIM DISCHARGE LETTER

PLEASE USE GUMMED LABEL IF AVAILABLE

SURNAME FOLBIGG	UNIT NUMBER 360390
OTHER NAMES Patrick	
ADDRESS 36 Rawson st MAYFIELD.	
DATE OF BIRTH 3/JUNE/90	M.O. ROB MORRIS.

91

Top Copy — Med. Records
Second Copy — L.M.O.
Third Copy — V.M.O.

Lmo DR C. MARLEY chgeable

Referred by: .....

Date of Admission: 18/10/90 Emergency/Elective Date of Discharge: 29/10/90.

Final Diagnosis:

1) Intractable Seizures probably Viral Encephalitis.

Other Diagnosis: 2) Bronchiolitis *(Brother died age 19 days last year in sleep)

2: 5month boy presented in apnoeic state, floppy, required

3: simple resuscitation by paramedics & was well

4: in hospital until developed series of generalized &

5: also right sided focal fits associated with low fevers.

Past History: Required large doses of IV phenobarbitone & then IV phenytoin to settle. Started on IV Acyclovir, → 7day course for presumed Herpes Encephalitis. Fit free from third day of Acyclovir.

Operations/Management:

~ ESF — Normal — Herpes Culture Negative.
~ Serum Herpes IgM pending (Westmead)
~ Cranial U/Sound Normal
— Cranial CT scan — Hypodense areas in temporal & occipital lobes.
? Encephalitis - demyelination.

Drugs on Discharge: (Dose, Frequency, Duration):

- Chest XRAY Slight hyperinflation & Tramlinings in keeping with bronchiolitis
- EEG — Left Frontal lobe discharge
- Metabolic WORK UP — pending (Adelaide Children's)
- Rectal biopsy — pending

Information Given to Patient:

— Serum: lactate, ammonia, Calcium, Mg, Glucose all normal
— Urine Metabolic screen: negative aminoacids & negative organic acids. Negative, lactic acid negative

Follow-up:

— Currently on phenytoin 45mg 12hrly phenobarbitone 240mg 12hrly
Levels done on day of discharge & will adjust if need be by phone
Review by Dr Morris 7days

Adverse drug reaction (specify drug):

Joseph Dzoda
Medical Officer (print name)

NEWCASTLE MATER MISERICORDIAE HOSPITAL
We Care

HISTORY, EXAMINATION AND PROGRESS NOTES

PLEASE USE CLEAR WRITING

36-03-90

FOLBIGG, Patrick
36 Rawson Street MAYFIELD 2304
DOB 03-Jun-1990 SEX M
WARD 4G
M.O.: DR. D. COOPER

M.O.

WARD:

92 SHEET

SIGN, DATE AND RECORD DESIGNATION FOR ALL ENTRIES

DATE

0600

19/10/90. 5month boy, apnoeic episode noted by mum at ~~4~~30 am. Brother Caleb died last year age 19 weeks from "SIDS".

Hx of presenting Illness.
- Has been snuffly past 3 days with dry coughs & some vomits post bottle feeds. Otherwise well, behaving normally. no fevers. Coughs not paroxysm
Has not received any medications
Has been in contact with other children neighbours with coughs & one with gastro.

- Seen by mum at 300 am became head hum coughing, at 430, mum heard him gasping. Was blue around the lips, lifeless, floppy making minimal respiratory effort & CPR not performed. Later made a high pitched cry.
- Revived slightly when paramedics administered Oxygen about 20 minutes later..

- ~~Other~~

Fam Hx
No Hx of epilepsy
No Hx of Neurological diseases

Immunisations - TAG/SABIN #1, #2

Development
- Smiling <6 weeks
- Laughs, vocalises
- Nearly rolls over
- Supports weight on legs
- Plays with fingers.

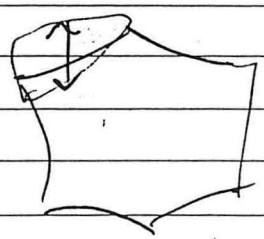
Birth born at 38½ weeks SVD (Western General

SIGN, DATE AND RECORD DESIGNATION FOR ALL ENTRIES

DATE

O/E. Initially T 35 RR PR 160 RR 60 BP 90/50
peripherally Cyanosed. Lethargic
responding only to painful stimuli. Dilated pupils but reactive. Pulse & Sweaty 88% 12L air ⇒ Oxygen via Hudson's Mask
After about 5 minutes - became much more alert & pink on air
Mummy head freely & arching back - (always does this)
Large anterior fontanelle, not tense, not bulging
Sacrally nose
Ears, Throat NAD

Chest: No recession No stridor
Widespread wheeze, Scattered Crackles

Abdo. 
liver span 7 cm
2 cm ↓ costal margin
no masses.

CVS: good pulses initially difficult to feel
after good pulses, all equal, good. femorals
- Normal precordium. No thrills.
- Apex not displaced
- S1 S2 No murmurs.

CNS. Head Cfx 44 cm = 75th centile
(no previous ones for comparison)
pupils = & reactive
fundi - R - well visualised normal
L - not well visualised
No gaze palsy nystagmus
V, VII ✓
IX, X, XI, XII ✓ ao

NEWCASTLE MATER MISERICORDIAE HOSPITAL
We Care

PLEASE USE GUMMED LABEL IF AVAILABLE

UNIT NUMBER 36-03-90

FOLRIGG, Patrick
36 Rawson Street MAYFIELD 2304
DOB 03-Jun-1990 SEX M
WARD 4G
M.O.: DR. D. COOPER

HSMR 1

SHEET

HISTORY, EXAMINATION AND PROGRESS NOTES

WARD: ____

Dose doe dose does

SIGN, DATE AND RECORD DESIGNATION FOR ALL ENTRIES

d

DATE

Tone – normal, normal posture but arches rock
Power – supports weight on legs
– moves all limbs against gravity
& against resistance.

Reflexes – not elicited
plantars – ↑ ↑.

Impression: ? Mild bronchiolitis, central apnoea
? Epileptic Seizure
? post ictal
? "Near SIDS"

Chest XRAY NAD
Finger prick 7 mmoles

Na 142 K 5.7 Urea 4.3 Creat 0.07 Alb 48
Ca 2.52 Mg 1.2

WCC 11.6 11% N
86% Lymphocytes
1% Bands
2% Monocytes

Hb 11.5 plats 434
Blood Culture – pending
* URINE (BAG) ++++ Glucose
Small blood
++ protein

For Head Ultrasound – form
EEG – form – need to book
NAA – form

1-1-85

SIGN, DATE AND RECORD DESIGNATION FOR ALL ENTRIES

DATE

To ward, with apnoea monitoring
& D/Cooper. NBR/ICU
S/B Tony Mullens (Anaesthetics Reg)

Elective Admission at 12 days o/age.

(for sleep study — normal
— Ba Swallow — no a/w)

please collect another Bag urine

3 Small Vomits — gastric fluid (obtain
in CAS — no resp distress with these

Continue oral feeds

18/10/99

Thanks. Coughing leading to gasping
cyanosis & effortless apnoeic. Remained
very ill-looking for the arrival in hospital
now - subdued
- little spontaneous movements
The DTR's in the lower limbs are
somewhat brisk
(L) Fundus disc indistinct
Nc 43.4.

Impression: the physical examination at the
moment is probably related to the recent
events — in a transient fashion
However needs - EEG
— U/S head
— C.T. BS if there is
another event

2G

EXPERT CERTIFICATE in the matter of: Death of Patrick FOLBIGG
Police -v-
Place: Date: 24.3.2000

Name: Ian Arthur WILKINSON
Address: C/John Hunter Children's Hospital Tel.No: 0249213000
Occupation: Paediatric Neurologist STATES:-

EXPERT CERTIFICATE
Section 177, Evidence Act 1995 No. 25

1. This statement made by me accurately sets out the evidence which I would be prepared, if necessary, to give in court as a witness. The statement is true to the best of my knowledge and belief and I make it knowing that, if it is tendered in evidence, I shall be liable to prosecution if I have wilfully stated anything which I know to be false or do not believe to be true.

2. I am 54 years of age.

3. I hereby certify:
My full name is **Ian Arthur WILKINSON**
My contact address is **C/John Hunter Children's Hospital**
I have a specialised knowledge based on the following training, study and experience:- See previous statement.

4. On 12 March 1999 and 8 October 1999 I made two statements in relation to the treatment and subsequent death of Patrick FOLBIGG.

5. On 24 March 2000 I spoke with Detective RYAN at the John Hunter Children's Hospital. He showed me a copy of a death certificate number 101831. I recognised my signature on this death certificate and that it related to the death of Patrick FOLBIGG.

EXHIBIT: I NOW SEEK TO PRODUCE DEATH CERTIFICATE 101831.

See Continuation Sheet ...

Witness: B. Ryan D/S/C. 24.3.2000

Signature: Ian Wilkinson

6. Detective RYAN also spoke to me about two CT scans relating to Patrick FOLBIGG. I have read the scan reports relating to these CT scans, however I do not remember speaking to Doctor DEZORDI or Doctor SMITH in relation to them. I also don't remember speaking with Mr and Mrs FOLBIGG about the scans and the possibility of child abuse.

Witness:_____ Signature:_____

NSW 15

NEW SOUTH WALES

Registration of Births, Deaths and Marriages Act, 1973

Medical Certificate of Cause of Death

10183

Name of deceased PATRICK FOLBIGG

Date of death 13/2 THIRTEENTH day of FEBRUARY 1991

Place of death NEWCASTLE MATER HOSPITAL

Age 8 MONTHS Date last seen alive by me 14/1/91

Did the deceased undergo an operation within 4 weeks of death? Yes ☐ No ☑

Approximate interval between onset and death*

CAUSE OF DEATH (BLOCK LETTERS)

PART I

Disease or condition directly leading to death †

(a) ASPHYXIA DUE TO NASAL OBSTRUCTION 1 HOUR

due to (or as a consequence of)

Antecedent causes (morbid conditions, if any, giving rise to the above-mentioned cause, stating the underlying condition last).

(b) EPILEPTIC FITS 4 MONTHS

due to (or as a consequence of)

(c)

PART II

Other significant conditions contributing to the death, but not related to the disease or condition causing it.

I hereby certify that I am a medical practitioner, currently registered, and that:

- I was in medical attendance ~~immediately before the above-named deceased's death~~; during the above-named deceased's illness terminating in his death;
- I viewed the body of the above-named deceased after death;

and that the particulars and cause of death above written are true to the best of my knowledge and belief.

- This certificate is signed pursuant to Section 24(7A) of the above-mentioned Act.

Signature [signature] Date 14/2/91

Full name of medical practitioner IAN ARTHUR WILKINSON (BLOCK LETTERS)

Residence 10 CURZON ST

NEW LAMBTON Postcode 2305

NEW SOUTH WALES

BIRTHS, DEATHS AND MARRIAGES REGISTRATION ACT 1995

BIRTH CERTIFICATE

REGISTRATION NUMBER
117019/1992

1 CHILD	
Family Name	FOLBIGG
Christian or Given Name(s)	SARAH KATHLEEN
Sex	Female
Date of Birth	14 October 1992
Place of Birth	John Hunter Hospital, New Lambton Heights
2 MOTHER	
Family Name	FOLBIGG
Maiden Family Name	MARLBOROUGH
Christian or Given Name(s)	KATHLEEN MEAGAN
Occupation	SALES ASSISTANT
Age	25 years
Place of Birth	BALMAIN, NEW SOUTH WALES
3 FATHER	
Family Name	FOLBIGG
Christian or Given Name(s)	CRAIG GIBSON
Occupation	SERVICE MANAGER
Age	31 years
Place of Birth	NEW LAMBTON, NEW SOUTH WALES
4 MARRIAGE OF PARENTS	
Date of Marriage	05 September 1987
Place of Marriage	NEWCASTLE, NEW SOUTH WALES
5 PREVIOUS CHILDREN OF RELATIONSHIP	CALAB G. DECEASED PATRICK A. DECEASED
6 INFORMANT(S)	
Name	C. FOLBIGG
Address	9 DOWER CLOSE, THORNTON 2322
	Father
7 REGISTERING AUTHORITY	
Name	B. A. Flett, Principal Registrar
Date	27 October 1992
8 ENDORSEMENT(S)	
Not any	

39

Before accepting copies, sight unaltered original. The original has a coloured background.

REGISTRY OF BIRTHS
DEATHS AND MARRIAGES

SYDNEY **17 Jan 2000**

I hereby certify that this is a true copy of particulars recorded in a Register in the State of New South Wales, in the Commonwealth of Australia

Registrar

JOHN HUNTER HOSPITAL PERINATAL DATABASE

Early Childhood Clinic Report - To be forwarded to the District Clinic with signed consent
or Place this sheet in the Baby's Personal Health Record

Surname: FOLBIGG
MRN: 472487
Address: 9 DOWER CLOSE,
THORNTON 2322.
Telephone: 664489

First names: KATHLEEN MEGAN
Date of Birth: 14/06/67
Marital status: married
Born in: Australia

ADMISSION: Admitted at 04:20 on 14/10/92. From: booked JHH - Private. Gestation: 39 weeks

HISTORY: Age: 25. Gravida 3 Para 2. Neonatal deaths 2. The last pregnancy occurred in 90.

THIS PREGNANCY: EDC 15/10/92 by US Scan before 22 weeks. Antenatal care by a specialist obstetrician from 8 weeks gestation. There were 1 admissions to hospital in the antenatal period. Early bleeding - single episode at <12 weeks. Threatened premature labour.

Tests: Blood group A positive, Blood group antibodies Nil, Rubella immune, Hepatitis B surface antigen negative, Syphilis serology negative.

Labour Onset: Commenced spontaneously at 03:30 on 14/10/92.

Course: No drugs or analgesics were used. Anaesthesia - perineal infiltration with local anaesthetic.

DELIVERY: First stage 2 hours, 0 minutes. Second stage 13 minutes. Presentation vertex OA. Baby delivered by spontaneous maternal effort by Specialist Obstetrician (DR. HOLLAND).

THIRD STAGE: The mother sustained a 1st degree tear. Placenta delivered by cord traction - complete. Third stage 3 minutes with Syntocinon. Total estimated blood loss 150 ml.

INFANT: Singleton liveborn FEMALE at 05:43 on 14/10/92 Birthweight was 3020 gms and head circumference 34.5 cms. Place of Birth = Hospital (JHH). Baby was discharged home on postnatal day *.

LOCAL DOCTOR: Dr MARLEY, KING STREET, NEWCASTLE 2300.

Blood sample for metabolic test (Guthrie) collected on 17/10/92. Baby was ~~breast~~/bottle feeding on discharge.
Weight of baby at discharge = 2940 grams (bare) or 3220 grams (clothed - no shawl).

Postnatal Problems (Mother or Baby) nicnc

Special Follow-up Clinic on ___/___/___ at _____ am/pm

I HEREBY GIVE PERMISSION FOR THIS INFORMATION TO BE SENT TO THE EARLY CHILDHOOD CENTRE.

Mother's signature: _____ Date: _____

NEW SOUTH WALES

BIRTHS, DEATHS AND MARRIAGES REGISTRATION ACT 1995

DEATH CERTIFICATE

REGISTRATION NUMBER
28534/1993

1 DECEASED	Family Name	FOLBIGG
	Christian or Given Name(s)	Sarah Kathleen
	Date of Death	30 August 1993
	Place of Death	9 Dower Cl, Thornton
	Sex and Age	Female 10 months
	Place of Birth	New Lambton Heights, NSW
	Period of Residence in Australia	Life
	Place of Residence	9 Dower Cl Thornton 2322
	Usual Occupation	-
	Marital Status at Date of Death	-
2 MARRIAGE(S)	Place of Marriage	
	Age when Married	
	Full Name of Spouse	
3 CHILDREN	In order of birth names and ages	
4 PARENTS	Father's Name	Craig Gibson FOLBIGG
	Mother's Name	Kathleen
	Mother's Maiden Family Name	MARLBOROUGH
5 MEDICAL	Cause of Death and Duration of last illness	Sudden infant death syndrome
	Name of Certifying Medical Practitioner or Coroner	Inquest dispensed with at East Maitland J.D. Harwood, Coroner
6 BURIAL or CREMATION	Date	02 September 1993
	Place	Newcastle Crematorium
7 INFORMANT	Name	C.G. Folbigg
	Address	9 Dower Cl Thornton 2322
	Relationship to deceased	Father
8 REGISTERING AUTHORITY	Name	B. A. Flett, Principal Registrar
	Date	15 September 1993
9 ENDORSEMENT(S)		Not any

10

Before accepting copies, sight unaltered original. The original has a coloured background.

REGISTRY OF BIRTHS
DEATHS AND MARRIAGES

SYDNEY **17 Jan 2000**

I hereby certify that this is a true copy of particulars recorded in a Register in the State of New South Wales, in the Commonwealth of Australia

Registrar

90

Death D15/1993 East Maitland IN

P79A

REPORT OF DEATH TO CORONER

NOTE:
(1) This form should be prepared in quadruplicate in all cases where a death is reported to the Coroner. The orginal and two copies should be forwarded to the Coroner. All statements in duplicate should be lodged with the Coroner no later than 28 days after receipt of inquest notice.
(2) The full name and address of all persons and the registered number of all motor vehicles concerned should be indicated.

Maitland — Police Station
North — Region
30 August 19 93

The Coroner, East Maitland

Morgue Register/Book No. _____

Death of Sarah Kathleen FOLBIGG (Christian Name) (Surname) Sex: F Age: 11 month

Address 9 Dower Close Thornton 2322 Marital State single

Time and Date of Death 1.30am Monday 30 August 1993

Place of Death 9 Dower Close Thornton 2322

By whom found Kathleen Megan FOLBIGG Address 9 Dower Close Thornton 2322

By whom reported to Police Deborah MARTIN Address Beresfield Ambulance Kendall St

By whom last seen alive Kathleen Megan FOLBIGG Address 9 Dower Close Thornton 2322

When last seen alive 12-12.30am Monday 30 August 1993

Time and date reported to Police 2.45am Monday 30 August 1993

Deceased a native of (Country) Australia

Occupation child
(If pensioner state type and authorities informed)

Name and Address and Telephone No. of nearest relative Kathleen Megan FOLBIGG and Craig Gibson FOLBIGG of 9 Dower Clse Thornton Relationship to Deceased ph.049-664489. Parents.

Name and Address of identifying person John Francis FOLBIGG (uncle)

Method of Identification (Visual, Dental, F/prints) Visual.

Chain of Identification [i.e. Relative or Friend (name) to Police (name) to other Police (name)] Relative John Francis FOLBIGG to police Senior Constable SAUNDERS.

Criminal Charges Preferred (Yes/No) - Details No

Property and clothing found on and with deceased _____

Miscellaneous Property Book Reference _____

How Property and clothing disposed of and on whose authority _____

Narrative of circumstances under which death took place. - The child is the families 3rd natural child. The previous 2 both beings SIDs victims within the past 5 years. The child had been in good health apart from of flu or cold type virus which had been treated by Dr Chris MARLEY of King and Perkins St Newcastle with prescription 'Flopen 125' 125mg/5ml (Flucloxacillin) prescribed on 180893 and last taken by the child about 26 or 270893 course not completed. The child had been an electronically monitored high risk SIDs candidate but not monitored for about the past week. The child was eating normally all foods similar to parents and last ate about 5.30pm 290893. The child was put to sleep in a single bed in the parents bedroom about 9pm 290893. Parents went to be about 9.30 or 10pm and child seen to be snoring. The mother heard the child turning over in its sleep about 12 or 12.30am 300893. The mother got up to toilet at 1.30am 300893 and could not hear the child breathing, turned on the bedroom light and saw that the child had blue colour to skin on face and a dischage from the nose. The mother roused the father who commenced CPR, ambulance from Beresfield attended and were unable to revive child.

If any previous illness, and deceased seen by doctor, particulars should be given. Where treated by a doctor a note should be obtained giving particulars of treatment of such doctor. If died within 24 hours of Anaesthetic - Forms A and B required from hospital and indicated at the start of the Narrative.

Signature. S.C.SAUNDERS. (Print Name)

Rank: Senior Constable

Annual leave 260993-091093. 051293-101293.

91

Coroner's Consent

ANNEXURE A

OFFICE COPY

Ambulance Service of New South Wales

LEVEL 1 2 3 4 (6)
REGION HR

TREATMENT REPORT

DATE	TIME	CASE NUMBER	PATIENT	SEX	AGE
30/8/03	24:23	Q 001	1 of 1	F	10/12

CALL TYPE	AUTHORITY	STATION/SECTOR	AUDIO NUMBER	RESPONSE	CARRIAGE
CA	PUBLIC CALL	Q911	133	2	C1

TIME BOOKED	TIME OUT	LOCATION	DEPART	DESTINATION	CLEAR
01:24	01:25	C1:48			03:07

PATIENT DETAILS

CODE	TITLE	SURNAME	FIRST NAME	DOB
3	MISS	FOLBIGG	SARA	14.10.92

ADDRESS: 9 BOWER CL. THORNTON — CODE: 2322

STRETCHER B / WALKING W / WHEELCHAIR C

☐ W/COMP ☐ 3RD PARTY

CHARGE CODE	AMOUNT	CHARGE KM
2		52

DESTINATION DETAILS

CODE: E899 — TITLE — SURNAME/BUSINESS — INITIALS

REGION NUMBER/WARRANT NUMBER — ADDRESS — CODE

OCCUPATION — EMPLOYED BY

PUNCTUAL

ODO IN	64117
ODO OUT	64065
TRIP KM	52

FROM: RES. CODE: 2322

TO — CODE: E899

HISTORY [State chief complaint first]

? SIDS. CODE 4

ASSIST CAR JSO 'c/ ? SIDS. O/A TOLD MOTHER FOUND CHILD 10MTHS. OLD (WEIGHT 10KG) IN BED NOT BREATHING. COMM. C.P.R. TILL ARRIVAL OF JSO. (PARENTS 3RD SIDS)

RE CASE No. Q604.

EXAMINATION

AIRWAY	☑ Clear	☐ Obstructed	
BREATHING	☐ Present	☐ Absent	
	☐ Shallow	☐ Deep	
CIRCULATION	☐ Present	☑ Absent	
Buccal Mucoses	☐ Pink	☐ Blue	☑ Pale
Skin temp.	☐ Normal	☑ Cold	☐ Hot
Sweating	☑ Nil	☐ Mod.	☐ Profuse
Vomiting	☑ Nil	☐ Small	☐ Large
Fitting	☑ Nil	☐ Yes	Number
Burns	☑ Nil	☐ Superficial	%
		☐ Deep	%
Blood Loss (External)	☑ Nil	☐ Under 500ml	☐ Over 500ml

Site

BURNS

F Fracture H Haemorrhage B Burn
L Lacerations T Tenderness P Pain
A Abrasion C Contusion
S Swelling D Dislocation

Specific observations ☐ Nil

8

Breath Sounds: VENT R-L.

X Position of Patient
→ Point of Impact
☒ Damaged Area
Seat Belt Not/Worn

FRONT

Estimated Impact Speed High Medium Low

ROAD USER: 1 Driver 2 Passenger 3 Motorcyclist 4 Pedal 4 Pedal Cyclist 5 Pedestrian 6 Others

Time	Pulse	Blood Pressure	Capillary Filling	Respiration Rate	Respiration Effort	Level of Cons.	Total score	Pupils Size T	Pupils Size R	Pupils Size L	Pupils React. R	Pupils React. L	GLASGOW COMA SCALE Eye Opening	Verbal Response	Motor Response	GCS Score
01:33	NIL	NIL	NIL	NIL		1		3	3	3	–	–	1	1	1	3
01:50	NIL	NIL	NIL	NIL		1		3	3	3	–	–	1	1	1	3
02:05	NIL	NIL	NIL	NIL		1		3	3	3	–	–	1	1	1	3

Vital Signs Points		Add Vital Sign Points
First	0	0 + 0 + 0 + 0 + 0 + 1 – 1
Since	0	0 + 0 + 0 + 0 + 0 + 1 – 1

Difference Between Total Score: ±

TREATMENT BEFORE ARRIVAL

0 Nil 1 CPR within 3 mins.
2 First Aid 3 Medical

Patient's state prior to arrival
A Conscious C Unknown
B Unconscious Mins.

PREVIOUS ILLNESS

☐ Nil ☐ Renal
☐ Unknown ☐ Stroke
☐ Hypertension ☐ Epilepsy
☐ Cardiac ☐ Psychiatric
☐ Respiratory ☐ D.I.T.
☐ Diabetes
☑ Other FULL

Allergies ☐ Nil ☐ Unknown

Present Medication ☐ Nil ☐ Unknown

Comments: I.O. INFUSION.

TREATMENT

AIRWAY: 1 Suction; (1) Oral Airway; 2 Nasal Airway; ☐ ETT

BREATHING: 1 RM; 2 RS; 3 ____(other); (1) IPPV

CIRCULATION: (1) Cardiac Compression

POSTURE: 1 Lateral; (2) Supine; 3 Sitting; 4 Legs Elevated

O_2 THERAPY: 1 High Conc. Mask; 2 Nasal Prongs; 3 28% Venimask; 1 14L 2 8L 3 4L; 1 Demand Valve

HAEMORRHAGE CONTROL: ☐ Dressing ☐ Elevation ☐ Pressure

IV CANNULATION: 1 Type ____; 2 Unsuccessful; 3 In Situ; Local given ☐ No ☐ Yes

INTUBATION: 1 Type ____; 2 Unsuccessful; 3 In Situ

ANALGESIA: E Entonox N Narcotic; 1 Not Effective 2 Part Effective 3 Effective

SPLINTS: 1 Hare 2 Thomas 3 Air 4 Cervical Collar; 5 KED 6 Other 7 Donway

MAST Suit - Inflated: 1 Under 500ml 2 Over 500ml (External blood loss)

TOURNIQUETS: 1 Rotating 2 Arterial 3 Lymphatic Time On ____

STRETCHER: 1 Carry Chair 2 Back Board 3 Jordon Frame; 4 Carry Sheet 5 Other

KITS: 1 Poison 2 Burns 3 Maternity 4 Other

BLOOD SAMPLE TAKEN: Glucose Est. ____ Blood Type & Cross Match ____

TYPENEX No.

RHYTHMS MONITORED [abbreviations in heavy print]

1 Sinus Rhythm
2 Sinus Bradycardia
3 Sinus Tachycardia
4 Perm. Atrial Conf.
5 Supra Vent. Tachy
6 Atrial Fib / Flut.
7 Idioventr. Rhythm
8 Asystole
9 Pacemaker Rhythm
10 Prem. Vent. Cont. Frequency ____ Runs ☐ R on T ☐ Multifocal ☐
11 Vent. Tachycardia
12 Vent. Fibrillation ☐
13 1° Heart Block
14 2° Heart Block
15 3° Heart Block
16 Junctional
17 Multiple Arrhyth.
18 Elect Mech. Dis.

TIME	ECG	TREATMENT	DOSE	ROUTE	SHOCK	RESULT
01:50	A	1:10,000 ADRENALINE	8ml	I.O.I.		PRIOR TO ARRIVAL
01:55	A	1:10,000 "	2ml	"		NIL
01:55	A	Na HCO3	10ml	"		"
01:57	A	CAL CHLORIDE	1ml	"		"
02:00	A	1:10,000 ADRENALINE	2ml	"		"
02:05	A	1:10,000 ADRENALINE	2ml	"		"
02:05	A	Na HCO3	5ml	"		"
		HARTMANNS		"		FLUSH

COMMENTS: KV. IV TRAINEE R. AVERY

CONDITION AT DESTINATION: ☐ IMPROVED ☒ UNCHANGED ☐ DETERIORATED

OFFICER I DRIVING: L. BISHOP

OFFICER 2: R. FOXFORD

SIGNATURE — ID# — SIGNATURE: Foxford — ID# 1010

CHILD/UNKNOWN RISKS

	WT. SCORE	BYPASS SYSTEM
AIRWAY		CATEGORY
BP		
CNS		DECISIONS FROM TRAUMA CHECKLIST
FRACTURES		
WOUNDS		
TOTAL		SUBTRACT 8 POINTS

ATTACH ECG STRIPS HERE

TRAUMA

1	BYSTANDER
1	Effect
	ROAD USER
	Severity
2	TREATMENT before arrival
8	Conscious/Unconscious
	SUCTION
	AIRWAY
	BREATHING
1	IPPV
1	CIRCULATION
2	POSTURE
	O_2 THERAPY
	Line
	DEMAND VALVE
1	CANNULATION
	INTUBATION
	ANALGESIA
	Effects
	SPLINTS
	SPLINTS
	MAST SUIT
	STUDY 1
	STUDY 2
	STUDY 3
3	ECG
	ECG
3	DC SHOCK
201	FLUID / DRUG
1	Units
293	FLUID / DRUG
1	Units
211	FLUID / DRUG
1	Units
217	FLUID / DRUG
1	Units
	FLUID / DRUG
	Units
19	PROTOCOL
16	PROTOCOL
	PROTOCOL
A	HAZARDS
	TRIAGE TOOL
	TRANSPORT DECISION
	MAIN COMPLAINT
	OTHER COMPLAINT
	OTHER COMPLAINT
	P.T.S. c.d.s. STROKE

ANNEXURE A

CASE No. 242 001

C.A.D. 133

TIME RECEIVED 01:25.

ETD ADVISED

STATION A

TIME OUT 01:25.

SUBURB Thornton

1 c/s 01:48.

OFFICERS ON CASE R. Foxford.

2 Relief 02:06

3 L. BISHOP.

3 Code 4 c/s 02:46.

4 R. AVERY.

4 03:07

5

5

6

6

7

7

8

8

CALLED BY

PHONE

NAME OR LOCATION

9 Dowler Close

CROSS STREET OR LANDMARK

NATURE OF EMERGENCY ? BABY S.I.D.S

No. OF INJURED

TO CODE (4) S.I.D.S W/O TRANSPORT.

Paramedic response	Yes	
Breathing difficulty		
Chest pain		
Severe bleeding		
Severe injury/burns		
Fitting		
Others		

RESCUE VAN

POLICE NOTIFIED 02:08

RECEIVED BY RQA

CONTROL OFFICER 1 R J Allan

CONTROL OFFICER 2

N.S.W. AMBULANCE SERVICE CALL SCHEDULE

GREG TAPP PRINTING (049) 61 6269

CASE No. 12 604

CAR No. 50

TIME RECEIVED 01:24

ETD ADVISED

STATION J.

TIME OUT 01:24

SUBURB Thornton.

1 c/s 01:30.

OFFICERS ON CASE D. MARTIN.

2 c/s 03:59.

3

3 Hosp 04:14.

4

4 (R) 04:23.

5

5 04:38.

6

6

7

7

8

8

CALLED BY mother.

PHONE 666489

NAME OR LOCATION BABY HEWETT.

9 DOWLER close.

CROSS STREET OR LANDMARK

NATURE OF EMERGENCY S.I.D.S.

No. OF INJURED

TO M.D. Hosp

Paramedic response	Yes	
Breathing difficulty		
Chest pain		
Severe bleeding		
Severe injury/burns		
Fitting		
Others		

RESCUE VAN

POLICE NOTIFIED 02:08.

RECEIVED BY RQA

CONTROL OFFICER 1 R J Allan

CONTROL OFFICER 2

N.S.W. AMBULANCE SERVICE CALL SCHEDULE

GREG TAPP PRINTING

ROYAL PRINCE ALFRED HOSPITAL

NSW INS FORENSIC MEDICINE
PO BOX 90
GLEBE NSW 2037

eJ

Requested by: HILTON

Patient No: (2247)0045824

Name: FOLBIGG, SARAH
Sex/Age: FEMALE UNKNOWN UNK

Location: NSW FORENSIC MED

DEPARTMENT OF MICROBIOLOGY

Drs Benn, Kappagoda & Macleod Enquiries: 516 8278

Virus

VIRUS CULTURE MB-93-53691 COLLECTED:31AUG93
SOURCE: LUNG RECEIVED: 31AUG93 1458

FINAL REPORT

NO VIRUS ISOLATED AFTER 10 DAYS. NO FURTHER REPORT WILL BE ISSUED UNLESS GROWTH OCCURS IN THE NEXT 2 WEEKS.

Miscellaneous

TISSUE/BIOPSY CULTURE MB-93-53691 COLLECTED:31AUG93
SOURCE: LUNG RECEIVED: 31AUG93 1458

FINAL REPORT

PROFUSE COLIFORM
PROFUSE STREPTOCOCCUS, ALPHA HAEMOLYTIC
SCANTY STAPHYLOCOCCUS AUREUS

TISSUE/BIOPSY CULTURE MB-93-54258 COLLECTED:02SEP93
SOURCE: SPLEEN RECEIVED: 02SEP93 1109

FINAL REPORT

MODERATE COLIFORMS OF 3 COLONIAL TYPES

TISSUE/BIOPSY CULTURE MB-93-54259 COLLECTED:02SEP93
SOURCE: TISSUE RECEIVED: 02SEP93 1109
LARGE INTESTINE

FINAL REPORT

MODERATE COLIFORMS OF 3 COLONIAL TYPES
MODERATE ENTEROCOCCUS FAECALIS
MODERATE DIPHTHEROIDS

TISSUE/BIOPSY CULTURE MB-93-54260 COLLECTED:02SEP93
SOURCE: TISSUE RECEIVED: 02SEP93 1109
SMALL INTESTINE

FINAL REPORT

PROFUSE COLIFORMS OF 2 COLONIAL TYPES

ROYAL PRINCE ALFRED HOSPITAL

NSW INS FORENSIC MEDICINE
PO BOX 90
GLEBE NSW 2037

Requested by: HILTON

Patient No: (2247)0045824
Name: FOLBIGG, SARAH
Sex/Age: FEMALE UNKNOWN UNK
Location: NSW FORENSIC MED

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Miscellaneous

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SOURCE: LUNG RECEIVED: 31AUG93 1458

FINAL REPORT

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PROFUSE STREPTOCOCCUS, ALPHA HAEMOLYTIC
SCANTY STAPHYLOCOCCUS AUREUS

TISSUE/BIOPSY CULTURE MB-93-54258 COLLECTED:02SEP93
SOURCE: SPLEEN RECEIVED: 02SEP93 1109

FINAL REPORT

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SOURCE: TISSUE RECEIVED: 02SEP93 1109
SMALL INTESTINE

FINAL REPORT

PROFUSE COLIFORMS OF 2 COLONIAL TYPES

Printed: 21SEP93 0327 *** End of Report *** MICROBIOLOGY

Sarah Kathleen **FOLBIGG** PM93/1673 (EJ)

NSW INSTITUTE OF FORENSIC MEDICINE

42-50 PARRAMATTA ROAD
PO BOX 90
GLEBE NSW 2037
PHONE (02) 660 5977
FAX (02) 552 1613

CORONERS ACT, 1980

Name: Sarah Kathleen Folbigg

PM Number: 93/1673

I, John Millar Napier Hilton, a registered medical practitioner, practising my profession at the New South Wales Institute of Forensic Medicine in the State of New South Wales, do hereby certify as follows:-

At 0800 hours, on the 31st day, of August 1993 at Sydney in the said State, I made a post mortem examination of Sarah Kathleen Folbigg.

The body was identified to J. Mullan of the New South Wales Institute of Forensic Medicine by Const Saunders of Maitland Police Station, as that of Sarah Kathleen Folbigg aged about 11 months.

The body was identified to me by the wristband marked E.45824

The forensic assistant in this case was Mr. S. Bannister.

Upon such examination I found:-

WEIGHTS:

Body weight	9.44 kgs
Brain	965 g
Heart	54 g
L.Lung	68 g
R.Lung	92 g
Liver	394 g
L.Kidney	34 g
R.Kidney	34 g
Spleen	32 g
Stomach & contents	80 g
Thymus	42 g
Thyroid	4 g

Crown heel	71 cm
Crown rump	47 cm
Chest	45 cm
Head circumference	45.5 cm

EXTERNAL EXAMINATION

The body temperature was 25° by the rectal route on admission to the Mortuary at 11.00 am on 30/8/93.
Rigor mortis was generalised.
Gravitational lividity was present in the posterior dependent distribution.
There was a minor degree of lividity present on the right side of the face with blanching of the left cheek and the left side of the forehead.

There was a surgical device issuing from the right knee.
A 1.5 cm scratch was present on the antero lateral aspect of the right upper arm.
Palmar skin creases normal.
Planter skin creases normal.

The body was that of a well nourished clean Caucasoid female.
There was minor abrading and drying of the lips.
The frenula were normal.

There were two tiny punctate abrasions present one immediately below the lower lip on the left side, the other slightly to the left side of the midline of the chin.

INTERNAL EXAMINATION:

The skull and membranes appeared normal.
The brain appeared normal on external examination and has been retained intact for full formalin fixation to be examined and reported on by the Department of Neuropathology.
Portion of higher cervical cord taken.
Middle ears were normal.
CSF was clear.
Tongue was in mouth.
Fauces were normal.

The uvula although of normal proportions appeared somewhat congested/haemorrhagic in its anterior surface.
When viewed at postmortem it was placed anterior to the epiglottis producing an obstructive element in the airway.
The epiglottis itself appeared relatively normal.

There was gastric content present in the trachea and major bronchi.
The larynx, trachea and major bronchi themselves appeared intrinsically normal.

Thoracic cavity:

Layers of the pleura separate.
No pleural effusions present.
Both lungs showed focal areas of collapse of a geographic pattern.
There was an occasional petechial haemorrhage present.
There was minor congestion and minimal oedema.
Pulmonary circulation appeared unimpaired.

The heart was normal in size shape and location.
Layers of the pericardium separate.
No pericardial effusions present.
There was a very occasional petechinum mesenterypresent on the epicardium.
Atria normal.
Intra atria septum was intact and the foramen ovale was firmly closed.
There was the usual cribriform multi focal pro patencies in the intraventricular septum.
The ventricular myocardium appeared normal.
Leaves and cusps of the various valves were healthy.
The great vessels were healthy and normally formed and distributed.

The thymus was normal in size shape and location.
There was an occasional petechial haemorrhage on the surface and within the substance of the gland.

Abdominal cavity:

Layers of the peritoneum separate.
No free intra peritoneal fluid found.
Abdominal organs were in the usual locations.

The stomach contained a moderate quantity of curdled milk ? eggwhite. It has been retained intact for examination in the Division of Analytical Laboratories.
Duodenum, Small and large intestine healthy.
Healthy appendix present.

Liver appeared normal on external examination and on serial sectioning.
The gall bladder was thin walled and contained a tiny quantity of brownish bile.
No calculi present.
Bile ducts appeared patent.

Pancreas appeared normal on external examination and on serial sectioning.
Spleen was normal in size shape and location.
Serial sectioning revealed no abnormality.
There was modest cervical and mesenteric lymph node enlargement.
Bone marrow appeared to be of normal cellularity and distribution.
Suprarenals normal in size shape and location.
Serial sectioning revealed no abnormality.

Kidneys normal in size shape and location.
In each case the capsules stripped readily revealing a smooth surface.
On coronal sectioning normal internal architecture was observed.
Pelves and ureters normal.
Bladder was normal and empty.
Genital organs were juvenile.

Bones, joints, skeletal muscles:

No abnormality detected.

Specimens retained:

Relevant tissues for histological examination.
Blood, liver, stomach and contents, bile to toxicology.
Representative samples for microbiology.
Spleen for D.N.A.
Liver for biochemistry.
Vitreous humor & CSF for biochemistry..

Sarah Kathleen **FOLBIGG** PM93/1673 (EJ)

PATHOLOGY SUMMARY

1. FOCAL PULMONARY COLLAPSE

2. MODEST PULMONARY CONGESTION AND MINIMAL OEDEMA

3. OCCASIONAL PETECHIAE ON PLEURA, EPICARDIUM AND ON AND IN THYMUS

4. CONGESTED ? HAEMORRHAGIC UVULA LYING ANTERIOR TO THE EPIGLOTTIS

5. ASPIRATION OF GASTRIC CONTENT (?ARTIFACTUAL)

In my opinion, based on what I have observed myself, my experience and training, and the information supplied to me:

A. Time and date of death: 1.30 am 30/8/93

B. Place of death: 9 Dower Close Thornton

C. Cause of death:

1. **DIRECT CAUSE:**
Disease or condition directly leading to death:

(a) **S.I.D.S.**

ANTECEDENT CAUSES:
Morbid conditions, if any, giving rise to the above cause, stating the underlying condition last:

(b)

(c)

2. **Other significant conditions contributing to the death but not relating to the disease or condition causing it:**

Sarah Kathleen **FOLBIGG**

PM93/1673 (EJ)

TO THE STATE CORONER,

(Signature) .....

SYDNEY

(Date) 25TH NOVEMBER, 1993

Microscopic examination:
Sarah Kathleen Folbigg PM 93/1673

Sections of uvular shows marked vascular congestion particularly of the pharyngeal aspect adjacent to the base.
One section of larynx shows a light mixed lymphocytic inflammatory infiltrate deep to the respiratory epithelium.

Section of salivary gland shows two small acute inflammatory foci in the interstitium
No viral occlusions were seen.

Tongue was normal.

Section of diaphragm including central tendon shows two foci of individual muscle fibrillary degeneration.

Section of spleen shows focal congestion.

Section of liver shows some vesiculation of hepatic nuclei.

Sections of adrenal were normal.

Sections of kidney shows some medullary congestion only.

Interstinal section shows autolysis only.

Section of parotid is normal.

Section of pancreas is normal.

Section of thyroid is normal.

Section of tonsils shows debris within the crypts.

Section of lymph nodes shows area of reactive change.

Section of thymus normal.

Section of heart were normal.

Section of lungs show some congestion and oedema.
In one section there is a light interstitial acute inflammatory infiltrate which could be seen around the occasional bronchiole.
Further section of lung shows multiple polymorpho nuclears within the lymphoid deposits and again some interstitial infiltration.

DEPARTMENT OF HEALTH NSW
INSTITUTE OF FORENSIC MEDICINE

42-50 Parramatta Road
PO Box 90
Glebe NSW 2037
Phone (02) 660 5977
FAX: (02) 552 1613

Name: Sarah Kathleen FOLBIGG

PM NO: 93/1673

Macroscopic Description of Brain:

The brain is examined after fixation in formalin. The dura mater and dural sinuses are normal. The leptomeninges are thin and transparent. The vessels at the base of the brain have a normal architectural pattern with no atheroma. The external surface of the cerebrum, cerebellum and brainstem appears normal. The cranial nerves appear normal.

The cerebrum is sectioned in the coronal plane in 10 mm slices. No abnormalities are seen on the cut surfaces of the cerebral cortex, hippocampus, white matter, basal ganglia, diencephalon, or ventricles.

The cerebellum is sectioned in the sagittal and parasagittal planes. No abnormalities are seen on the cut surfaces of the cerebellar cortex, white matter, or dentate nuclei.

The brainstem is sectioned in the transverse plane in 5 mm slices. No abnormalities are seen on the cut surfaces of the midbrain, pons or medulla oblongata.

Macroscopic Diagnosis:

Normal brain.

Microscopic Brain Examination

Sections from brain stem and cerebrum shows no abnormalities.

Sarah Kathleen **FOLBIGG** PM93/1673 (EJ)

Microscopic Diagnosis

Normal Brain.

Dr R Pamphlett
NEUROPATHOLOGIST

25

Expert Certificate in the matter of : the death of Laura Elizabeth FOLBIGG

Place Adamstown N.S.W. Date : 09.03.99

Name : Christopher George Marley

Address : 24 Morgan St
Adamstown 2289

Tel. No : 02 49528283

Occupation : Medical Practitioner / General Practitioner . STATES:-

EXPERT CERTIFICATE
Section 177, Evidence Act 1995 No. 25

1. This statement made by me accurately sets out the evidence which I would be prepared , if necessary , to give in court as a witness. The statement is true to the best of my knowledge and belief and I make it knowing that , if it is tendered in evidence, I shall be liable to prosecution if I wilfully stated anything which I know to be false or do not believe to be true .

2. I am 41 years of age.

3. I hereby certify:
My full name is Christopher George Marley
My contact address is 24 Morgan St Adamstown 2289.
I have a specialised knowledge based on the following training, study and experience :-
In 1980 I graduated with a Bachelor of Medicine and Surgery with Honours from the University of NSW.
In 1998 I attained the Fellowship to the Australian College of General Practice .
Between 1981 to 1986 I worked as a resident medical officer and a medical registrar of a number of NSW Public Hospitals and in Swansea , United Kingdom .
From 1986 until to date I have worked as a General Practitioner in a number of General Practices as a vocationally registered General Practitioner.

witness J.P. Ward
J. P. WARD

Signature C Marley

4. I have examined Kathleen Megan Folbigg [d.o.b 14.06.67] on the 8.7.87, 27.11.87 , 12.01.88 , 19.01.88 , 23.02.90 ,12.11.90 , 27.12.90 , 08.01.91 ,15.07.91 , 17.01.92 , 07.02.92 , 06.01.93 , 24.09.93 ,11.05.94 . Mrs Folbigg was last seen at the General Practice I worked at that time was on the 18.01.95 by Dr S . Crawley .

During these visits Mrs Folbigg was treated for hand dermatitis, contraception , pregnancy diagnosis , back pain ,gynaecological problems, ear pain and on the 24.09.93 counselling about Sarah Folbigg's death .

I have examined Master Patrick Folbigg [d.o.b. 03.06.90] on the 24.09.90 , 03.11.90 , 12.11.90 , 27.12.90 and the 25.01.91.

I saw Patrick for vaccinations ,mild viral infections , and mild childhood illnesses . His last visit to me was on the 25.01.91 , this visit was for scripts, and for treatment of a fungal skin rash . Patrick had been under the care of a paediatric neurologist , Dr Ian Wilkinson for a seizure disorder and visual disturbance secondary to an occipital infarct.

I have examined Sarah Folbigg [d.o.b. 14.10.92] on 04.03.93 ,17.03.93 ,20.05.93 and on the 16.06.93. Sarah was last seen at King ST Group Practice on the 26.08.93 .

During these visits Sarah was given 3 of her childhood vaccinations , treated for a viral infection of mild severity , and for a fungal skin rash. On the 26.08.93 she was seen by Dr Peter Hopkins for a croupy cough . This was her last recorded visit at this practice.

I have no recollection of ever meeting or treating Laura Folbigg.

When collecting the medical records from the practice I used to work at I did not receive any records pertaining to Caleb Folbigg .In my opinion this was because he died before attending this practice as he was only 19 or 20 days old when he died.

5. Based wholly or substantially on the above knowledge , I am of the opinion that

1. To loose four children to SIDS must be devastating to the family and must be regarded as an extremely rare event in the world.
2. My consultations of the Folbigg children , Sarah and Patrick were typical of many other consultations with children of the same age. The only difference being Patrick's visual problems and seizure disorder.[both problems though related are rare in General Practice .] .
3. I saw no sign of neglect on either child .
4. Mrs Folbigg impressed me as a caring and concerned parent as did her husband Craig.
5. Mrs Folbigg and Mr Folbigg never gave me reason to suspect that either person had a significant psychiatric disorder.[my last recorded consultation with Mr Folbigg was on the 28.11.89.]
6. On 20.12.93 Professor J. M. N. Hilton[director of the N.S.W. Institute of Forensic Medicine] felt Mr Folbigg may suffer from Obstructive Sleep Apnoea .

Witness J.P. Ward J.P. HARD Signature

New South Wales Police P.190
v3.0

STATEMENT in the matter of:

Place: Coffs Harbour Police Station
Date : 8 October 1999

Name: Deborah Ann MARTIN
Address: 23 Clarence St, Port Macquarie 2444 **Tel. No.:** 0265 838010
Occupation: Ambulance Officer **STATES:-**

1. This statement made by me accurately sets out the evidence which I would be prepared, if necessary, to give in court as a witness. The statement is true to the best of my knowledge and belief and I make it knowing that, if it is tendered in evidence, I shall be liable for prosecution if I have wilfully stated in it anything which I know to be false, or do not believe to be true.

2. I am 36 years of age.

3. My date of birth is the 29.4.63.

4. I have been employed by the New South Wales Ambulance Service, since 1984. I currently hold the title of Quality Manager. I am currently on Maternity Leave. My duties, as Quality Manager, include managing programs for the Ambulance Service and the movement towards accreditation.

5. When this incident happened, I was the Station Officer at the Beresfield Ambulance Station. There were four other Ambulance officers, employed at Beresfield. As Station Officer, I was responsible for the overall managing of the Ambulance station. I was the most senior Ambulance Officer at that station.

6. At the time of this incident, I had completed a 10 week course in Advanced Life Support and I had a degree in Health Science, from the Charles Sturt University, Bathurst.

Witness: _____ **Signature:** _____

STATEMENT (continued) in the matter of:
Name: Deborah Ann MARTIN

7. About 1.25am on the morning of the 30.8.93, I was called to this incident, by the Ambulance Controller in Newcastle, via the phone at Beresfield Ambulance Station. The Controller told me that the job, involved a possible Sids death (Sudden Infant Death Syndrome), involving a young child.

8. About 1.30am, I arrived at the residential premises of 9 Dower Close, Thornton. I was a single unit Ambulance officer.

9. When I first arrived at the premises, the interior lights were on. I couldn't say if there were any vehicles parked at the house. I was met at the front door by Mrs. Kathy FOLBIGG. She was sobbing, when I first met her. She just directed me, to the main bedroom.

10. The house was a single storey brick house, with a tiled roof. From the front door, there was an open loungeroom and from that loungeroom, there is a small hallway, which leads into the main bedroom. There was no one in the loungeroom, when I walked through.

11. When I walked into the bedroom, I saw Mr. Craig FOLBIGG, performing CPR (Cardio Pulmonary Resuscitation) on the child, Sarah FOLBIGG, on the floor in the bedroom. From memory, Craig FOLBIGG may have been performing CPR on Sarah FOLBIGG on the bed, when I walked in. I then directed him to move Sarah to the floor. This is a little bit different, to what I wrote on the Ambulance Treatment Report Form, Case No. 604 **(ANNEXURE A).**

Witness: _____ Signature: Deborah Martin

12. There was no other person in the bedroom, when I entered, other than Craig FOLBIGG, Kathy FOLBIGG and Sarah FOLBIGG. In that room, there was a double bed, which appeared to have been slept in, as it was unmade. There was no baby's basinet or cot, in that room. From what I can remember, Craig and Kathy, never said anything, when I first entered the room.

13. It appeared to me, that Craig was performing CPR, properly on Sarah, when I walked into the bedroom. I had to return to the Ambulance vehicle to collect more equipment, because I was a single unit officer. When I was out at the vehicle, I called in a Code 2, on the Ambulance radio. This means Cardiac Arrest, so that the Controller can dispatch another Ambulance team, to the scene. I was only out of the bedroom for a couple of minutes. When I left the bedroom, Craig was performing CPR and Kathy, that's what I called Mrs. FOLBIGG, was hovering, around him and Sarah. There didn't appear to me, to be any tension, between Craig and Kathy, when I was present.

14. When I returned to the bedroom, Craig was still performing CPR and Kathy was still standing there, next to him. I couldn't get any IV (Intravenous) access, to either Sarah's arm or leg, so I inserted an Intra Osessous (Needle), into the proximal aspect of her tibia (Leg). I can't remember which leg, I put it into.

15. Sarah was approximately a 10 month old baby. She was fully clothed. She had one of those little Bond brand ski suits on. When I first saw her, she was Cynosed around the mouth (Meaning she appeared blue in that area) and she had mucus and vomit in her mouth. She wasn't breathing.

Witness: _____ Signature: Deborah Martin

16. I gave Sarah Adrenaline and Hartmanns fluid. Adrenaline is for the heart, to basically kick start the heart and the Hartmanns is for fluid replacement, to push the drugs through. I gave Sarah four lots of 200 micrograms of Adrenaline, 1:10,000. I gave Sarah one lot of Hartmanns, approximately 100 mils.

17. Then the paramedics from Hamilton Ambulance Station arrived. Those officers were Louise BISHOP, whom is now ALDERSON and Robert FOXFORD and a Trainee Ambulance Officer, Rodney AVERY.

18. They gave Sarah Calcium Chloride and Bicarbonate. These are cardiac drugs, which I am not allowed to administer. I can't remember if Craig appeared upset or not at that stage, but I do remember Kathy sobbing.

19. At approximately 2.10am, we stopped the drugs and CPR on Sarah, because the child was Asystolic (The child had no electrical activity in her heart). As far as we were concerned, Sarah was then deceased.

20. We told Mr. and Mrs. FOLBIGG, that she had failed to respond to the drug therapy and she was deceased. Craig and Kathy became very upset. I then said to Mrs. FOLBIGG, "Go and get the baby some other clothes." I removed the ski suit from the baby, for the Police. I also said to her, "Where is the last bottle she had? And what food did you give her?" I can't remember what food she mentioned, but she handed me a bottle from the kitchen and I put it in a plastic bag, separate from the ski suit. This was normal procedure. I gave those bags to a Police officer at the scene. I can't remember if he was a uniformed officer or not, or his name.

Witness: _____ Signature: Deborah Martin

21. I had rung the Ambulance Controller from the residence and informed them that the child was deceased and I required the Police. The Controller told me, that there was a lengthy delay before the Police could arrive there and we would have to wait there.

22. I may have taken the sheets, from the baby's cot, but I can't remember. The baby's cot was in another room. I didn't see any other children at the premises. I remember seeing an Apnea monitor in the cot. The Apnea Monitor is like an electric blanket, which the baby lies on top of, which detects when they are breathing or not.

23. Whilst I was waiting for the Police, I had a number of conversations, with Mr. and Mrs. FOLBIGG. I remember Mrs. FOLBIGG saying to me, "We've had two other cot deaths, both were males. One was three weeks old and the other ten months old. They think that I have some sort of genetic abnormality in my male children, so I am surprise that I've lost a girl." Craig said to me, "We've recently moved Sarah from the Apnea Monitor, which we got from the SIDS Foundation. We took her off it, because it kept alarming, she was rolling off it."

24. I thought that this was strange, because if it was alarming, you would think, that they would have taken her for medical advice or treatment.

25. After the Police arrived, we departed from the scene about 3.59am, to Maitland Hospital, arriving at 4.14am. I conveyed the baby in my Ambulance to the Casualty Section, where I handed her over, to the nursing staff.

Witness: _____ Signature: x Deborah Martin

26. In the days following the 30.8.93, I rang Mr. and Mrs. FOLBIGG, to see if it was appropriate to drop around to get a form signed, to prevent them getting an account for the Ambulance transport. I went to the house, around lunch time. Craig answered the door. I went in and gave them the form. We spoke for a few minutes and I then left. They appeared to me, to be visible upset, when I spoke to them on this occasion.

27. Within weeks of the job, I received a Thankyou card and letter from the FOLBIGGS, thanking me, for my assistance.

28. It is a requirement, when you attend a job, that a Patient Treatment Report Form is completed at the first available opportunity. I completed this form at the scene and left a copy at the Maitland Hospital.

29. Attached hereto, marked **ANNEXURE A**, is a three page copy of that New South Wales Ambulance Patient Treatment Report Form, Case No. 604, which I completed, in relation to this incident.

30. On the Patient Treatment Report Form, under the heading Glasgow Coma Scale, I note an error, where I have written the scale as 4,6,5,15. This Glasgow Coma Scale measures the level of consciousness. This should have read 1,1,1,1.

31. I am unable to determine the body temperature of Sarah FOLBIGG, when I first arrived at the scene.

32. In relation to the airway, I marked on the Patient Treatment Form, vomiting NIL, which I altered at the time, to be small, because of the mucus in the babies mouth.

Witness: _____ Signature: _____

8.10.98

3M

ANNEXURE A

OFFICE COPY

Ambulance Service of New South Wales

LEVEL 1 2 3 4 5
REGION N S

TREATMENT REPORT

DATE	JULIAN	CASE NUMBER	PATIENT	SEX
30/8/93	243	0604	1 of 1	F

CALL TYPE	AUTHORITY	STATION NUMBER	RADIO NUMBER	RESPONSE	CATEGORY
CA	PUBLIC	0902	550	1	C1

TIME BOOKED	TIME OUT	LOCATION	DEPART	DESTINATION	CLEAR
0124	0124	0130	0359	0414	0438

PATIENT DETAILS

CODE	TITLE	SURNAME	FIRST NAME	D.O.B.	STRETCHER
B	BABY	FOLEGG	SARAH	14-10-92	S

ADDRESS: 9 DOWER CLOSE THORNTON — CODE: 2322

TWOCOMP ☐ 3RD PARTY ☐ DEBTOR DETAILS

CHGE CODE	AMOUNT	CHGE KM
L		36

DEBTOR CODE ____ TITLE ____ SURNAME/BUSINESS ____ INITIALS ____

PENSION NUMBER/WARRANT NUMBER ____ ADDRESS ____ CODE ____

OCCUPATION ____ EMPLOYED BY ____

CODE IN	876
CODE OUT	840
LOAD KM	30

FROM: Residence — CODE: 2322

TO: MAITLAND HOSPITAL — CODE: 2309

(2) UNC

HISTORY (State chief complaint first) SIDS

0 A found 10 mths ♀ lying on bed / floor down
0 E Pt mother stated that child has not been awake since placing child in bed this PM. Child usually wakes c. Midnight. Mother checks her as she had not eaten. Child parents doing CPR on arrival. Child recently removed from monitor

EXAMINATION

AIRWAY	☐ Clear	☑ Obstructed
BREATHING	☐ Present ☐ Shallow	☑ Absent ☐ Deep
CIRCULATION	☐ Present	☑ Absent
Buccal Mucosa	☐ Pink ☐ Blue	☑ Pale
Skin temp.	☑ Normal ☑ Cold	☐ Hot
Sweating	☑ Nil	☐ Mod. ☐ Profuse
Vomiting	☑ Nil	☑ Small ☐ Large
Fitting	☑ Nil	☐ Yes Number
Burns	☑ Nil	☐ Superficial % ☐ Deep %
Blood Loss (External)	☑ Nil	☐ Under 500ml ☐ Over 500ml

Site .....

INJURIES

F Fracture H Haemorrhage B Burn
L Laceration T Tenderness P Pain
A Abrasion C Contusion
S Swelling D Dislocation

Specific observations ☐ Nil
(1) Supra-ventricular tachycardia
(2) Mildly agitated
(3) This is 3rd episode
Breath Sounds Of some (1) 3 weeks (2) 10 mths

X Position of Patient
→ Point of Impact Nil Children alive.
▨ Damaged Area
Seat Belt Not / Worn
Estimated Impact Speed High Medium Low

ROAD USER: 1 Driver 2 Passenger 3 Motorcyclist
4 Pillion 5 Pedal Cyclist 6 Pedestrian 7 Others

	Time	Pulse	Blood Pressure	Capillary Filling	Respiration Rate	Respiration Effort	L Level of Cons.	Total Score
	0138	0	0	0	0		1	
	0205	0	0	0	0		1	
Vital Signs Points	First	0	Add Vital Sign Points 0 + 0 + 0 + 0 + 1 = 1					
	Final	0	0 + 0 + 0 + 0 + 1 = 1					

Difference Between Total Score 1 0

Pupils — Size T R L; Reactivity R L

GLASGOW COMA SCALE

Eye Opening	Best Motor Response	Verbal Response	Total
4	6	5	15

Eye Opening: 4 Spontaneous, 3 To Speech, 2 To Pain, 1 Nil
Best Motor Response: 6 Obeys, 5 Localises, 4 Withdraws, 3 Abnormal Flexion, 2 Extension, 1 Nil
Verbal Response: 5 Orientated, 4 Confused Conversation, 3 Inappropriate Words, 2 Incomprehensible Sounds, 1 Nil

TABLE 14 or less | 13-11 | 10-8 | 7-5 | 4 or less

TREATMENT BEFORE ARRIVAL

0 Nil 1 CPR within 3 mins. 2 First Aid 3 Medical

Patient's state prior to arrival: A Conscious C Unknown (U) Unconscious Mins. .....

PREVIOUS ILLNESS
☑ Nil ☐ Renal ☐ Unknown ☐ Stroke ☐ Hypertension ☐ Epilepsy ☐ Cardiac ☐ Psychiatric ☐ Respiratory ☐ G.I.T. ☐ Diabetes ☐ Other .....

Allergies ☑ Nil ☐ Unknown .....

Present Medication ☑ Nil ☐ Unknown .....

Comments .....

TREATMENT

AIRWAY: 1 Suction; (1) Oral Airway; 2 Nasal Airway; ☐ ETT

BREATHING: 1 RM; 2 RS; 3 ..... [other]; (1) IPPV

CIRCULATION: 1 Cardiac Compression

POSTURE: 1 Lateral; (2) Supine; 3 Sitting; 4 Legs Elevated

O_2 THERAPY: 1 High Conc. Mask; 2 Nasal Prongs; 3 28% Ventimask; 1 14L (2) BLS; 1 Demand Valve

HAEMORRHAGE CONTROL: ☐ Dressing ☐ Elevation ☐ Pressure

IV CANNULATION: 1 Type Insta Pressure; 2 Unsuccessful; 3 In Situ; Local given ☑ No ☐ Yes

INTUBATION: 1 Type .....; 2 Unsuccessful; 3 In Situ

ANALGESIA: E Entonox N Narcotic; 1 Not Effective 2 Part Effective 3 Effective

SPLINTS: 1 Hare 2 Thomas 3 Air 4 Cervical Collar 5 KED 6 Other 7 Donway

MAST Suit - Inflated: 1 Under 500ml 2 Over 500ml (External blood loss)

TOURNIQUETS: 1 Rotating 2 Arterial 3 Lymphatic Time On .....

STRETCHER: 1 Carry Chair 2 Back Board 3 Jordon Frame 4 Carry Sheet 5 Other

KITS: 1 Poison 2 Burns 3 Maternity (4) Other IV

BLOOD SAMPLE TAKEN: Glucose Est. ..... Blood Type & Cross Match .....

TYPENEX No.

RHYTHMS MONITORED (Abbreviations in heavy print)

1 Sinus Rhythm; 2 Sinus Bradycardia; 3 Sinus Tachycardia; 4 Prem. Atrial Cont.; 5 Supra Vent. Tachy; 6 Atrial Flb / Flut.; 7 Idiovent. Rhythm; (8) Asystole; 9 Pacemaker Rhythm; 10 Prem. Vent. Cont. Frequency ..... Runs ☐ R on T ☐ Multifocal ☐; 11 Vent. Tachycardia; 12 Vent. Fibrillation; 13 1st Heart Block; 14 2nd Heart Block; 15 3rd Heart Block; 16 Junctional; 17 Multiple Arrhythmia; 18 Elect. Mech. Disen.

TIME	ECG	TREATMENT	DOSE	ROUTE	SHOCK	RESULT	AUTH
0135	8	ADRENALINE 1:1000	200mg	IOT		NIL	P/C
0140	8	ADRENALINE 1:10000	200mg	IOT		NIL	P/C
0145	8	ADRENALINE 1:10000	200mg	IOT		NIL	P/C
0150	8	ADRENALINE 1:10000	200mg	IOT		NIL	P/C
0135		HARTMANNS	c.100mls	IOT		FLUSH	P/C

CHECKED
CASE SHEET
DETAIL CHECKED & FOUND CORRECT

Mother states child weighs 9kg

O.I.C. — STATION OFFICER

COMMENTS: ASSISTED O/S BY 133 Report # 001

Kelly Craig 66489

CONDITION AT DESTINATION ☐ IMPROVED ☐ UNCHANGED ☐ DETERIORATED

OFFICER 1 DRIVING: D. MARTIN — SIGNATURE — EMP. No. 11015

OFFICER 2 — SIGNATURE — EMP. No.

CLINICIAN UNDER 15 YRS

	WT. SCORE
AIRWAY	
BP	
CNS	
FRACTURES	
WOUNDS	
TOTAL	

BYPASS SYSTEM — CATEGORY

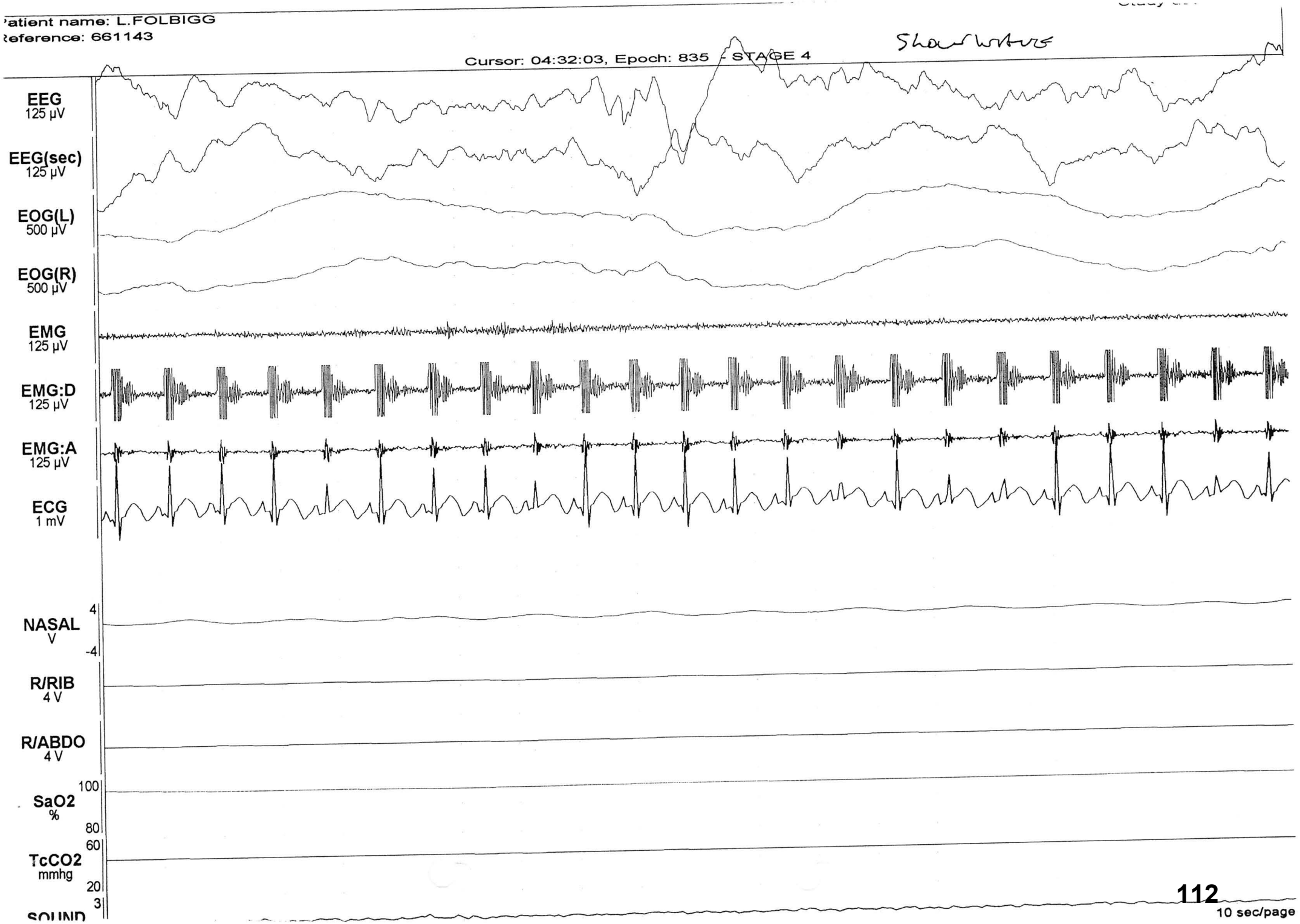
TRAUMA CHECKLIST — DECISIONS FROM TRAUMA CHECKLIST >

SUBTRACT 6 POINTS

ATTACH ECG STRIPS HERE

TRANSIT

	BYSTANDER
	Effect
	ROAD USER
	Severity
0	TREATMENT before arrival
B	Conscious/Unconscious
	SUCTION
1	AIRWAY
	BREATHING
1	IPPV
	CIRCULATION
2	POSTURE
2	O_2 THERAPY
	Libs
	DEMAND VALVE
1	CANNULATION
	INTUBATION
	ANALGESIA
	Ellems
	SPLINTS
	SPLINTS
	MAST SUIT
	STUDY 1
	STUDY 2
	STUDY 3
8	ECG
	ECG
	ECG
	D.C. SHOCK
11	FLUID / DRUG
4	Units
	FLUID / DRUG
	Units
	FLUID / DRUG
	Units
	FLUID / DRUG
	Units
	FLUID / DRUG
	Units
02	PROTOCOL
16	PROTOCOL
	PROTOCOL
	HAZARDS
	TRIAGE TOOL
	TRANSPORT DECISION
	MAIN COMPLAINT
	OTHER COMPLAINT
	OTHER COMPLAINT
	P.T.S. < 8 IS SERIOUS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EEG(sec)
125 µV
EOG(L)
500 µV
EOG(R)
500 µV
EMG
125 µV
EMG:D
125 µV
EMG:A
125 µV
ECG
1 mV
NASAL
V
4
-4
R/RIB
4 V
R/ABDO
4 V
SaO2
%
100
80
TcCO2
mmhg
60
20
3
SOUND
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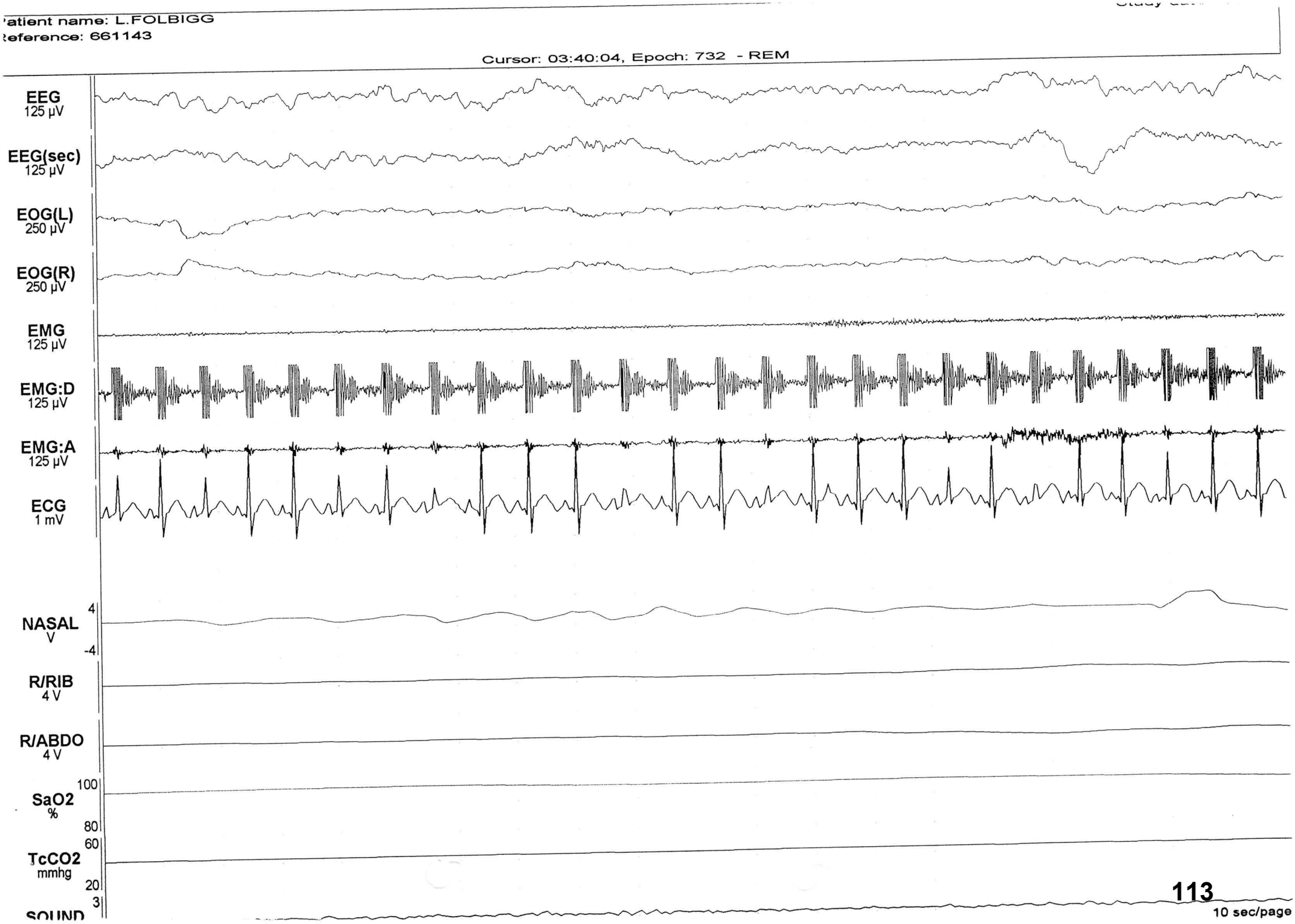
10 sec/page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EOG(L)
250 µV
EOG(R)
250 µV
EMG
125 µV
EMG:D
125 µV
EMG:A
125 µV
ECG
1 mV
NASAL
V
4
-4
R/RIB
4 V
R/ABDO
4 V
SaO2
%
100
80
TcCO2
mmhg
60
20
3
SOUND
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10 sec/page

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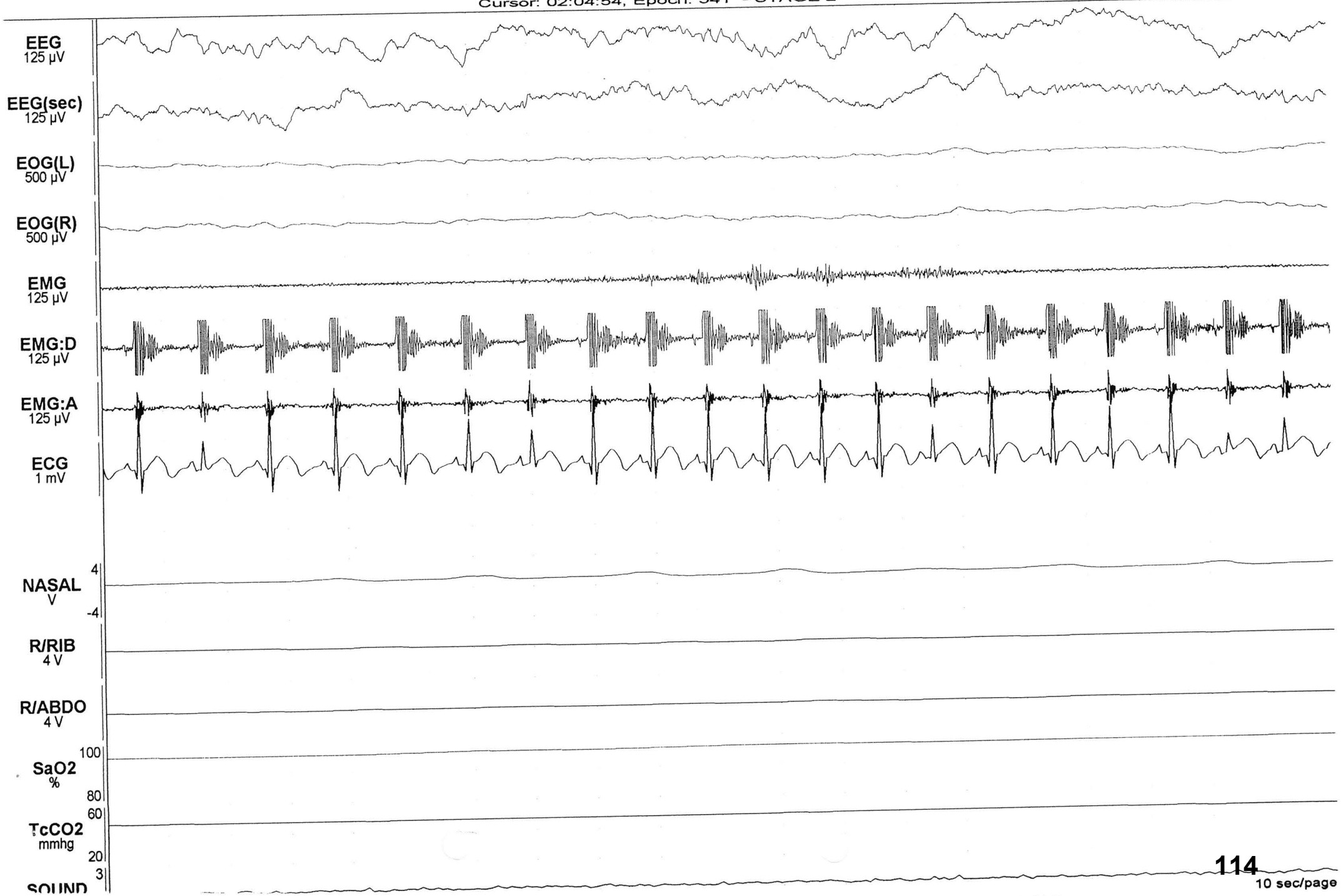

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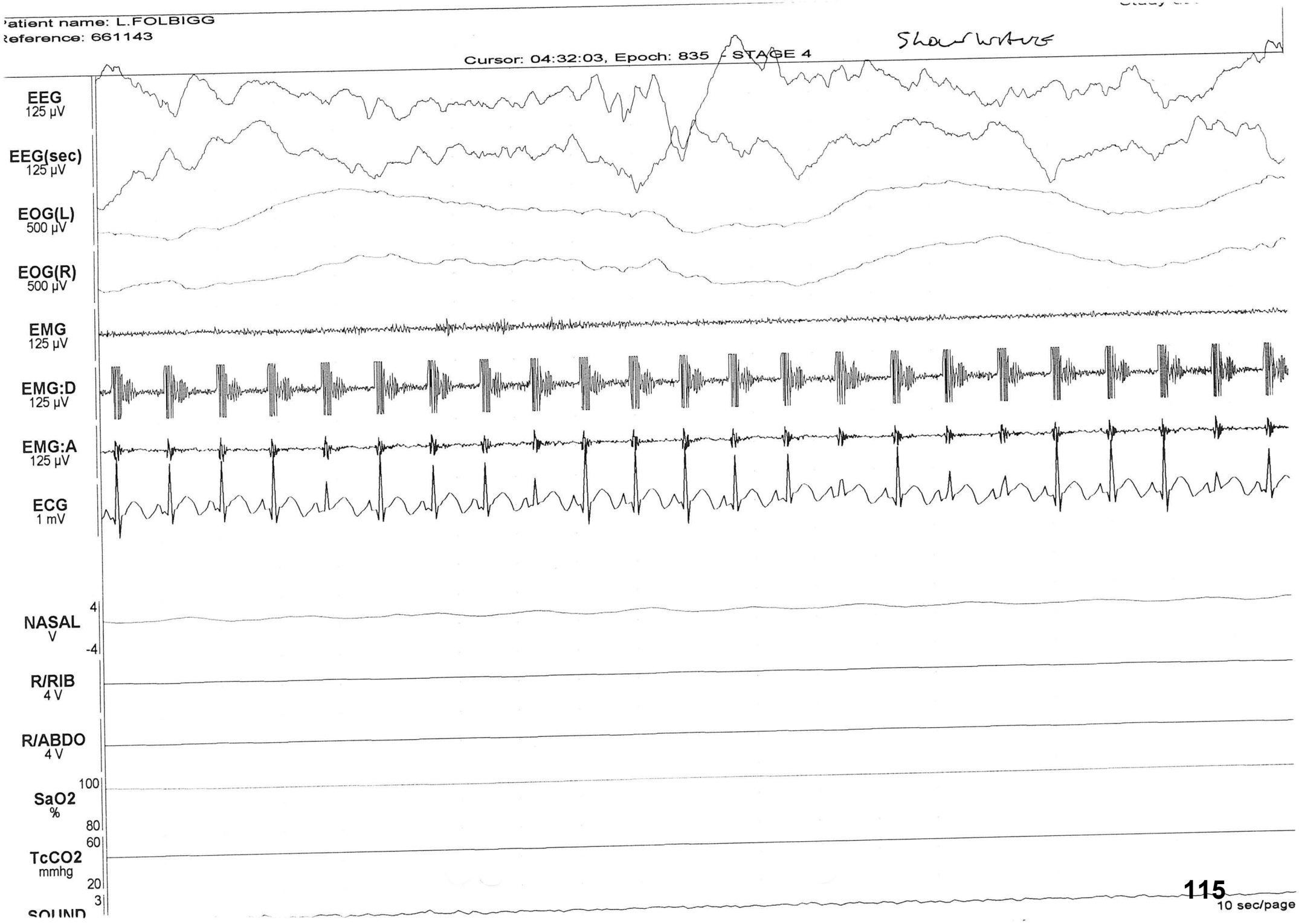
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EEG(sec)
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EOG(L)
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EOG(R)
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EMG
125 µV
EMG:D
125 µV
EMG:A
125 µV
ECG
1 mV
NASAL
V
4
-4
R/RIB
4 V
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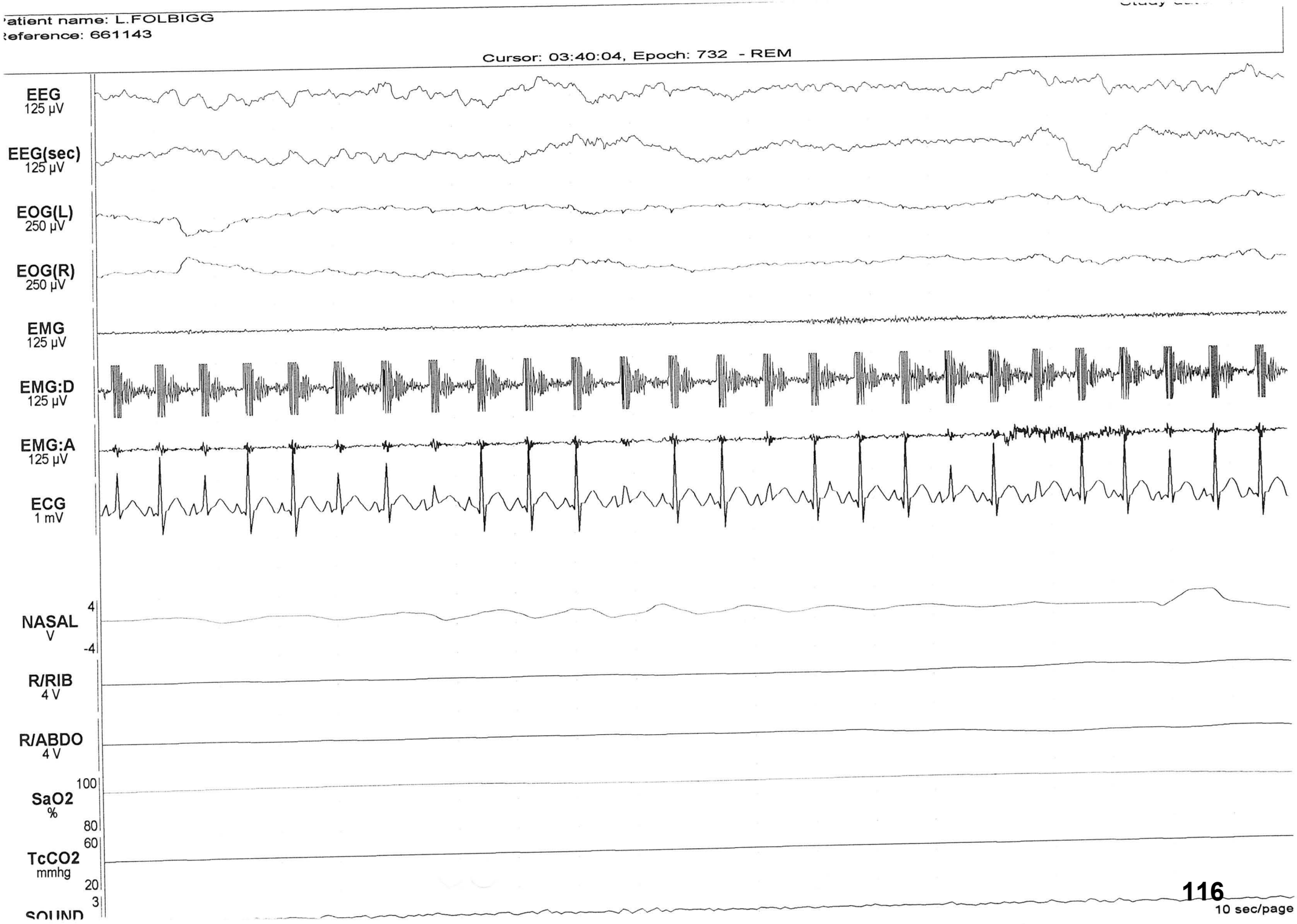
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EOG(R)
250 µV
EMG
125 µV
EMG:D
125 µV
EMG:A
125 µV
ECG
1 mV
NASAL
V
4
-4
R/RIB
4 V
R/ABDO
4 V
SaO2
%
100
80
TcCO2
mmhg
60
20
3
SOUND
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10 sec/page

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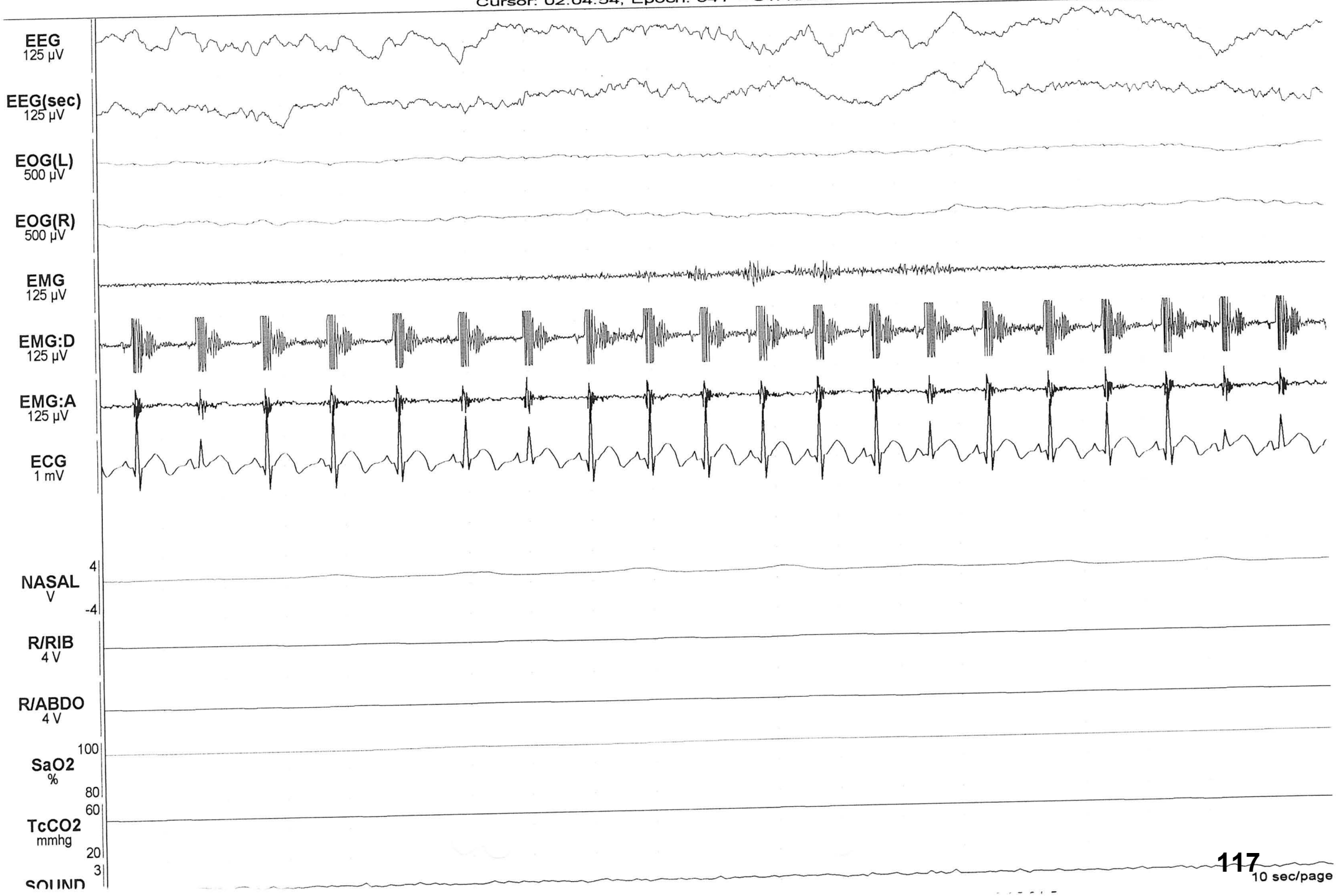

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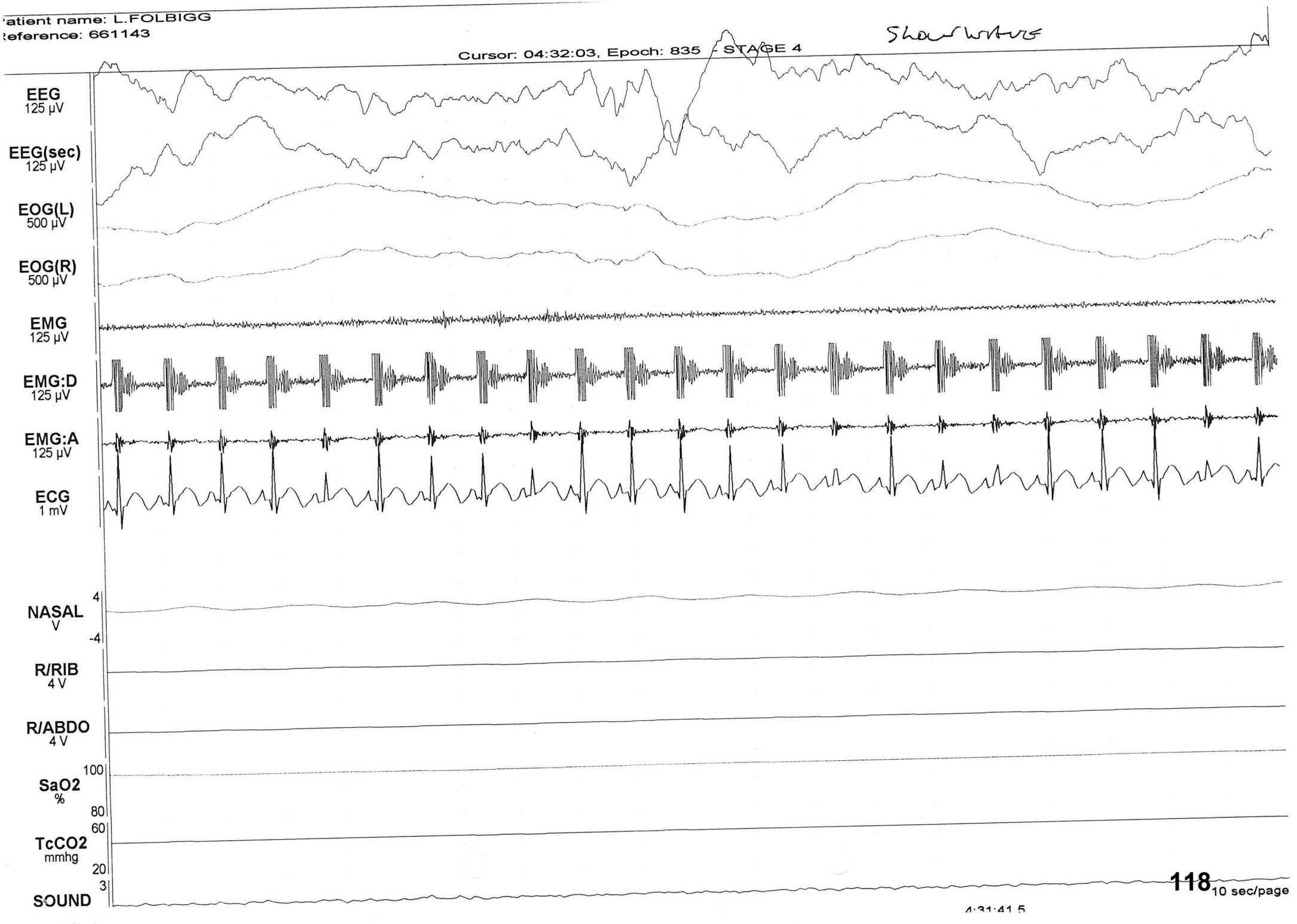
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125 µV
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EOG(L)
500 µV
EOG(R)
500 µV
EMG
125 µV
EMG:D
125 µV
EMG:A
125 µV
ECG
1 mV
NASAL
V
4
-4
R/RIB
4 V
R/ABDO
4 V
SaO2
%
100
80
60
TcCO2
mmhg
20
3
SOUND
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118
10 sec/page

Patient name: L.FOLBIGG
Reference: 661143

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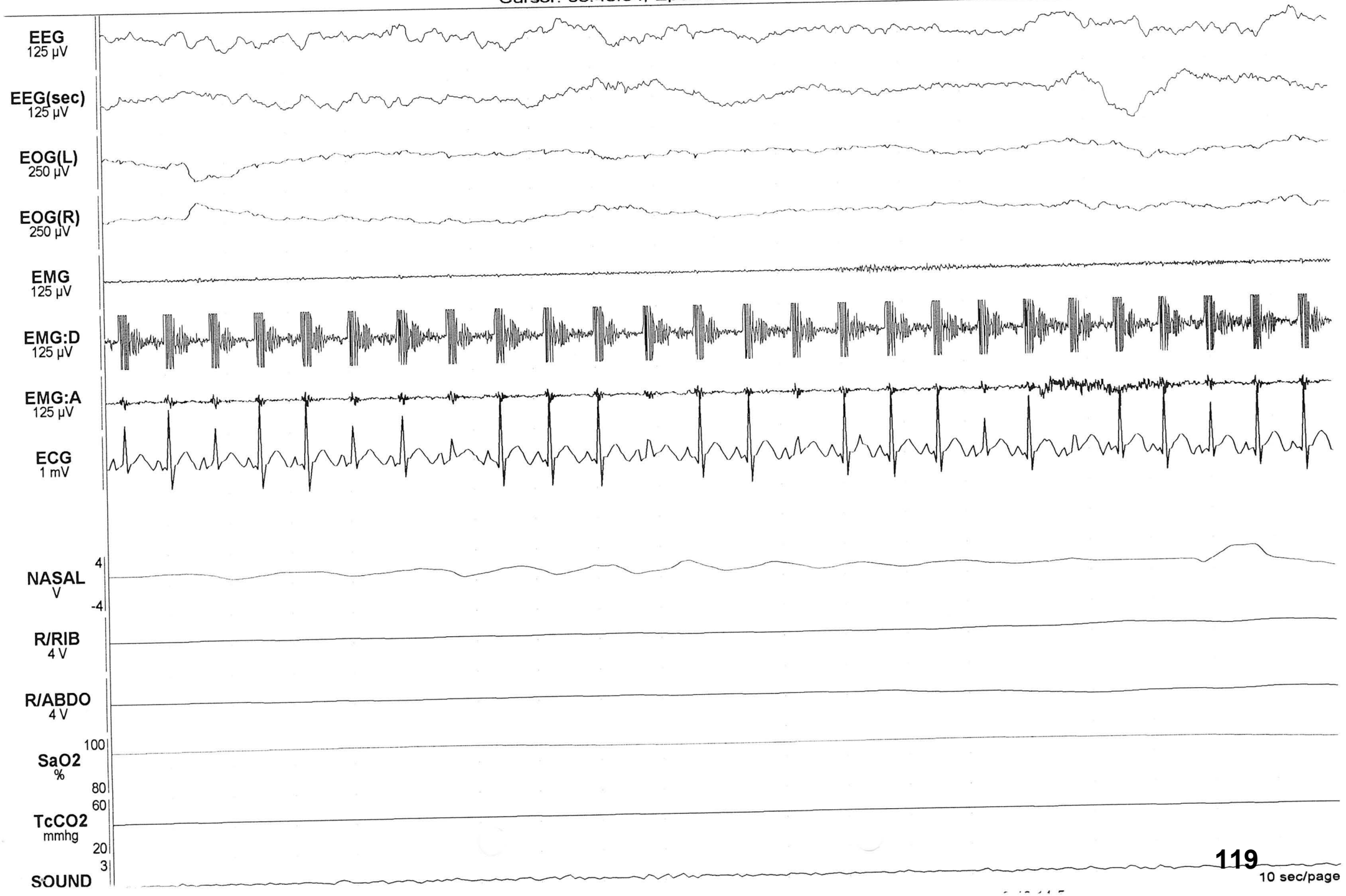

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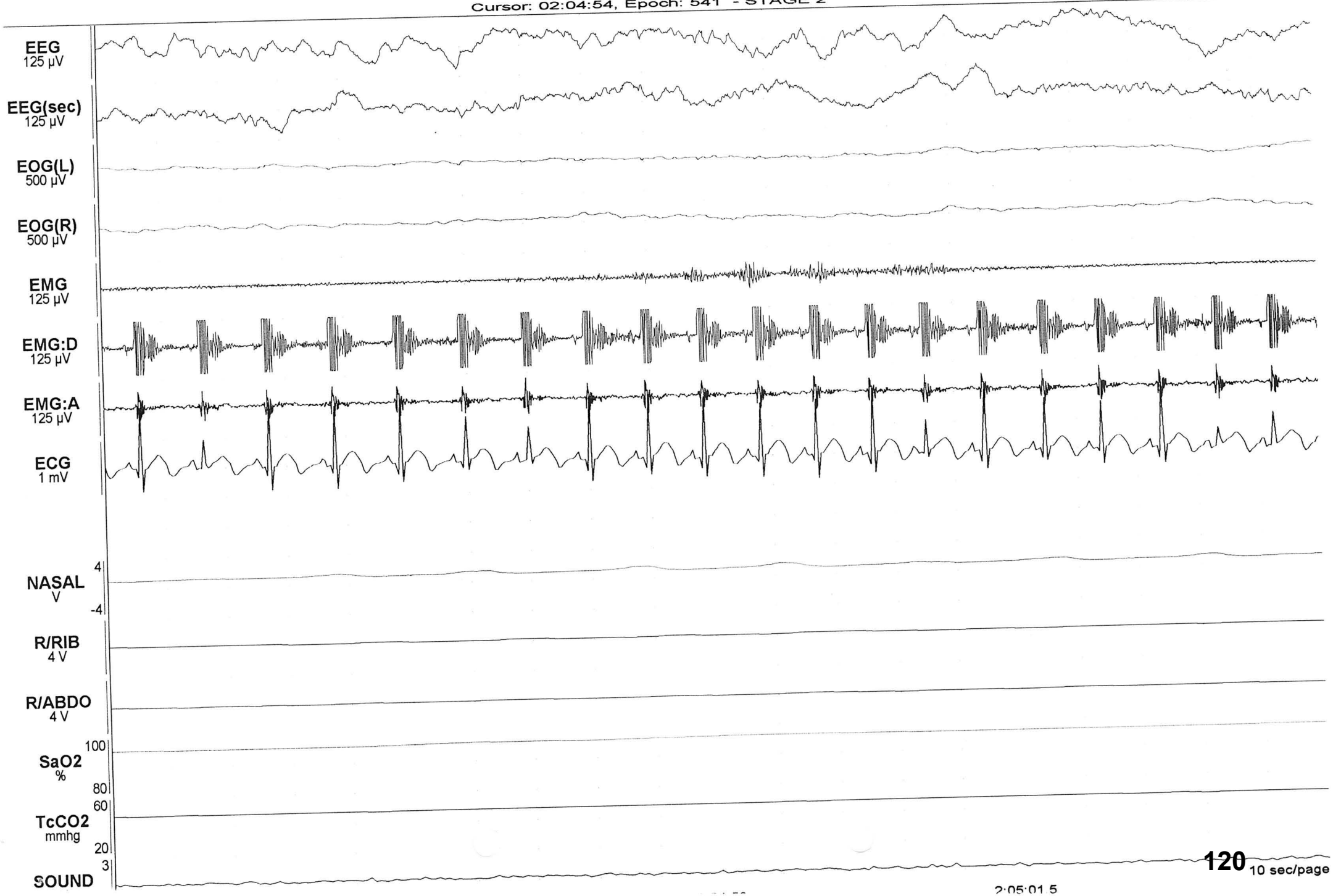

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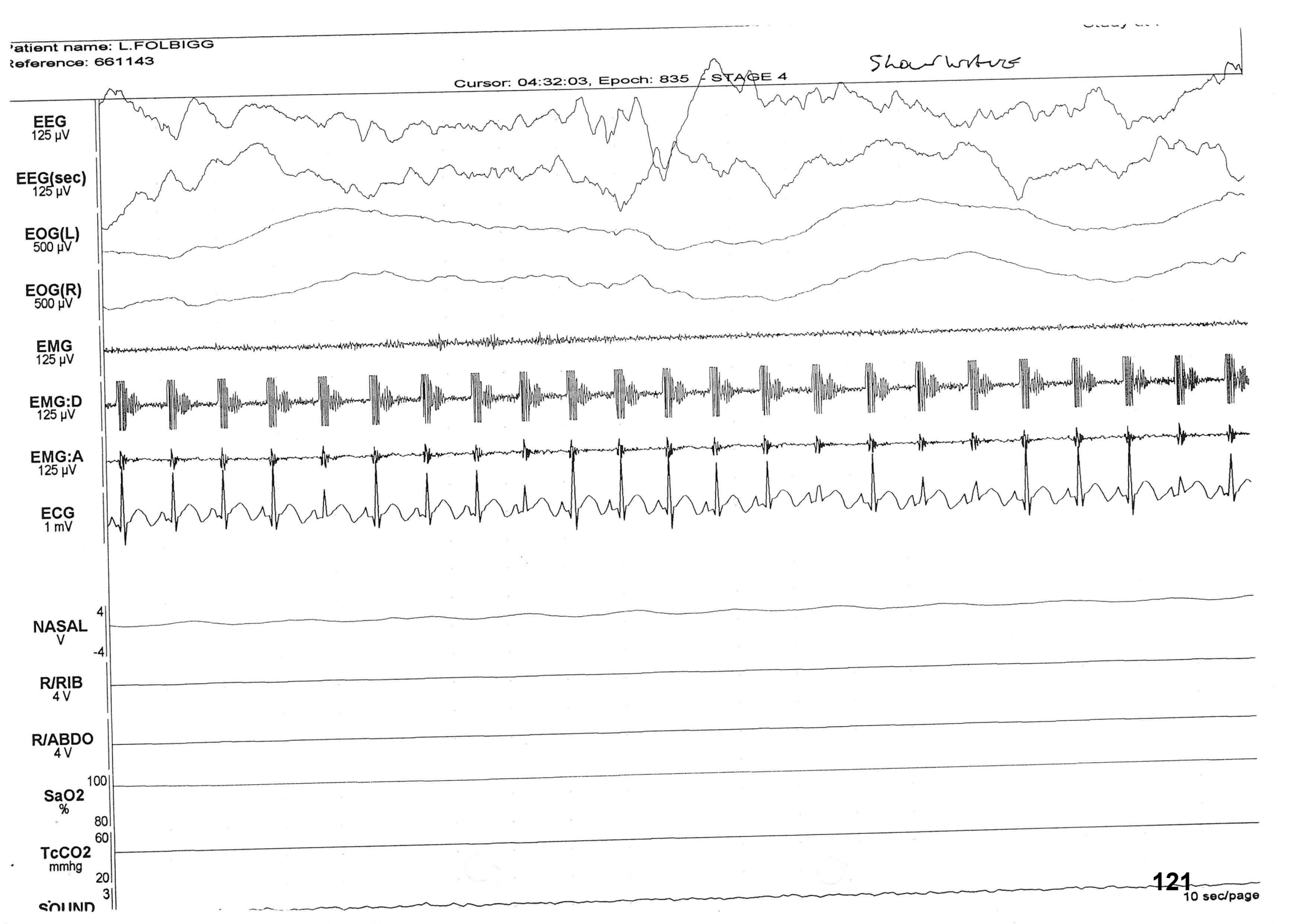
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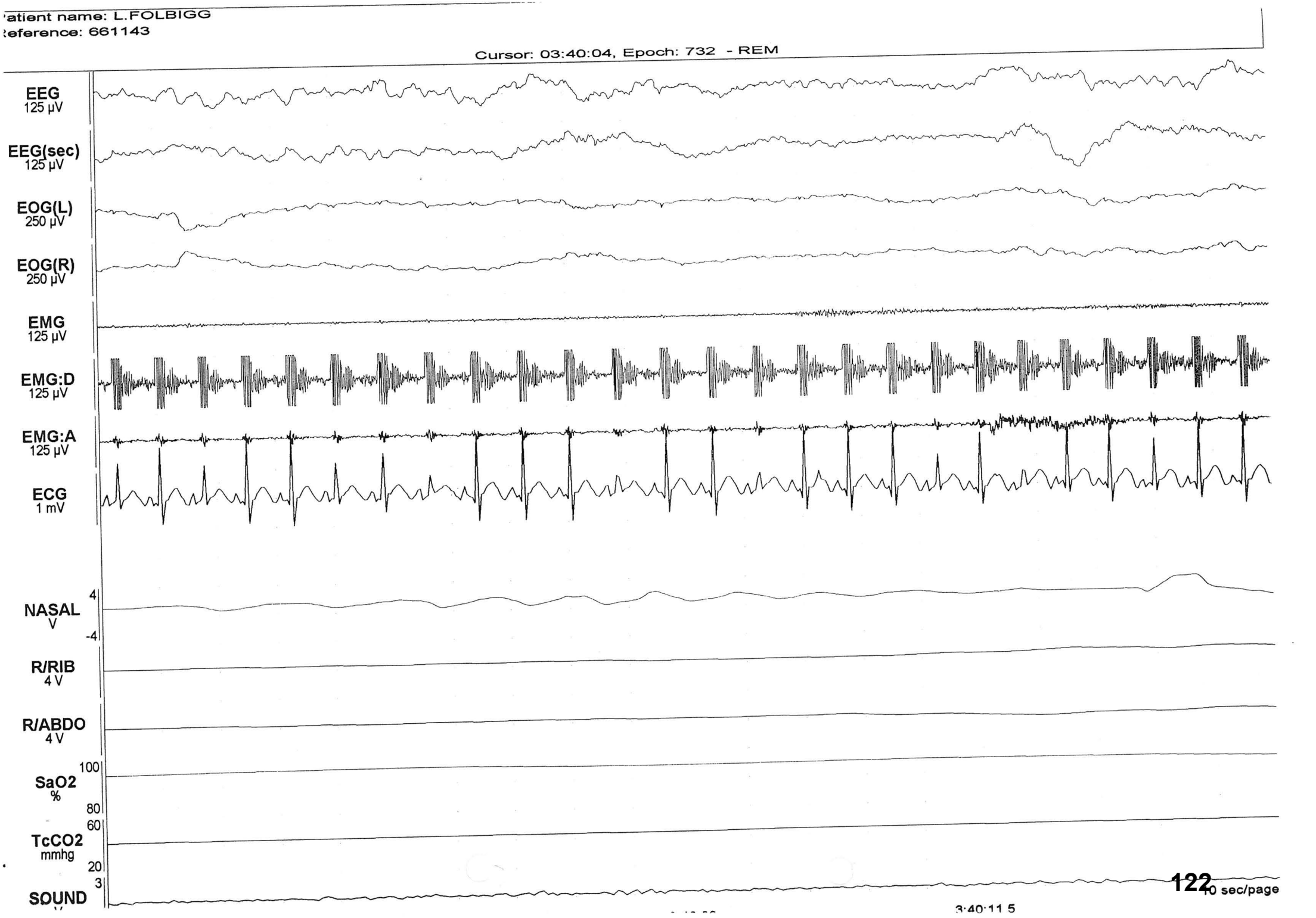
125 µV
EEG(sec)
125 µV
EOG(L)
500 µV
EOG(R)
500 µV
EMG
125 µV
EMG:D
125 µV
EMG:A
125 µV
ECG
1 mV
NASAL
V
4
-4
R/RIB
4 V
R/ABDO
4 V
SaO2
%
100
80
TcCO2
mmhg
60
20
3
SOUND
121
10 sec/page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eference: 661143
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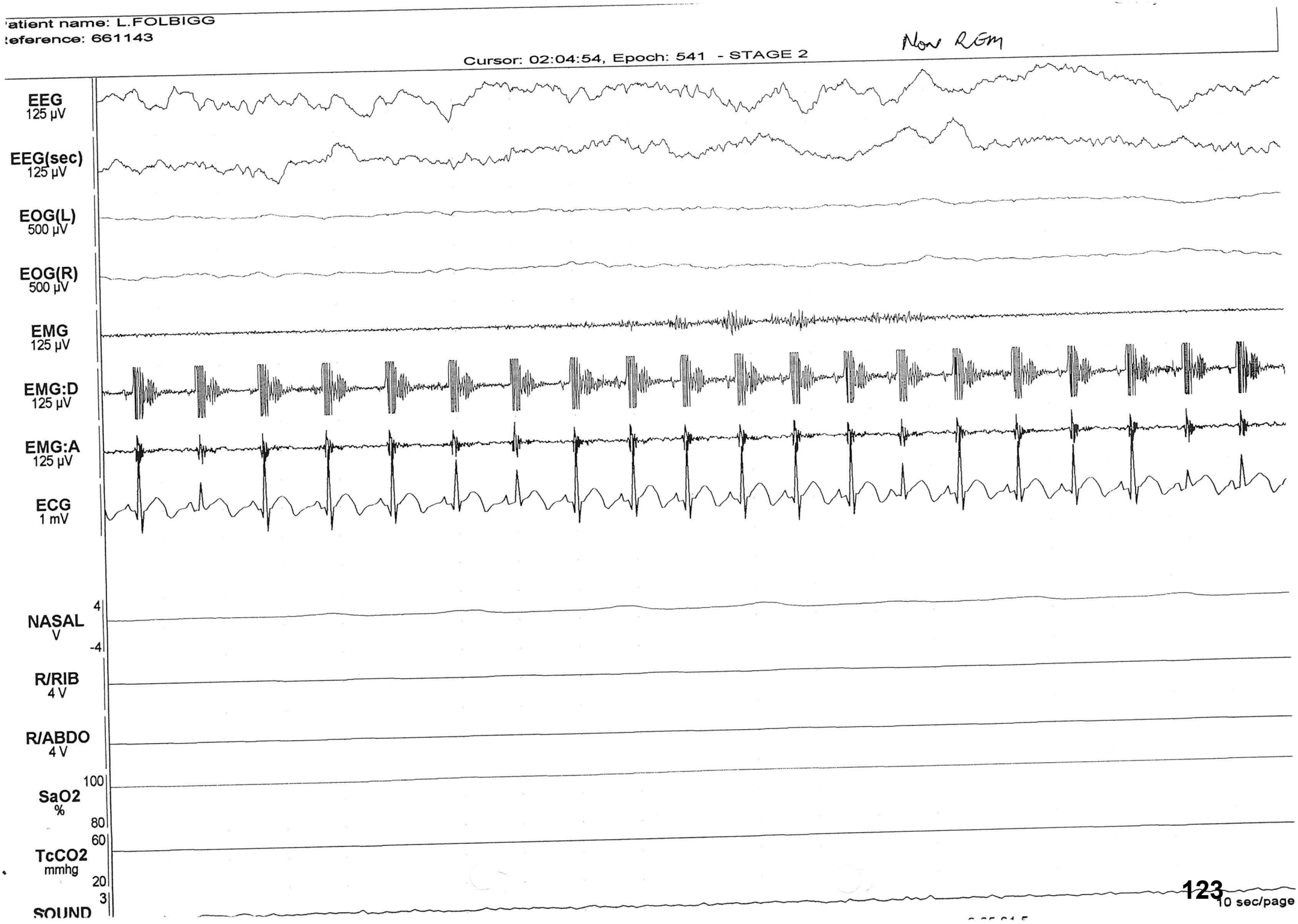
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eference: 661143
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EOG(L)
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EMG
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125 µV
EMG:A
125 µV
ECG
1 mV
NASAL
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-4
R/RIB
4 V
R/ABDO
4 V
SaO2
%
100
80
60
TcCO2
mmhg
20
3
SOUND
10 sec/page

NEW SOUTH WALES

BIRTHS, DEATHS AND MARRIAGES REGISTRATION ACT 1995

BIRTH CERTIFICATE

REGISTRATION NUMBER
56425/1997

58

1 CHILD Family Name Christian or Given Name(s) Sex Date of Birth Place of Birth	FOLBIGG Laura Elizabeth Female 07 August 1997 Singleton Hospital, Singleton
2 MOTHER Family Name Maiden Family Name Christian or Given Name(s) Occupation Age Place of Birth	FOLBIGG MARLBOROUGH Kathleen Megan Sales Assistant 30 years Balmain, NSW
3 FATHER Family Name Christian or Given Name(s) Occupation Age Place of Birth	FOLBIGG Craig Gibson Sales Manager 35 years Newcastle, NSW
4 MARRIAGE OF PARENTS Date of Marriage Place of Marriage	05 September 1987 Kotara, NSW
5 PREVIOUS CHILDREN OF RELATIONSHIP	Calab G., deceased Patrick A. deceased Sarah K. deceased
6 INFORMANT(S) Name Address	K. FOLBIGG 8 Millard Cl Singleton 2330 Mother C. FOLBIGG 8 Millard Cl Singleton 2330 Father
7 REGISTERING AUTHORITY Name Date	B. A. Flett, Registrar 01 September 1997
8 ENDORSEMENT(S) Not any	

Before accepting copies, sight unaltered original. The original has a coloured background.

REGISTRY OF BIRTHS
DEATHS AND MARRIAGES

SYDNEY 04 Apr 2000

I hereby certify that this is a true copy of particulars recorded in a Register in the State of New South Wales, in the Commonwealth of Australia

Registrar

N.S.W. MIDWIVES DATA COLLECTION

Unit Record Number 4 7 2 4 8 7

HOSPITAL SINGLETON HOSPITAL CODE Q 2 1 7

(1st Name) KATHLEEN

Family Name FOLBIGG

Address 8 Millard Close Singleton Postcode 2330

D.O.B. Mother 14 06 67 (day month year)

Country of Birth (Mother)

Australia ☑ England ☐ Lebanon ☐ New Zealand ☐ Philippines ☐ Vietnam ☐ Other ☐

Ethnic Origin (Mother)

Caucasian ☑ (Aust) Aboriginal ☐ Asian ☐ Other ☐

Patient Classification

Hospital ☑ Private ☐

Marital Status

Married/Defacto ☑ Never married ☐ Div./Sep./Wid. ☐

PREGNANCY

Has mother had a previous pregnancy greater than 20 weeks?

Yes ☑ No ☐

If yes, specify number of previous pregnancies > 20 weeks 03

THIS PREGNANCY

Date of LMP 28 10 96 (day month year)

Maternal medical conditions

Diabetes mellitus ☐
Essential hypertension ☐
Hepatitis B+ ☐

Obstetric complications

APH — Placenta praevia ☐ — Placenta abruptio ☐ — Cause unknown ☐
Pregnancy induced hypertension ☐
Gestational diabetes ☐
Prelabour rupture of membranes (> 24 hours) ☐
Threatened premature labour ☐
Blood group isoimmunisation ☐

Procedures and operations

Cervical suture ☐
Amniocentesis (< 20 weeks) ☐
C.V.S. (< 20 weeks) ☐

Antenatal care

Duration of pregnancy at first visit (weeks) 06

Not booked (tick if applicable) ☐

Research question

Did the mother smoke at all during pregnancy?

Yes ☐ No ☑

If yes, approximately how many cigarettes did she smoke each day on average in the second half of pregnancy?

none ☐ > 10 day ☐ ≤ 10 day ☐ unknown ☐

LABOUR AND DELIVERY

Labour onset

Spontaneous ☑ Induced ☐ No labour ☐

If labour onset spontaneous:

Augmented with A.R.M. ☐
Augmented with oxytocins/prostaglandins ☐

If labour induced:

Oxytocins/prostaglandins ☐
A.R.M. only ☐
A.R.M. with oxytocins/prostaglandins ☐
Other ☐

Presentation

Vertex ☑ Breech ☐ Other ☐ Unknown ☐

Other procedures

Epidural block ☐ Episiotomy ☐

Type of delivery

Normal vaginal ☑ Vacuum extr. ☐ Forceps ☐ Vaginal breech ☐ Forceps rotation ☐

Caesarean section:

Before labour onset (elective) ☐ In labour (emergency) ☐

Complications

P.P.H. (600 mls +) ☑
Retained placenta ☐
Third degree tear ☐
Major puerperal infection ☐
Shoulder dystocia ☐

BABY

Place of birth (baby)

Hospital theatre/labour ward ☑
Birthcentre ☐
Planned birthcentre/Labour ward delivery ☐
Planned homebirth ☐
Planned homebirth/Hospital admission ☐
Born before arrival ☐

Record No. 7 5 3 7 0 3

Sex: M ☐ F ☑ Indet. ☐

Birthdate: 07 08 97 (day month year)

Plurality: Single ☑ Multiple ☐

If multiple birth, total number

If multiple birth, specify baby number

Birthweight (grams): 3260

Estimated gestation age (weeks): 41

Apgar (1 minute): 09

Apgar (5 minutes): 10

Neonatal morbidity:

Did the baby require admission to a separate facility/nursery for special care or observation?

Yes ☐ No ☑

BIRTH DEFECTS

Does this baby have a birth defect?

Yes ☐ No ☑

If Yes, specify

Office use only: minor ☐ major ☐

DISCHARGE STATUS — MOTHER AND BABY

MOTHER

Discharged ☑ Transferred ☐ Died ☐

BABY

Discharged ☑ Transferred ☐ Stillbirth ☐ Neonatal death ☐ Transferred and died ☐

Baby's date of discharge or transfer 11 08 97 (day month year)

Baby transferred to:

If baby died, date of death (day month year)

Signature of Midwife at discharge

Public Health Act 1991

606201

9/96

Hospital Copy

125

Form No. MR44/PR16

SUDDEN INFANT DEATH P534

DEATH SCENE INVESTIGATION CHECKLIST

(Sudden deaths of children up to two years of age are notifiable)

1. When inquiring into the circumstances of the death of a child two years and younger this form is to be completed and forwarded to the Coroner with completed form P79A
2. Contact the Duty Forensic Pathologist immediately on Glebe 9660 5977 or

Carefully explain to the parents, family or carer the need to fully explore the circumstances in an attempt to establish the cause of death. Do not hurry the interview.

TELEPHONE INTERPRETER SERVICE
131 450 (24 HRS)

INFANT'S DETAILS

Surname: **FOLBIGG** Place of Birth: **Singleton Hospital**
First Name/s: **Laura Elizabeth** Date of Birth: **07/08/97** D/M/Y
Female ☑ Home address: **8 Millard Close,**
Suburb: **Singleton** State: **NSW** Postcode: **2330**
Place of death (Street): **Singleton Hospital**
Suburb: **Singleton** State: **NSW** Postcode: **2330**

MOTHER'S DETAILS

Surname: **FOLBIGG** Date of Birth: **14/06/67** D/M/Y
Address at birth of infant: **8 Millard Close,** First Name/s: **Kathleen Megan**
Suburb: **Singleton** State: **NSW** Postcode: **2330**

MEDICAL INFORMATION

Weight at birth: **7lbs**
Premature birth? **No** ☑
Does infant have siblings: **No** ☑
Infant been immunised? **Yes** ☑
Normal Delivery? **Yes**
eg. Triple Antigen (or DPT), Polio (Sabin)
Comments:
Comments:

Is there a past history of unexpected Infant Death in the family? **Yes**
If yes, names: **Three previous siblings have died and these deaths were attributed to SIDS.**

Was infant previously healthy? **Yes** ☑ Comments:
Did infant have any illnesses or changes in behaviour in the past two weeks especially the last 24 hours? **Cold** ☑
Had the infant received any prescription or over-the-counter medication? **Yes** ☑
If yes, type: **Demozin** Amount: **3.5ml** Time of last dose: **27.2.99**
Did the infant have any falls or sustain any injury recently? **No**
Comments:
How was the infant being fed? **Solids** Brand of formula:
Other: **Milk and solid food**
Any feeding problems? **No** When infant last fed? **7am 1.3.99**
Describe: Breakfast
Name & address local doctor: **Doctor INNIS of Singleton Heights Medical Centre**
Location of Early Childhood Centre attended: **of**

Request the "Personal Health Record" (blue book) of the infant. Explain to the parents that it is required by the medical officer to assist with regard to the cause of death and will be returned

Any illnesses in the family? **No** ☑ Describe:

Any family member on medication? **No** ☑ Describe:

Anyone in household smoke? **Yes** ☑ How many live with infant? **Two**

Who found the infant? **Natural mother** Time: **12.05pm**

How did the infant come to be found? **Noise** ☑
Specify: **Started coughing and checked 5 minutes later.**

Was any resuscitation attempted? **Parent** ☑
Describe: **CPR by parent and then ambulance**

Where was the infant when found? **Other** ☑
Describe: **Single bed**
Type of mattress: **Innerspring**
Describe:

Were there items covering the infant's head? **No** ☑
List the items:

Was the infant sleeping alone? **Yes** ☑ With whom:

Position of infant when put down: **Side** ☑

Position of infant when found: **On Back** ☑

Were there any recent changes in sleeping pattern? **No** ☑
Describe:

Was the infant found in an unusual sleeping position? **No** ☑
Describe: **Laying on back**
What clothing was the infant wearing at the time? **disposable nappy, tights and shirt**

SUPPORT ORGANISATIONS
The Grief Counselling Service available through the NSW Institute of Forensic Medicine Glebe (02) 9660 5977 (24 hours) including the Counselling Service Westmead Coroners Office and the Sudden Infant Death Association SIDA 1 800 651 186 (24 hours) are available to assist parents and families in the event of sudden infant death. Refer next of kin to these organisations or contact them on the family behalf.

Name of Officer: [redacted] Rank: [redacted]
Location: [redacted] Phone: [redacted]

Investigating Officer to complete this section with own observations:

Evidence of drug/alcohol abuse? **No** ☑ Describe:
Describe general condition of premises? **Clean and well maintained**
Specify which room the infant was found: **infant's own bedroom**

Room temp: **Comfortable** ☑ Ventilation: **Comfortable** ☑
General Comments: House appeared a safe environment for children.

Royal Alexandra Hospital for Children

AMO .....

WARD .....

Date of Admission 3/2/98

066-11-43

15

FOLBIGG Laura
8 MILLARD CLOSE SINGLETON 2330
DOB 07-Aug-1997 SEX F CLASS 00
PH.: 02 6572162 AMO SETON

INFANT HISTORY: Born at Singleton Hospital Family Rank: 4/4

Prenatal Influences:

Delivery: Full term, vaginal delivery

Gestation wks Birth Weight g 7lbs4 Apgar Scores: 8, 10

Perinatal: O_2 requirements: nil Jaundice: yes, no Rx Tube feeding: nil

Discharged Day day 5

FEEDING HISTORY: Breast feeding 2/52 then bottle

PREVIOUS HEALTH: Infectious diseases: Measles Mumps

Chickenpox Rubella Other (stage age of onset)

Other illnesses or accidents:

Operations: Nil

Blood Transfusions: Previous Corticosteriod Therapy:

ROUTINE IMMUNISATION (TICK OR WRITE APPROXIMATE DATE GIVEN IN BOX)	1st dose 2 months	2nd dose 4 months	3rd dose 6 months	12 months	18 months	5 years or pre-school	10-16 years
POLIO (SABIN)	✓	✓					
DTP (Triple antigen) or CDT	✓	✓					
MEASLES/MUMPS/RUBELLA							
H. INFLUENZAE type b (Hib)•	✓	✓					
HEPATITIS B••: Three doses at 0, 1-2 and 6 months if indicated			1st dose		2nd dose		3rd dose
CATCH UP IMMUNISATION REQUIRED?					YES		NO

- Hib TITER indicated for all infants except Aboriginal infants who are given PedvaxHIB instead. If commencing Hib TITER aged between 6-12 months give 2 doses 2 months apart and a booster at 18 months. If aged 12-14 months give one dose then booster at 18 months. If aged 15 months or older only one dose required.
- •• Indicated for Aboriginal, Asian, SE Asian, Indian, Pacific Island, Central European, Mediterranean, Middle Eastern, African and South American children.

FAMILY HISTORY: Consanguinity

Father: Age: Ethnic group: Occupation:

Mother: Age: Ethnic group: Occupation:

Siblings: 2 brothers 1 deceased
1 sister

Miscarriages:

Stillbirths:

Deaths: SIDS x 3

Chronic or heritable illness in parents or siblings:

SOCIAL HISTORY:

ALLERGY / ADVERSE DRUG REACTION: NKA

CURRENT MEDICATION: Nil

MILESTONES: Present Age: years 6 months

Smiled 4 weeks. Sat unaided 5 months. Crawled months

Walked unaided months. Speech

ROYAL ALEXANDRA HOSPITAL FOR CHILDREN (NEW CHILDREN'S HOSPITAL)

MP 3/94

Royal Alexandra Hospital for Children

AMO .....

WARD .....

Date of Admission .....

066-11-43

FOLBIGG Laura
8 MILLARD CLOSE SINGLETON 2330
DOB **07-Aug-1997** SEX **F** CLASS **00**
PH. : **02 6572162** AMO **SETON**

HISTORY OF PRESENT ILLNESS Date: 3/2/98

Obtained from: –

By (PLEASE PRINT) K. THIRU

LMO

Address:

PC 6/12 ♀ for sleep study

HPI
- Apnoea once / night
- lasts 10 sec
- resolves spontaneously
- on commencing SIDS monitor
- not aware of apnoeas when awake
- sleep study at 2/12 and 5/7 age → mild mixed apnoeas
- NIL snoring at night
- sleeps 20:30 → 06:30, 2 to 3 daytime sleeps ~1 hour
- nose breathing

FHx
3 x SIDS - 20/7
- 9/12
- 10/12

Meds
Nil regular

Allergies
N/K.

O/E : Well
Distressed from being
Probes stuck on feet

Chest AE good R=L
BS vesicular
? added sounds

HS dual ↑ rate
? murmur

Throat - tonsils not enlarged / inflam
Ears - T.M. poorly visualised L > R

Plan
Admit under Dr Seth
Sleep study tonight
Consent ✓

D/C in AM

[signature]

ROYAL ALEXANDRA HOSPITAL FOR CHILDREN (NEW CHILDREN'S HOSPITAL)

MP 3/94

Trolley: 49	Pathologist: Cala, Allan	For Asst: O'Neill, Darren
	Supervisor:	Protocol: Homicide - Female

Surname: **Folbigg**
Given Name(s): **Laura Elizabeth**
Aliases:
Case Number: **99-9322**

Weight: 12.
Height: 80.5cm
Age: 1 years 8 months
Sex: Female

Identity Known **Y**
Identified By: Mr. De Wit
Police Station: UNDERTAKER
Identified To: Carruthers, Darren
Date/Time: 01/03/99 07:32 PM

Brain	1154
Heart	62
Left Lung	122
Right Lung	114
Liver	930
Left Kidney	36
Right Kidney	36
Spleen	46
Pancreas	/
Prostate	/
Thyroid	/
Other	

Specimens for Analysis

Lung/Spl.
CSF/O/R/N → Bacto
CSF → Biochem.
B/L/S/U/B → DAL

Distinguishing Features

AUTOPSY

Date: 1/3/99
Time: 9 am / pm

Cause of Death

1. Undetermined.
2.

Chris Nicoll
Prof. Hilton
HC 47cm
CC 49 cm
AC 44.5cm
FL 12.0cm
TC 2.7cm

Notes and Comments

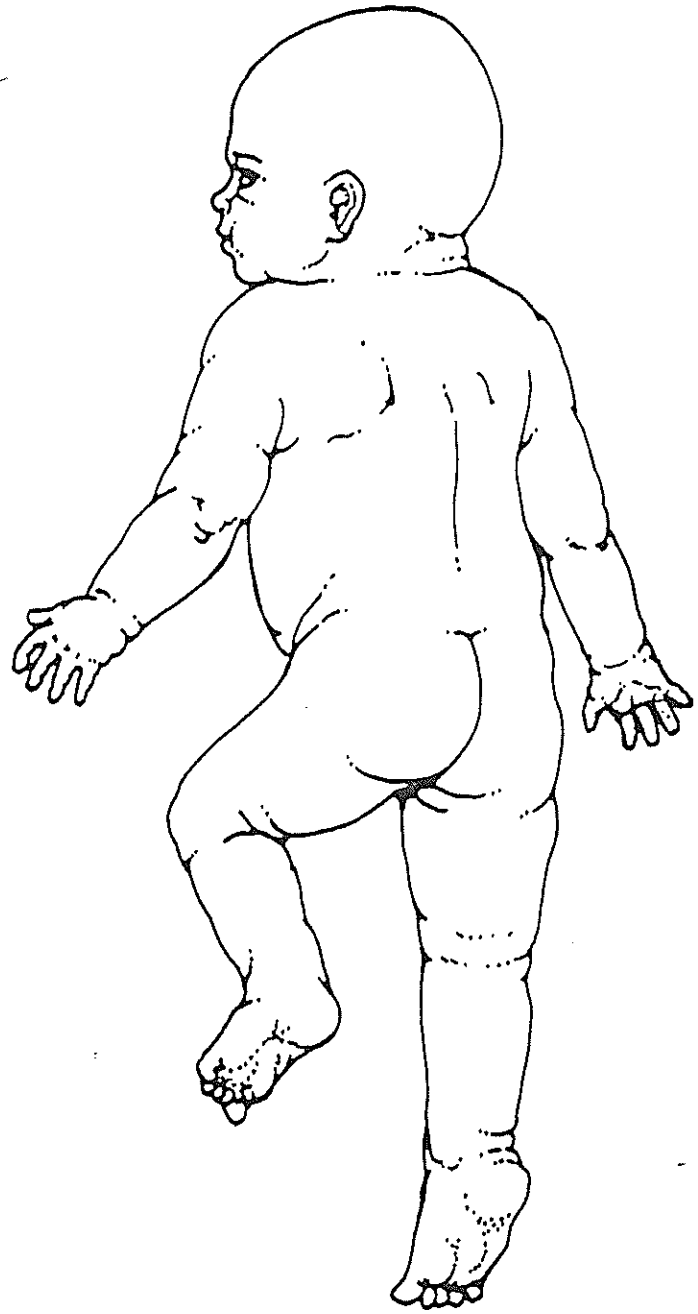
* No viral culture media in IOFM at time of PM.
→ No viral cultures able to be done.

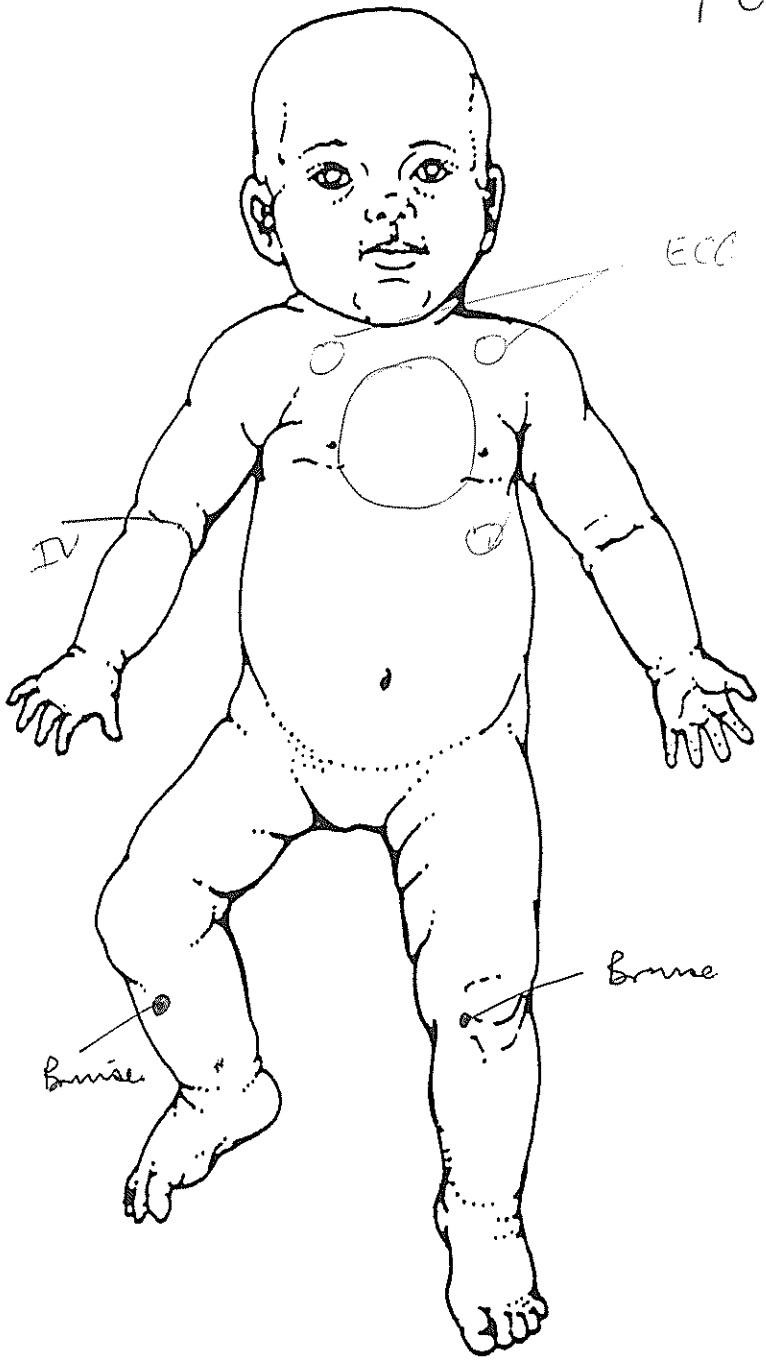
N.S.W. INSTITUTE OF FORENSIC MEDICINE

99-9322

FOLBIGG

HC





NSW POLICE SERVICE
REPORT OF DEATH TO CORONER

Local Area Command: Hunter Valley LAC Phone No.02 65787499/60499
Report Date: 1 March, 1999
COPS Event No: Morgue Register/Book No.

The Coroner, Singleton Death of: Laura Elizabeth FOLBIGG

Sex: Female Age: 1 D.O.B: 07/08/97
Address: 8 Millard Close, Singleton Marital State: Single
Time and Date of Death: 12.45pm on 1/3/99
Place of Death: Singleton Hospital
Time and Date found: 12.05pm on 1/3/99
By whom found: Kathleen Megan FOLBIGG Address: 8 Millard Close, Singleton
Reported to Police by: Brian WADSWORTH Address: Singleton Ambulance
Last seen alive by: Kathleen Megan FOLBIGG Address: 8 Millard Avenue, Singleton
When last seen alive: 11am 1.3.99
Time & Date Reported: 12.55pm 1.3.99 Occupation: Child
Deceased a native of: Australia Strait Islander/Aborigine: Not Applicable
Pension type: Govt. Authority Informed: New South Wales Police Service
Military/Invalid Disability: Not Applicable
Next of Kin: Craig Gibson FOLBIGG Relationship to deceased: Natural Father
Address: 8 Millard Close, Singleton Phone: 02 65721624
Identifying person: Craig Gibson FOLBIGG Address: 8 Millard Avenue, Singleton
Deceased's doctor: Paul INNIS of Singleton Heights Surgery, Singleton Phone: 02 65731577
Method of Identification (Visual, Dental, F/prints): Visual
Chain of Id (ie. Relative or Friend (name) to Police (name) to other Police (name): Natural father to Detective Ryan
Criminal Charges Preferred: Suspicious Circumstances: Detectives Attending: Yes
Detective/s: D/S/C Ryan and D/Sgt WELLS LAC: Hunter Valley
Crime Scene: Yes Investigator/s: D/S/C Glen WARD Region: Hunter
Crash Investigation: No Investigator/s: Region:

Property/Clothing found on deceased: Disposable nappy, tights and shirt
Property Book Ref: Clothes with deceased Property/clothing disposal:
Authorised by: Phone:
Coroner's pamphlet handed to: John FOLBIGG

Note
(1) This form should be prepared in quadruplicate in all cases where a death is reported to the Corner. The original and two copies should be forwarded to the Coroner. All statements in duplicate should be lodged with the Coroner no later than 28 days after receipt of inquest notice.

Body Mgt. No 391361
4.13

Reporting Officer: Bernard Michael RYAN Investigating Officer: . . . .
Rank: Detective Senior Constable Rank: . . . . . .
Station: Hunter Valley LAC Station: . . . . . .
Annual Leave: . . . . . .
Signature: . . . . . .

Continued overleaf

CAT 81503

If any previous illness, and deceased seen by doctor, particulars should be given. Where treated by a doctor a note should be obtained giving particulars of treatment of such doctor. If died within 24 hours of Anaesthetic -

Narrative of circumstances under which death took place. Craig Gibson FOLBIGG (DOB: 21.11.61) and Kathleen Megan FOLBIGG (DOB: 14.6.67) are a married couple currently residing at 8 Millard Close, Singleton. Prior to this incident, all three of Mr and Mrs FOLBIGG's previous children have died whilst still in their infancy. These deaths were apparently attributed to SIDS, with Professor John HILTON of the Institute of Forensic Medicine being involved with the investigation of these deaths. These deceased children's names were Kaleb Gibson FOLBIGG, Patrick Allan FOLBIGG and Sarah Kathleen FOLBIGG.

On 7 August, 1997 Mr FOLBIGG gave birth to Laura Elizabeth FOLBIGG at the Singleton Hospital. At the time the child weighed approximately 7lb and 3/4 ounces and was born full term. For the first twelve months of her life Laura's sleeping habits were monitored by medical staff at the Westmead Hospital. (Ms Margaret TANNER - Westmead Hospital). Her family doctor was Doctor SANDERS of Singleton and Doctor INNIS of Singleton Heights Medical Practice. The child's last visit to a doctor was with Doctor INNIS in early February, 1999 for her 18 month vaccination.

About 6.20am on 1/3/99 the child awoke at the family home and was suffering from flu/cold symptoms which had plagued her for the previous seven days. Mrs FOLBIGG had given the child approximately 3ml of Demozin on 27.2.99 for these symptoms and this was the last medication that the child had taken for the illness. Mrs FOLBIGG stated that her child appeared in a bad mood, however did not appear to be seriously ill. During the morning Mrs FOLBIGG dressed the child in a disposable nappy with a pair of floral tights and a pink T shirt. She then took her to a Singleton gymnasium and to visit Mr FOLBIGG at his place of employment. About 11am that day Mrs FOLBIGG returned to her home with her child who had fallen asleep whilst travelling in the car on the way home. Once at the family home Mrs FOLBIGG placed her in the child's bed which is inside the child's own room. She placed her onto the bed so she was laying on her right side on top of a downer on the single bed. She also placed a pollen rug over the torso of the child.

About half an hour to an hour later Mrs FOLBIGG was inside the house when she heard the child coughing. This cough did not cause any alarm to Mrs FOLBIGG and she waited approximately five minutes until she went into her child's room to check on her. When she entered the room she saw her child laying on her back on the bed and her face was extremely pale. She checked her and saw that she wasn't breathing. She immediately picked her up, carried her to the breakfast bar in the kitchen and commenced CPR. During this process Mrs FOLBIGG also telephoned 000 and raised the alarm.

At 12.14pm that day Singleton ambulance officers attended the premises and observed Mrs FOLBIGG performing CPR on the deceased. They commenced treatment and found that the child had no pulse and was not breathing. They continued attempts to revive the child during a journey to the

The contents of this document is based on the initial information obtained when Police attend a death. Because of this, inaccuracies may come to light at a later date.

Name: Bernard Michael RYAN Signature: .....

Rank: Detective Senior Constable Date: 1 March, 1999
Registered No: 25495

Singleton Hospital. These attempts were unsuccessful as were the attempts by Doctor Tuan AU who pronounced life extinct at 12.45pm that day.

Singleton detectives attended the Singleton Hospital at 1.30pm that day and conducted preliminary interviews with both Mr and Mrs FOLBIGG. Mr FOLBIGG formally identified the deceased to Detective Senior Constable RYAN. At this time it was noticed that skin to the face and back was discoloured - possibly due to lividity. This discolouring appeared more severe to the left side of the deceased.

Detective WARD of Maitland Crime Scene Unit attended 8 Millard Close, Singleton and examined the scene. The bedding was seized and entered as exhibits at Singleton Police Station. It was apparent that the house occupied by Mr and Mrs FOLBERG appeared to be a healthy environment with furniture and fixtures modified for child safety. The house was a relatively new structure and did not appear to contain high levels of dust or drafts.

The Singleton coroner, Mr Ron WOODROW was informed of this incident by telephone and the deceased was conveyed to Glebe by Government contractors.

Reporting Officer: Bernard Michael RYAN Investigating Officer: . . . .
Rank: Detective Senior Constable Rank: . . . . . .
Station: Hunter Valley LAC Station: . . . . . .
Annual Leave: . . . . . .

Signature: . . . . . .

Continued overleaf

NSW INSTITUTE OF FORENSIC MEDICINE
INTERIM REPORT

To State Coroner Date: 1 MAR 99

Laura Elizabeth FOLBIGG 99/9322

GIVEN NAMES SURNAME CASE No.

Interim Cause of Death:

I. Direct Cause –
Disease or condition directly leading to death (a) Undetermined
(due to or following)

Antecedent Causes –
Morbid conditions, if any, giving rise to the above cause, stating the underlying condition last (b) .....
(due to or following)

(c) .....

II. Other significant conditions contributing to the death, but not relating to the disease or condition causing it .....

Significant Injuries Were Not Present ☑

Significant Injuries Were Present ☐

Specify below if not mentioned in Cause of Death:

.....

.....

Further investigations are being performed:

Specimens retained at IOFM		*Forensic Science Laboratories*		*Analytical Laboratories*		*Other Laboratories*	
Samples of tissue for histology	☑	Blood	☐	Blood	☑	Blood for virology	☐
Whole organs for examination:		Hair	☐	Liver	☑	Bacterial culture	☑
Brain	☑	Fingernails	☐	Stomach contents	☑	Viral culture	☐
Heart	☐	Oral/rectal/vaginal swabs	☐	Urine	☑	Biochemistry	☑
Other	☐	Other	☐	Bile	☑	Other	☐
Blood for storage	☐			Vitreous	☐		
				Other	☑		

Remarks:

The body may be released ☐

The body may not be released ☑ Needs XRays

Give reason for non-release:

Signature: AD Cala

Print Name: A.D. CALA

CENTRAL SYDNEY LABORATORY SERVICE

NSW INS FORENSIC MEDICINE
PO BOX 90
GLEBE NSW 2037

Requested by: CALA, A

Patient No: (2247)0999322
Name: FOLBIGG, LAURA
Sex/Age: FEMALE 01/01/98 14 MOS
Location: NSW FORENSIC MED

DEPARTMENT OF MICROBIOLOGY

Dr R Benn, Dr C MacLeod. Enquiries: 9515 8278

Fluids

CSF CULTURE MB-99-014698 COLLECTED:02MAR99
SOURCE: CEREBROSPINAL FLUID RECEIVED: 02MAR99 0904

PRELIMINARY

No growth overnight.
Further incubation in progress.

Miscellaneous

TISSUE/BIOPSY CULTURE MB-99-014696 COLLECTED:02MAR99
SOURCE: SPLEEN RECEIVED: 02MAR99 0904

PRELIMINARY

Moderate coliforms of 2 colonial types
Profuse alpha haemolytic Streptococcus
Moderate Staphylococcus epidermidis (presumptive)
Further incubation in progress.

Printed: 03MAR99 1327 *** End of Report *** Page: 1

Ref No: MB-99-014696

CENTRAL SYDNEY LABORATORY SERVICE

NSW INS FORENSIC MEDICINE
PO BOX 90
GLEBE NSW 2037

Requested by: CALA, A

Patient No: (2247)0999322
Name: FOLBIGG, LAURA
Sex/Age: FEMALE 01/01/98 14 MOS
Location: NSW FORENSIC MED

DEPARTMENT OF MICROBIOLOGY

Dr R Benn, Dr C MacLeod. Enquiries: 9515 8278

Fluids

CSF CULTURE MB-99-014698 COLLECTED:02MAR99
SOURCE: CEREBROSPINAL FLUID RECEIVED: 02MAR99 0904

PRELIMINARY

No growth overnight.
Further incubation in progress.

Faeces

STOOL CULTURE MB-99-014699 COLLECTED:02MAR99
SOURCE: RECTAL SWAB RECEIVED: 02MAR99 0918

AMENDED REPORT

Profuse normal enteric flora isolated.
No Salmonella/Shigella/Campylobacter isolated.

Miscellaneous

TISSUE/BIOPSY CULTURE MB-99-014695 COLLECTED:02MAR99
SOURCE: LUNG RECEIVED: 02MAR99 0904

PRELIMINARY

Profuse Post mortem contaminants.
Further incubation in progress.

TISSUE/BIOPSY CULTURE MB-99-014696 COLLECTED:02MAR99
SOURCE: SPLEEN RECEIVED: 02MAR99 0904

PRELIMINARY

Moderate coliforms of 2 colonial types
Profuse alpha haemolytic Streptococcus of 2 colonial types
Moderate Staphylococcus aureus (presumptive)
Identification and susceptibilities proceeding.

Printed: 04MAR99 1326 *** End of Report ***

Ref No: MB-99-014

CENTRAL SYDNEY LABORATORY SERVICE

NSW INS FORENSIC MEDICINE
PO BOX 90
GLEBE NSW 2037

Requested by: CALA, A

Patient No: (2247)0999322
Name: FOLBIGG, LAURA
Sex/Age: FEMALE 01/01/98 14 MOS
Location: NSW FORENSIC MED

DEPARTMENT OF MICROBIOLOGY

Dr R Benn, Dr C MacLeod. Enquiries: 9515 8278

Fluids

CSF CULTURE MB-99-014698 COLLECTED:02MAR99
SOURCE: CEREBROSPINAL FLUID RECEIVED: 02MAR99 0904

FINAL REPORT

No growth

Faeces

STOOL CULTURE MB-99-014699 COLLECTED:02MAR99
SOURCE: RECTAL SWAB RECEIVED: 02MAR99 0918

AMENDED REPORT

Profuse normal enteric flora isolated.
No Salmonella/Shigella/Campylobacter isolated.

Miscellaneous

TISSUE/BIOPSY CULTURE MB-99-014695 COLLECTED:02MAR99
SOURCE: LUNG RECEIVED: 02MAR99 0904

FINAL REPORT

Profuse Post mortem contaminants.
Profuse coliform

Ref No: MB-99-014696

CENTRAL SYDNEY LABORATORY SERVICE

NSW INS FORENSIC MEDICINE
PO BOX 90
GLEBE NSW 2037

Requested by: CALA, A

Patient No: (2247)0999322

Name: FOLBIGG, LAURA
Sex/Age: FEMALE 01/01/98 14 MOS

Location: NSW FORENSIC MED

DEPARTMENT OF MICROBIOLOGY

Dr R Benn, Dr C MacLeod. Enquiries: 9515 8278

Miscellaneous

TISSUE/BIOPSY CULTURE MB-99-014696 COLLECTED:02MAR99
SOURCE: SPLEEN RECEIVED: 02MAR99 0904

FINAL REPORT

Moderate coliforms of 2 colonial types
Profuse alpha haemolytic Streptococcus of 2 colonial types
Moderate Staphylococcus aureus

SUSCEPTIBILITIES

	S.aureus MIC	INTERP
Penicillin	<=0.125	S
Flucloxacillin	<=4	S
Cefazolin	<=4	S
Eryth/Clinda	<=1	S

Printed: 09MAR99 1327 *** End of Report *** Page: 2

2N

EXPERT CERTIFICATE in the mater of:

Police - v -

Place: Date:

Name: John William CASH

Address: 16 Broughton Street, SINGLETON NSW 2330 Tel. No: (02) 6571 1077

Occupation: Medical Practitioner States:

EXPERT CERTIFICATE
Section 177, Evidence Act 1995 No. 25

1. This statement made by me accurately sets out the evidence which I would be prepared, if necessary, to give in court as a witness. This statement is true to the best of my knowledge and belief and I make it knowing that, if it is tendered in evidence, I shall be liable to prosecution if I have wilfully stated anything which I know to be false or do not believe to be true.

2. I am 54 years of age.

3. I hereby certify:
My full name is John William Cash.
My contact address is 16 Broughton Street, Singleton, NSW 2330.
I have a specialised knowledge based on the following training, study and experience:-
30 years experience as a Medical Practitioner.
MBBS FRACGP
Visiting Medical Officer at Singleton Hospital.

4. At about 1.00 am on 22 June 1998 at Singleton Hospital, I examined Laura Folbigg. She was said to have had a slight upper respiratory infection for several days and had developed a croupy cough that night. On examination the child showed no distress and no signs of respiratory difficulties. Her chest was clear. Her throat contained a small amount of mucus. Her ears were clear. She had no neck stiffness and her abdomen was soft. She was afebrile.

I diagnosis of Upper Respiratory Tract Infection and mild croup was made.

Witness:..... Signature:.....
9/8/99

In view of the family history of three siblings having died with Sudden Infant Death Syndrome, she was admitted for observation. She was treated with intranasal oxygen and observed overnight.

On review later that morning her observations, having remained stable, and appearing to be in no distress, she was discharged home. Her parents were asked to take her to her usual local medical officer for follow up.

She was reviewed on 23 June 1998 at my surgery. She had signs of an upper respiratory infection but was in no distress. As outlined in our records, she was seen on several occasions from September 1997 until August 1998 with minor signs of upper respiratory infection never requiring antibiotics.

On one occasion she had vomiting and diarrhoea which settled on conservative management.

Laura was up to date with her routine immunisations which had been done at the surgery.

Witness:.....Signature:.....

9/8/99

2m

EXPERT CERTIFICATE in the matter of:

Police -v-Folbigg

Place: Singleton **Date:**

Name: Paul Innis
Address: 32 Fleet Street BRANXTON NSW 2335 **Tele. No:** (02) 6573 1577

Occupation: General Medical Practitioner **STATES:-**

EXPERT CERTIFICATE
Section 177, Evidence Act 1995 No. 25

1 This Statement made by me accurately sets out the evidence which I would be prepared, if necessary, to give in court as a witness. The statement is true to the best of my knowledge and belief and I make it knowing that, if it is tendered in evidence, I shall be liable to prosecution if I have willfully stated anything which I know to be false or do not believe to be true.

2 I am 31 years of age.

3 I hereby certify:

My full name is Paul Francis Innis
My contact address is 32 Fleet Street BRANXTON NSW 2335

I have specialized knowledge based on the following training, study and experience:-

MBBS Sydney University 1995.

4 My medical consultations with Laura Folbigg were as follows:
The 14 of August 1998 Laura was aged One. She had had Flu like symptoms for Five days with symptoms of coughing, sleep disturbance, had been of her food, had no fevers and was continuing to have wet nappies. She had had two episode Croup in the past. Her back ground included no allergies and her immunisations were up to date. On examination chest clear no signs of respiratory distress and her throat was red. I diagnosed a viral upper respiratory track infection and advised Mum to treat her symptoms with Panadol and fluids.

The 19th of October 1998 Laura presented with a burn on her left forearm and palm. The burn was superficial with the skin sloughing off and a plan of daily dressing was instituted. Each day over the next Eight days the burn dessing was changed and the wound was healing well I last saw her in relation to the burn on th 30th October 1998 in which stage the burns had healed quite well

Signature _____ 15/3/99

Witness Therese Gray. 15/3/99

The 19th Januaray 1999 Laura presented with her Mum. Her Mum had claimed she had a rash present for five days. At that stage she was Seventeen months old. Her diet was normal she continued to have normal number of wet nappies. She had no upper respiratory track symptoms and was behaving normally. The rash was itchy. On examination she had a macular red rash and her throat was also red. The rash was distributed on her shoulders, upperarms and down her arms. I diagnosed a allergic rash and prescribed Phenergan to treat this rash. At that stage she weighed 12 kilos and a dose of 2.5 - 5mg of Phergan orally three times a day as needed was precripted I told Mum to bring her back for review if the rash didn't go away over the next few days.

Laura represented on the 22nd of January 1999 for review of the rash. She had fevers over the last few days. I diagnosed a viral rash and on examination her throat was red but no additional treatment was prescripted.

The 5th February 1999 Laura's Mum brought her in for her 18 month Immunisation. At that stage she was well. On examination her throat and ears were clear and I advised our Nurse she may have her 18 month Immunisation. She received her Hib vaccine and Infanrix.

5 Based wholly or substantially on the knowledge, I am of the opinion that

During the seven month period, in which I saw Laura Folbigg approximately 13 times I'm of the opinion that Laura was a normal and healthy child. I was not aware of any chronic illness or disabilities. Her death was completely unexpeted.

Dr Paul F Innis MBBS

Signature _____ 15/3/99

Witness Sheree Gray. 15/3/99

New South Wales Police **P.190**
v2.9

STATEMENT in the matter of:
Death of FOLBIGG children

Place: Morisset Police Station
Date : 1 September 1999

Name: Louise Ann ALDERSON
Address: 35 Lakeview Street, Boolaroo **Tel. No.:** 02 49586479
Occupation: Ambulance Paramedic **STATES:-**

1. This statement made by me accurately sets out the evidence which I would be prepared, if necessary, to give in court as a witness. The statement is true to the best of my knowledge and belief and I make it knowing that, if it is tendered in evidence, I shall be liable for prosecution if I have wilfully stated in it anything which I know to be false, or do not believe to be true.

2. I am 35 years of age.

3. I am a qualified Ambulance Paramedic with the Ambulance Service of New South Wales. I have been employed with the service for the past sixteen years with twelve of those years as a paramedic.

4. On Wednesday the 1st of September, 1999 I spoke with Detective RYAN at the Morisset Police Station. Detective RYAN showed me copies of two Ambulance Report Sheets, numbers Q001 and Q604. I read these two documents and recognised that they related to an incident which I attended at 9 Dower Close, Thornton at 1.48am on 30 August, 1993. At that time, I was stationed at Hamilton as a Paramedic and was performing duty with Ambulance Officers Robert FOXFORD and Rod AVERY. We attended this residence to assist Ambulance Officer Debra MARTIN who was treating a young child for a suspected Sudden Infant Death Syndrome.

5. I remember attending the premises to assist Debra who had

Witness: _____ **Signature:** Louise Alderson

been off at the premises for sometime. I walked into a bedroom of the house and saw a baby laying on the floor with Debra obviously treating the baby. The baby had a intra-ossessous needle inserted into one leg and attempts were being made to resuscitate the baby.

6. According to the notes made by Robert FOXFORD he, Rod or myself administered the following prescribed drugs via the intra-ossessous needle: 6ml of adrenaline, 15ml of sodium bicarbonate and 1 ml of calcium chloride. These drugs are the protocol for treatment due to a reading of asystole on the ECG monitor. From memory the asystole was recorded prior to my arrival at the scene and it was obvious that the baby was deceased prior to my arrival also.

7. I do not recall what the baby looked like on external examination. From reading Robert FOXFORDs' notes I can only state that they are a correct recording.

Witness: _____ **Signature:** Louise Alderson

B. R...
12/5/c
1.9.99

3s

New South Wales Police

STATEMENT in matter of: Death of FOLBIGG children

Place: Singleton Police Station

Date: 15 September 1999

Name: WADSWORTH, Brian **Tel No.:** 02 65732174

Address: 12 Gardiner Cr, Singleton

Occupation: Level Four Ambulance Officer

States:-

1. This statement made by me accurately sets out the evidence which I would be prepared, if necessary, to give in court as a witness. The statement is true to the best of my knowledge and belief and I make it knowing that, if it is tendered in evidence, I shall be liable to prosecution if I have wilfully stated in it anything which I know to be false or do not believe to be true.

2. My age is 37 years.

3. I am a level four ambulance officer attached to the Singleton Ambulance Station. I have been employed with the service for the past twelve years and reached level four qualification around 1991.

4. On Monday the 1st of March, 1999 I was working at the Singleton Ambulance Station with Ambulance officers Harold PICTON and Ted SMITH. I was the most qualified officer working that day.

5. At 12.12pm that day, Harold and I left the Ambulance Station responding to call of a child not breathing and CPR being performed. This call was to a house at 8 Millard Close, Singleton. Harold drove the ambulance vehicle straight to this address and we arrived there at 12.14pm that day.

6. As soon as Harold stopped the vehicle I alighted and carried the oxy-viva and drug box into the house via the front

Witness: _____ **Signature:** _____

door. I saw a woman leaning over a small child who was laying in the supine position on what appeared to be a breakfast bar. The woman appeared to be performing CPR on the child. The woman was crying and I think there was another woman in the house. I went straight up to the child, checked the vital signs and found that the child was not breathing and had no pulse. I looked inside the child's mouth and did not see any blood, vomit or foreign object. I tilted the child's head slightly ensuring that the airway was clear. I then continued CPR for a period. Harold then took over from me and continued CPR. I walked around to the other side of the breakfast bar on the kitchen side and placed an intravenous line into the child's right cubital fossa (inside right elbow). Whilst I was doing this the ECG monitor was applied to the child at 12.17.32 that day. This monitor showed that the child was in bradycardia. There baby still had no pulse at this stage. Harold resumed CPR and I obtained the appropriate drugs in accordance with the ambulance protocols. Around this time, I asked the woman who had been performing CPR, "Can you tell me what happened?"
She said, "I heard her coughing in the bedroom and when I checked her five minutes later I found her not breathing. "
She also indicated that the child had been suffering from a runny nose and coughing for a couple of days.

7. Between 12.19pm and 12.28pm that day I administered three doses of adrenalin 1:10,000 (2.5ml per dose) as per protocol. At this time, the child was placed on the stretcher and taken to the Ambulance vehicle parked in the driveway. Ted and I got into the back of the vehicle with the child and Harold drove. I administered another similar dose of adrenalin en route to the Singleton Hospital. We arrived at the Hospital at 12.32pm that day where the child was treated by Hospital staff. Shortly after the doctor pronounced life extinct.

Witness: _____ Signature: _____

8. When I was treating the child I noticed that her skin was warm to touch and cyanosis was present. By cyanosis I mean the child had a blue colouring around the lips and face.

9. At the hospital I completed Patient report V70724 in relation to this incident.

EXHIBIT: I NOW SEEK TO PRODUCE PATIENT REPORT V70724.

10. I also printed the ECG report relating to this incident whilst at the Hospital that day.

EXHIBIT: I NOW SEEK TO PRODUCE THAT ECG REPORT.

Witness: _____ Signature: _____

B. Ryan
D/S/C
15.9.99

Witness: _____ Signature: _____

3T

Ambulance Service of New South Wales

PATIENT REPORT

V 70724

INCIDENT NO. 4934

STATION NUMBER	RADIO NUMBER	DAY OF WEEK	DATE
31800	265	MONDAY	01/03/9

TITLE	PATIENT'S SURNAME	FIRST NAME	D. o. B.	SEX	Yr if > 2	AGE Mo
BABY	FOLBIGG	LAURA	07 08 97	F	2	1

ADDRESS: 8 MILLARD CL SINGLETON POST CODE 2330

CHARGE Km: 12 AMOUNT

PENSION / CONCESSION NUMBER

DEBTOR CODE 7 2 8 0 3 TITLE

BUSINESS NAME OR GUARDIAN IF PATIENT < 18 YRS

ADDRESS POST CODE

CHARGE CODE: 1 PENSION 2 HEALTH FUND 3 SCHOOL SUBS OR GROUP 4 WORKERS COMPENSATION 5 HOSPITAL CONTRACT 6 HOSPITAL CONTRACT: 1ST OR 2ND STAGE 7 AIR AMBULA

PATIENT'S LOCATION: ADDRESS CODE 2330

TRANSPORTED ? NO ▶ TREATMENT ONLY REFUSED TRANSPORT DECEASED : NIL TREATMENT

YES ▶ TO SINGLETON HOSP. CODE 9101

ODO IN	200729
ODO OUT	200717
TRIP Km	12

ARE YOU THE FIRST AMBULANCE CREW ON LOCATION ? ✓ YES NO

AUTHORITY / M.R.N. PUBLIC.

CALL PRIORITY 1 ✓ 2 3

PATIENT CATEGORY 1 OF 1 71

Appointment Time	BOOKED	OUT	LOCATION	DEPART	DESTINATION	CLEAR	STATION
	1208	1212	1214	1229	1232	1329	1341

HISTORY

Chief Complaint: NIC VITAL SIGNS CALLED TO CHILD NOT BREATHING - CPR BEING PERFORMED BY MOTHER DOING CPR ON CHILD ON KITCHEN BENCH. ON A. PT C. NIC RUBS, NIC RESPS, PUPILS DILATED WARM TO TOUCH, CYANOSIS RESIDENT. MOTHER STATED CHILD HAD BEEN HEARD COUGHING & WHEN CHECKED APPROX 5 MIN LATER FOUND NOT BREATHING & MOTHER THEN COMMENCED CPR. ON NIC RESPONSE TO A.

PRIOR TO ARRIVAL
- ☐ Conscious ☐ Unknown
- ✓ Unconscious

ALLERGIES
- ☐ Nil ✓ Unknown

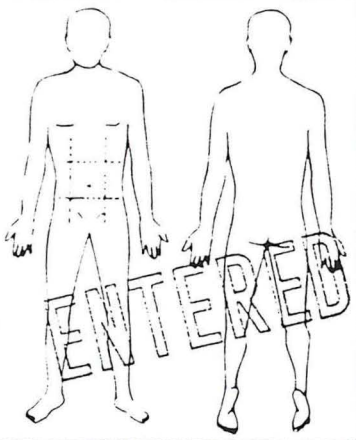
PREVIOUS ILLNESS
- ☐ Nil ✓ Unknown
- ☐ Cardiac ☐ Respiratory
- ☐ Diabetes ☐ Hypertension
- ☐ Stroke ☐ Epilepsy
- ☐ G.I.T ☐ Other
- ☐ Renal ☐ Psychiatric

PRESENT MEDICATION
- ☐ Nil ✓ Unknown

ASSESSMENT

AIRWAY	Clear	✓ Obstructed
BREATHING	Present	✓ Absent
	Shallow	Deep
CIRCULATION	Present	✓ Absent
Buccal Mucosa	Pink	Pale
		✓ Blue
Neck Veins	Normal	Distended
Skin Temp.	✓ Normal	Hot Cold
Sweating	✓ Nil	Moderate Profuse
Blood Loss	✓ Nil	< 500 mls > 500 mls
Burns	✓ Nil	%
Vomiting	✓ Nil	Yes
Fitting	✓ Nil	Yes

TRAUMA



Fracture
Laceration
Abrasion
Swelling
Haemorrhage
Tenderness
Contusion
Dislocation
Burns
Pain

ROAD CRASH
1 Driver
2 Passenger
3 Pedal Cyclist
4 Motorcyclist / Pillion
5 Pedestrian

X Position of Patient
> Point of Impact

Estimated Impact Speed: Low Medium High

Seatbelt / Helmet : Worn Not Worn Unknow

Time	Pulse	Blood Pressure	Resp. Rate	Glasgow Coma Scale: Verbal Response + Motor Response + Eye Opening = Score	Pupils: Size R	Size L	React R	React L	When applicable: BS L	BS R	BSL mmol / l	SaO2 %
1215	NIC		NIC	1 + 1 + 1 = 3	●	-	●		X	X		
				+ + =								
				+ + =								
				+ + =								
				+ + =								

Temperature °C per

OBSERVATIONS

Verbal: 5 Oriented, 4 Confused, 3 Inappropriate, 2 Incoherent, 1 Nil
Motor: 6 Obeys, 5 Localises, 4 Withdraws, 3 Flexion, 2 Extension, 1 Nil
Eye Opening: 4 Spontaneous, 3 To Voice, 2 To Pain, 1 Nil

● Dilated Normal ○ Constricted > Larger < Smaller = Equals

+ Present − Absent

Breath Sounds: N Normal, D Decreased, W Wheezes, C Crackles, X Nil

OFFICE COPY

ATTACH RHYTHM STRIPS HERE

PATIENT MANAGEMENT

AIRWAY	1	✓ Suction	2	✓ Oral Airway	3	Nasal Airway	4	ETT mm	5	Other	6	Intubatio Unsucce	
BREATHING	1	Pressure Head	2	✓ Bag / Mask	3	✓ IPPV	4	Ventilator	5	✓ Other			
CIRCULATION	1	✓ E.C.M.	2	MAST (inflated) *Pressure :*			3	Other					
POSTURE	1	Sitting			2	Lateral			3	✓ Supine			
O_2 THERAPY	1	28% Ventimask	2	High Conc. Mask	3	Low Flow	4	High Flow	5	100% Demand	6	Other	
ANALGESIA	1	Entonox Effective	2	Entonox Not Effective	3	Narcotic Effective	4	Narcotic Not Effective					
SPLINTS	1	Traction	2	Cervical	3	KED / RED	4	Other					
STRETCHER	1	Carry Sheet	2	Spine Board	3	Jordon Frame	4	Scoop	5	Stokes	6	✓ Other	
TOURNIQUET	1	Lymphatic	2	Arterial		Time On :							
PROTOCOLS		16											
DRUGS / FLUIDS		201											
TRIALS	A:		B:		C:			Protocol Variation					

DRUGS / FLUIDS / SHOCKS

IV Cannula : 20g.

TIME	ECG	TREATMENT	DOSE	ROUTE	SHOCK	RESULT
1219		ADRENALINE 1:10,000	2.5ml	IV	/	NIL
1226		ADRENALINE 1:10,000	2.5ml	IV	/	NIL
1229		ADRENALINE 1:10,000	2.5ml	IV	/	NIL
1232		ADRENALINE 1:10,000	2.5ml	IV	/	NIL
		KIDS DOSES FLUSHED W/ 0.9% NACL				

E.T.T. Checked : ☐ Post Intubation ☐ Arrival at Hospital

COMMENTS

RHYTHMS MONITORED

1 BRADYCARDIA
2 SINUS RHYTHM
3 TACHYCARDIA
4 VT
5 VF
6 ✓ ASYSTOLE
7 ✓ OTHER
8 MULTIPLE RHYTHMS

CARDIAC ARREST

Yes	No	
1 ✓	2	CPR Prior to Arrival
1	2	Effective
1	2 ✓	Witnessed

INITIAL RHYTHM

1 VT 3 Asystole
2 VF 4 ✓ Other

N/A Time of First Shock
NIL Total Shocks

TRAUMA TRIAGE

TRANSPORT DECISION (1 - 7) OTHER COMPLAINT
MAIN COMPLAINT (V 1 to M12) OTHER COMPLAINT

PATIENT'S CONDITION AT HOSPITAL

1 IMPROVED 3 DETERIORATED
2 UNCHANGED 4 ✓ RESUSCITATION CEASED IN EMERGENCY ROOM

Print Names	Signature	Emp. No.	Level
Officer 1 driving H. PICTON		710448	3
Officer 2 reporting G. WADSWORTH		W1629	4
Officer 3 Dr Other T. SMITH		S10553	3

SKILLS

Drugs	MAST	Defib	I.V	I/Osseous	T/P thorax	Intubate
✓			✓			

Details checked by Officer | Initial | Report checked by S.O | COMPLETE LEGIBLE CORRECT | NO | YES | Checked by | Punched by | Initial | Medical Review | Initial

Form

...E 300D ...GE REPORT

| DATA |

INCIDENT DATE: 01/08/99
CALL TIME ...: 12:12
AGENCY/UNIT: 265
PERSONNEL...: Boron / Woodworth / Smith

| EPISODE DATA |

PATIENT NAME.: LAURA FOLBIGG.
M( ) DATE OF BIRTH: 07/08/97
INCI[illegible] 4934
NOTES: CARDIAC ARREST.

| REPORT DATA |

EPISODE DATE..... 1 MAR 99
EPISODE TIME.....12:17:01
SOURCE FORMAT..... MCM
REPORTED BY... Q REPORTER
SOFTWARE VERSION... D

| DEVICE DATA |

SERIAL NUMBER..... 003666
PROTOCOL..... MANUAL
SOFTWARE VERSION..... G
CATALOG NO. 920000

EVENT LOG:

1 MAR 99

12:17:01 UNIT ON
12:17:30 DEFIB. ELECTRODE
12:25:45 "CHECK PATIENT" GIVEN
12:26:00 "CHECK PATIENT" [illegible] ARES
12:32:51 "CHECK PATIENT" G[illegible]EN
12:33:06 "CHECK PATIENT" CLEARED
12:40:37 UNIT OFF

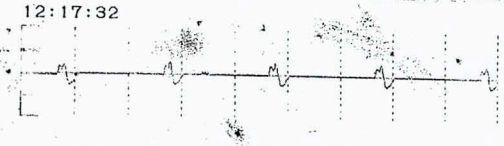
ELAPSED TIME EVEN[illegible]

00:00:00 UNIT [illegible]:01
0:29 [illegible]

RECORDED ECG

CODE: 1 MAR 99 12:17:01 *PAGE 1

12:17:32



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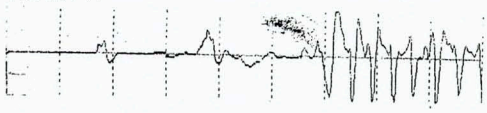

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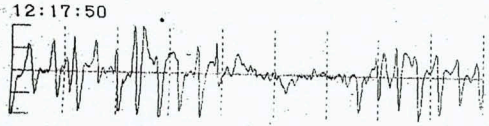

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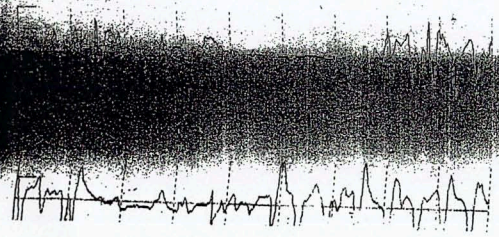

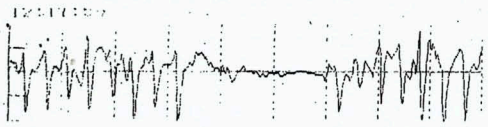

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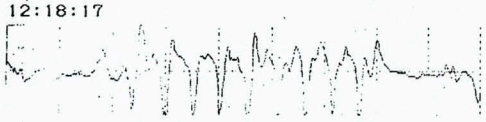

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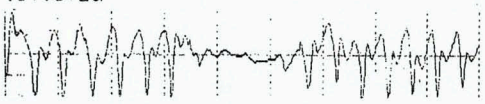

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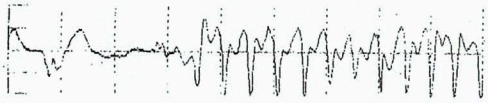

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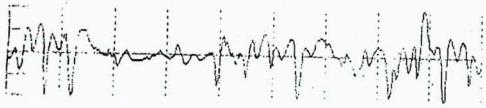

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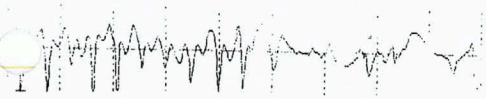

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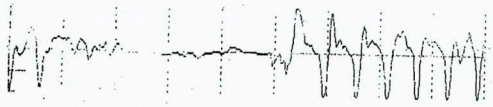

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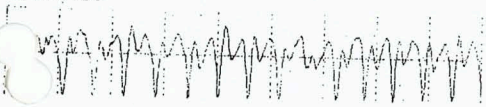

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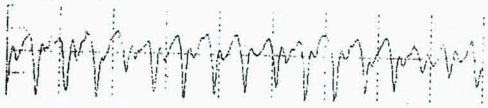

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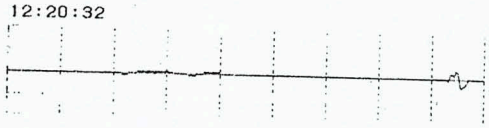

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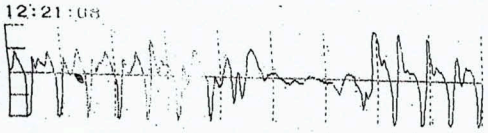

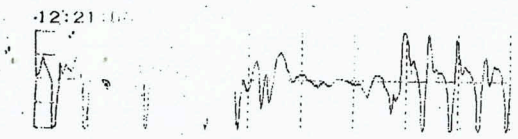

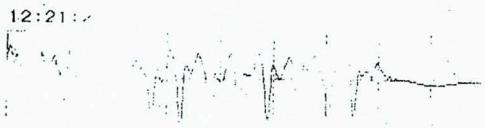

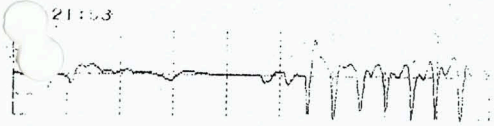

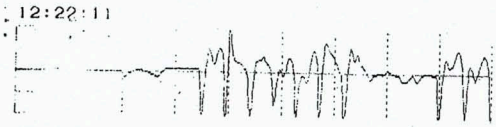

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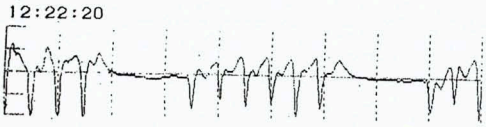

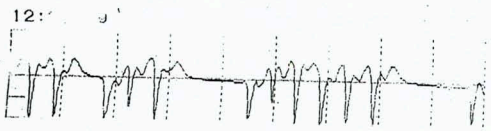

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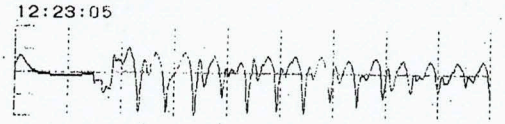

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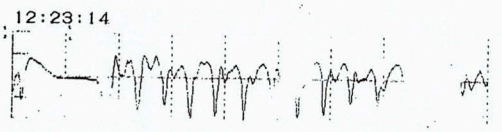

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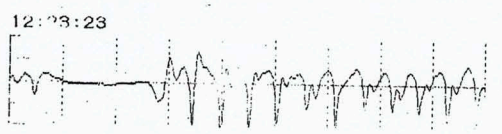

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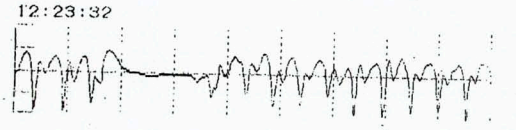

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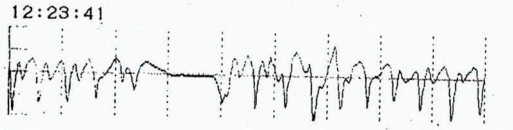

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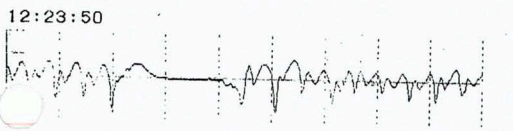

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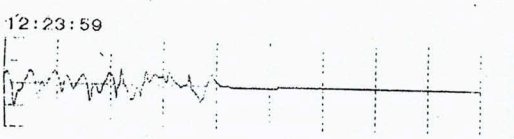

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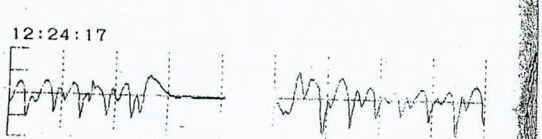

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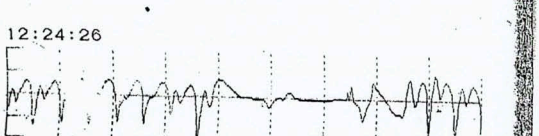

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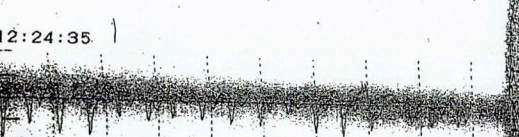

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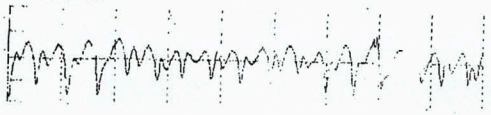

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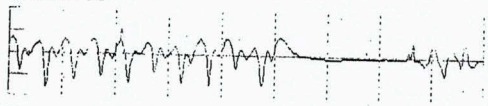

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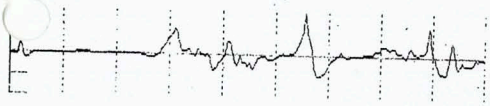

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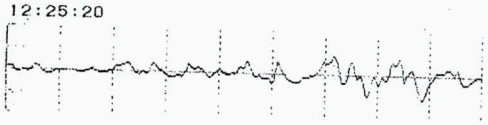

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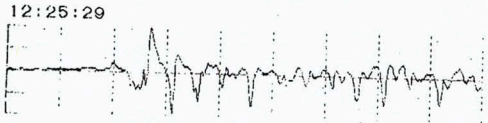

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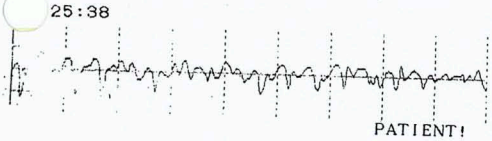

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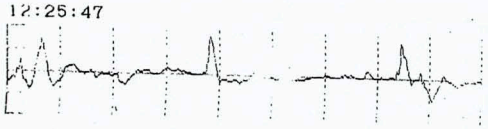

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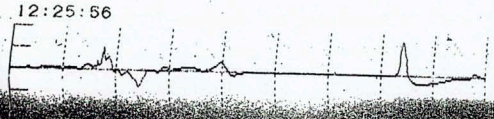

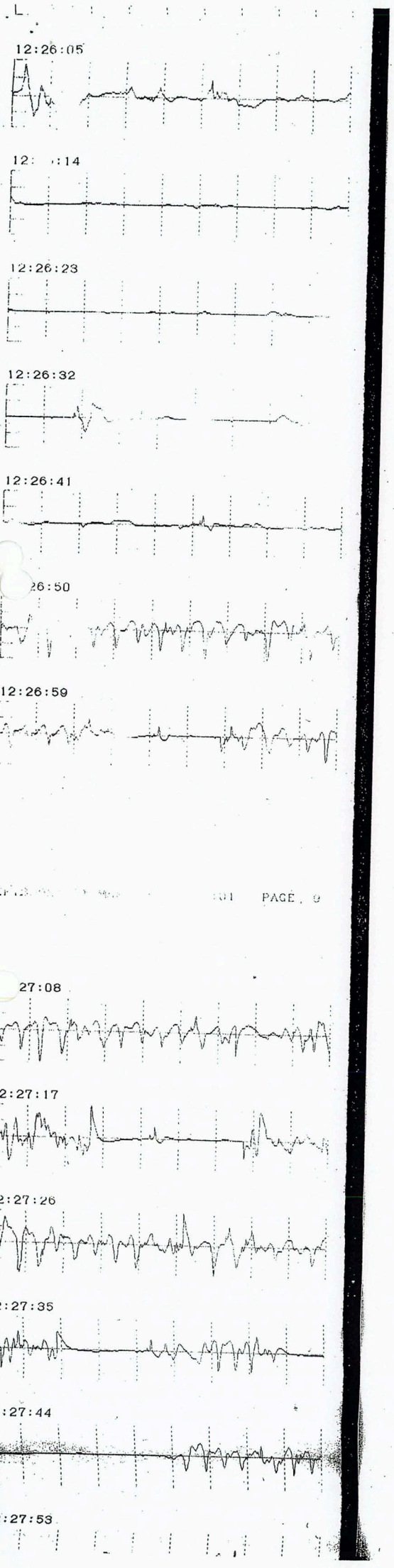
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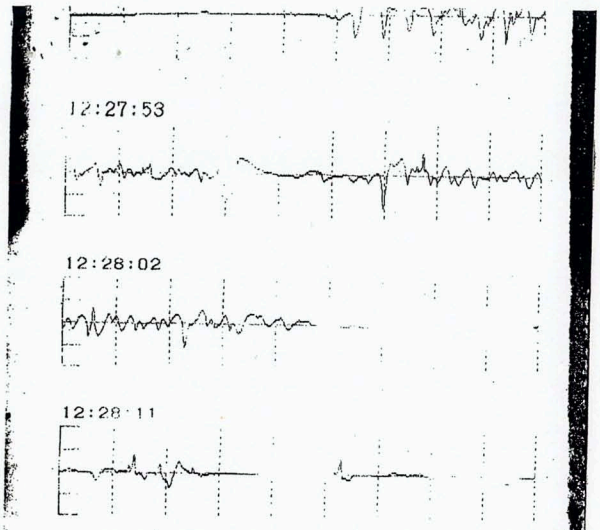

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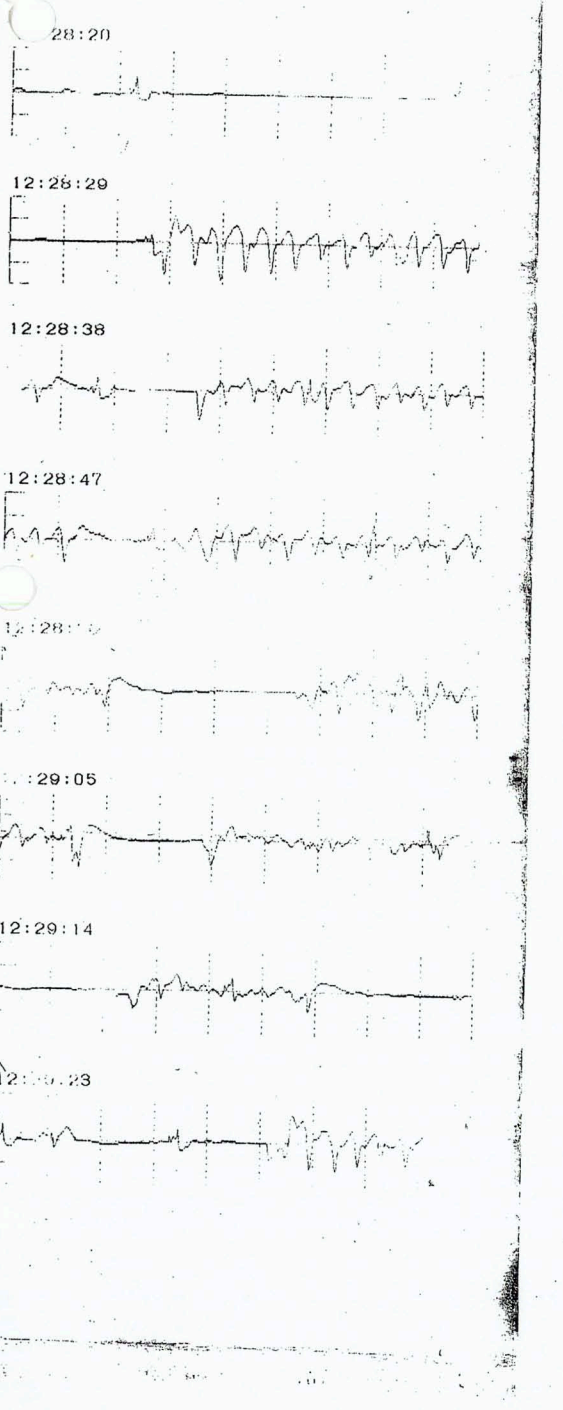

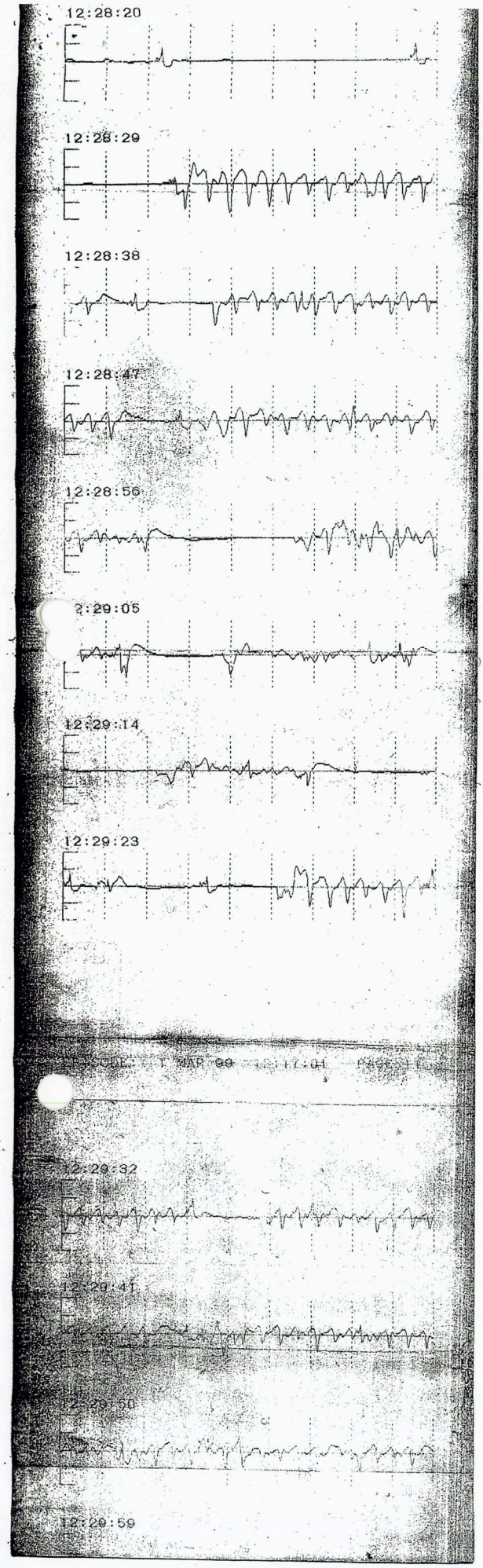
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New South Wales Police

STATEMENT in matter of: Death of FOLBIGG children

Place: Singleton Police

Date: 15 September 1999

Name: PICTON, Harold Francis **Tel No.:** 02 65722455

Address: 95 George Street, Singleton

Occupation: Ambulance Officer - Station Officer

States:-

1. This statement made by me accurately sets out the evidence which I would be prepared, if necessary, to give in court as a witness. The statement is true to the best of my knowledge and belief and I make it knowing that, if it is tendered in evidence, I shall be liable to prosecution if I have wilfully stated in it anything which I know to be false or do not believe to be true.

2. My age is 57 years.

3. I am the Station Officer in charge of the Singleton Ambulance Station. I am a level three ambulance officer and have been in the service for the past twenty nine years.

4. On Monday the 1st of March, 1999 I was performing duty at the Singleton Ambulance Station with Ambulance Officers Brian WADSWORTH and Ted SMITH. Brian is a level four officer and Ted is a level three.

5. At 12.12pm that day, Brian and I left the Station en route to 8 Millard Close, Singleton to respond to a twenty month old child not breathing at that location. Apparently the mother of the child was performing CPR at scene.

6. At 12.14pm that day, we arrived at the address. Brian as the treating officer entered the house first whilst I call on scene and obtained certain equipment. I walked into the house about thirty seconds behind Brian and I saw him leaning over a

Witness: _____ Signature: _____

small child who was laying in the supine position on a breakfast bar inside the house. He was performing CPR on the child and I saw a woman sitting on a chair behind Brian and she appeared upset. She was screaming and crying.

7. I took over from Brian and checked for a pulse and breathing on the child. I couldn't identify either, so I continued CPR. At this time I noticed that the child was wearing a pair of floral tights and a small top. She was warm to touch. The ECG monitor was attached to the child and registered asystole. Brian administered the protocol (Drugs) to the child via an intravenous line which failed to restore a cardiac output. The ECG monitor was still registering asystole. A short time later Ted SMITH arrived and assisted with CPR.

8. At 12.29pm that day the child was placed in the ambulance. Ted and Brian continued to treat the child whilst I drove the ambulance vehicle to the Singleton Hospital. At 12.32pm that day we arrived at the Hospital where the child was treated by Doctor AU and other medical staff. Life was pronounced extinct at the Hospital and the oral airway was removed. Brian completed the Patient Report (incident number 4934) and I signed the report as the driver attending the incident. I noticed that Brian reported conversation from the mother of the child. I cannot confirm this conversation because I can't remember any conversation about the child's history. I was concentrating on treating the child and was not listening to the mother at the house.

Witness: _____ Signature: _____
B. Ryan D/S/C

Witness: _____ Signature: _____

2L

EXPERT CERTIFICATE in the matter of: Kathleen FOLBIGG
Police -v-
Place: Westmead Coroners Office **Date:** 23/11/99

Name: Christopher SETON
Address: 5/3 Pacific Ave, Tamarama **Tel.No:** 02 98453437
Occupation: Paediatrician **STATES:-**

EXPERT CERTIFICATE

Section 177, Evidence Act 1995 No. 25

1. This statement made by me accurately sets out the evidence which I would be prepared, if necessary, to give in court as a witness. The statement is true to the best of my knowledge and belief and I make it knowing that, if it is tendered in evidence, I shall be liable to prosecution if I have wilfully stated anything which I know to be false or do not believe to be true.

2. I am **42** years of age.

3. I hereby certify:
My full name is **Christopher Denis SETON.**
My contact address is **New Childrens Hospital Westmead.**
I have a specialised knowledge based on the following training, study and experience:-
I completed MBBS (Bachelor of Medicine/Surgery) in 1980, FRACP (Fellow of the Royal College of Physicians) in 1991 and have studied clinical and research aspects of Sudden Infant Death Syndrome since 1990. I am currently employed as Staff Specialist in the Sleep Disorders Unit at New Childrens Hospital at Westmead. I have performed these duties for the past five years.

4. I first met Craig and Kathleen FOLBIGG on the 3rd of May 1996 at my rooms at Randwick after they were referred to me by Doctor Quentin KING from Fairholme Surgery in Singleton. They presented at my office seeking advice about the potential risk of SIDS after

See Continuation Sheet ...

Witness: _____ **Signature:** _____

they had lost three of their previous children. I remember I was told by Mr and Mrs FOLBIGG that their previous children had all died of SIDS. During this consultation, I discussed many aspects relating to SIDS prevention and risk assessment that could be applied to any future children.

5. The history supplied by Mr and Mrs FOLBIGG was highly suggestive of familial clustering of obstructive apnoea. This included Mr FOLBIGG suffering heavy snoring as did his siblings, and Patrick and Sarah FOLBIGG also suffering from snoring.

6. In January 1997 I was notified by Mr FOLBIGG that he and his wife were expecting the birth of their fourth child. I wrote back to them offering a full investigation by the Sleep Disorder Unit in regards to their expected baby and the risk of SIDS. Mr and Mrs FOLBIGG agreed with this approach. I was later informed that Mrs FOLBIGG gave birth to her daughter, Laura Elizabeth FOLBIGG at the Singleton Hospital on 7 August 1997.

7. On Tuesday the 19th of August 1997 I examined Laura at the Sleep Disorders Unit at Westmead. Laura was admitted and underwent polysomnography (sleep study) which demonstrated mild central apnoea and no obstructive apnoea. Laura also underwent full biochemical, blood and metabolic investigation which were normal. Laura was discharged on the 21st of August 1997 and her parents were supplied a corometric home cardiorespiratory monitoring device which was designed to record and download breathing and heart information during Laura's sleep. Mr and Mrs FOLBIGG were instructed on CPR and how to operate the monitor.

8. Laura was monitored on the machine for approximately twelve months without complication. Initially there was evidence that Laura suffered from a mild central apnoea which improved and was totally normal by February 1998.

EXHIBIT: SEE SLEEP STUDY REPORT DATED 3RD FEBRUARY 1998.

9. Laura did not at any time show signs of suffering from obstructive apnoea which is a potentially inherited breathing disorder associated with SIDS.

10. In March 1998 Margaret TANNER who is a nurse/consultant with the Sleep Study Unit approached me with a letter addressed to her by Mr Craig FOLBIGG. I read this letter where Craig expressed concern that his wife was not utilising the monitor during Laura's daytime sleeps and was not as diligent as maybe she should. I wrote to Mr FOLBIGG and expressed my concern that he and his wife should continue to use the device due to the family history. I requested that they consult with me.

11. On the 30th of April 1998 I saw Mr and Mrs FOLBIGG with Laura in Sydney where it became clear to me that the monitoring was becoming tedious for both Mr and Mrs FOLBIGG. I encouraged them to continue monitoring Laura in all her sleeps until her first birthday and I asked to review Laura at that time. I did not see the FOLBIGG family after this day. I am not sure of the exact date when the monitor was returned to the New Childrens Hospital but it approximated Laura's first birthday.

12. In March 1999 I was informed that Laura had died whilst at home in Singleton.

13. Based wholly or substantially on the above knowledge, I am of the opinion that Laura FOLBIGG did not at any time fit the profile of a high risk SIDS patient. Laura suffered mild central apnoea which had completely resolved by six months of age.

14. I have spoken to Detective RYAN from the Singleton Police Station and supplied him with my complete medical file relating to

Laura Elizabeth FOLBIGG.

Witness: _____ Signature: _____

The New Children's Hospital
Royal Alexandra Hospital for Children

Chris Seton
Consultant Sleep and Respiratory Paediatrician
Tel: (02) 9845 3437 Fax: (02) 9845 3439

DEPARTMENT OF RESPIRATORY MEDICINE: SLEEP DISORDERS UNIT

SLEEP STUDY REPORT

CS:CF

Tuesday, 7 October 1997

RE: LAURA **FOLBIGG**, DOB: AUGUST 7, 1997
8 MILLARD CLOSE, SINGLETON. NSW. 2330
NCH.MRN: **0662860**

NAME OF STUDY: NOCTURNAL POLYSOMNOGRAM SLEEP STUDY.
DATE OF STUDY: THURSDAY, OCTOBER 2, 1997.

CLINICAL REPORT:
This study demonstrates moderate central apnoea of infancy. Sleep-disordered breathing has demonstrated a mild improvement since the previous sleep study. There are occasional mixed apnoeas and hypopnoeas, but the obstructive component of sleep-breathing is not severe, and there is no bradycardic response to apnoea or hypopnoea.

Home apnoea monitoring should continue and downloaded information from this monitor will be carefully observed by the New Children's Hospital. All other SIDS-protective factors should continue to be undertaken diligently. Extra precaution including serious consideration of hospitalisation, should be undertaken if Laura develops an upper respiratory tract infection. A further sleep study will be organised for two months time.

Chris Set—.

C. SETON,
STAFF SPECIALIST,
SLEEP DISORDERS UNIT

cc. Dr. D. Sanders, Fairholme Surgery, 16 Broughton Street, Singleton. NSW. 2330

The New Children's Hospital
Royal Alexandra Hospital for Children

Chris Seton
Consultant Sleep & Respiratory Paediatrician
Tel: (02) 9845 3437 Fax: (02) 9845 3439
Email: ChristS1@nch.edu.au

17th February, 1998

Sleep Study Report

Dr. D. Sanders,
Fairholme Surgery,
16 Broughton Street,
SINGLETON. N.S.W. 2330

Re: Laura FOLBIGG: D.O.B.: 7.8.97
8 Millard Close, Singleton. NSW 2330

Name of Study: Polysomnogram
Date of Study: 3rd February, 1998

Clinical Report

Her sleep breathing has now normalised. There is no evidence of upper airway obstruction in sleep. Sleep quality is excellent. Home apnea monitoring should continue, but a further Sleep Study will not be required unless there is evidence of new or changing sleep breathing symptoms.

The family should re-consult with Dr. Seton at their convenience during their next visit to Sydney, and ideally this should be undertaken in two to three months' time.

Chris Set.

Chris Seton,
Staff Specialist, Sleep Disorders Unit

18/03/98

DEAR MARGARET,

I HOPE THIS LETTER FINDS YOU WELL.
KATHY LAURA AND I ARE FINE.
LAURA IS GOING GREAT GUNS NOW THAT SHE HAS SHAKEN ALL THE LITTLE BUGS SHE HAD, SHE IS IN THE PROCESS OF LEARNING TO CRAWL AT THE MOMENT AND DOES NOT LIKE IT ONE BIT," WHY CRAWL WHEN MUM OR DAD CAN CARRY ME OR GET THINGS FOR ME" (I BET THAT'S WHAT SHE THINKS). WE HAD PIXIE PHOTO'S DONE AND I HOPE YOU DON'T MIND BUT I HAVE SENT YOU ONE.
I'M SORRY IT ISN'T ANY BIGGER BUT AT LEAST IT WONT GET BENT IN THE MAIL.
OUR LAST REPORT ON LAURA'S SLEEP STUDIES WAS VERY GOOD AND I WAS PLEASED TO SEE THAT CHRIS WANTED THE HOME MONITORING TO CONTINUE.
I PERSONALLY FIND THE FLASHING LIGHTS OF THE MACHINE COMFORTING ALTHOUGH THE ALARMS ARE FRIGHTENING AS YOU ARE TOTALLY UNAWARE OF WHAT AWAITS YOU WHEN YOU GET TO LAURA. HAPPILY SO FAR ALL HAS BEEN FINE.
STRANGELY THOUGH I FEEL THAT KATHY FINDS IT ALL TEDIOUS AND FRUSTRATING AND WOULD PROBABLY RATHER NOT USE IT AT ALL, MERELY ENTRUSTING LAURA'S SURVIVAL TO FATE!
YOU WOULD THINK THAT AFTER ALL SHE HAD BEEN THROUGH AS A MOTHER SHE OF ALL PEOPLE WOULD BE MORE DILIGENT WITH THE MONITORING.
IS IT NECESSARY THAT LAURA BE MONITORED THROUGH HER DAY TIME SLEEPING AS I'M MORE THAN SURE THAT KATHY DOES NOT DO THIS , HAVE YOU NOTICED THIS ON THE DOWN LOADS ?
I'M SORRY IF I SOUND PARANOID BUT WITH EVERY PASSING DAY I FALL THAT MUCH FURTHER IN LOVE WITH THIS CHILD AND TRULY COULD NOT BEAR HER NOT BEING A PART OF MY LIFE.
WE WILL BE SEEING CHRIS SEATON IN MAY SOME TIME AND I AM PREPARING A LIST OF QUESTIONS TO BRING UP IN OUR DISCUSSION WITH HIM THEN.
ANY WAY I DIDN'T MEAN FOR THIS LETTER TO BE ANYTHING MORE THAN A HELLO AND A THANK YOU ONCE AGAIN FOR ALL YOUR HELP SO FAR.
I LOOK FORWARD TO TALKING TO YOU ON OUR NEXT DOWNLOAD
SINCERELY YOURS
CRAIG, KATHY AND LAURA.

The New Children's Hospital

Royal Alexandra Hospital for Children

Chris Seton
Consultant Sleep & Respiratory Paediatrician
Tel.: (02) 9845-3437 Fax: (02) 9845-3439
E-Mail: ChristS1@nch.edu.

CS/cr

1st February, 2000

Detective Senior Constable Bernard Ryan,
Criminal Investigation Unit,
Singleton Police Station,
22 Hunter Street,
SINGLETON. N.S.W. 2330

Dear Detective Ryan,

Following our discussion and meeting late last year about Laura Folbigg, I have now, as agreed, investigated the date of return of Laura's home apnoea monitor.

The monitor was returned to The New Children's Hospital on 20th January, 1999. The last record of breathing information, which was downloaded from the monitor via modem to The New Children's Hospital, was on 25th August, 1998.

On the basis of this information, I cannot definitely determine whether or not Laura was monitored in sleep during the period from 25th August, 1998 until 20th January, 1999.

I hope this information is helpful in your investigation.

With kind regards.

Yours sincerely,

Chris Seton

CHRIS SETON,
Staff Specialist, Sleep Disorders Unit

NSW INSTITUTE OF
FORENSIC MEDICINE

42-50 PARRAMATTA ROAD
PO BOX 90
GLEBE NSW 2037
PHONE (02) 9660 5977
FAX (02) 9552 1613

CORONERS ACT, 1980

AUTOPSY REPORT

Name: Laura Elizabeth FOLBIGG

Institute Case No: 99/9322

Age: 1 year 8 months

Sex: Female

Identification Process:

Identified by Mr De Wit - Undertaker - Beresfield Funerals Pty Ltd

Identified to D Carruthers

Identified as Laura Elizabeth FOLBIGG

Method used Visual

Identity confirmed by -

Pathologist: Allan David Cala

Pathologist's qualifications: MBBS Dip RACOG FRCPA

Time of autopsy: 9.00 pm

Date of autopsy: 1 March 1999

Place of autopsy: NSW Institute of Forensic Medicine, Glebe

Autopsy Assistant: D O'Neill

OPINION:

In my opinion, based on what I have observed myself, my experience and training, and the information supplied to me:

A. Time and date of death: **Sometime between approximately 11.00 am and 12.45 pm on 1.3.99**

B. Place of death: **Singleton Hospital**

C. Cause of death:

1. **DIRECT CAUSE:**
Disease or condition directly leading to death:

(A) **UNDETERMINED**

ANTECEDENT CAUSES:
Morbid conditions, if any, giving rise to the above cause, stating the underlying condition last:

(B)

(C)

2. **Other significant conditions contributing to the death but not relating to the disease or condition causing it:**

REPORT SUMMARY AND OPINION:

This 20 month old child, Laura FOLBIGG, was the fourth child born to Craig and Kathy FOLBIGG, of Singleton.

The first born child, Caleb, was born on 1.2.89. He apparently "had classic stridor, which was particularly noticed during feeding" (in a letter written by Dr Chris Seton, Paediatrician, to Dr Quentin King, General Practitioner, on 3.5.96). He was allegedly last seen alive by his mother at about 0100 20.2.89, and was found deceased again by his mother at 0253 later that morning on 20.2.89. A coronial post mortem examination at Newcastle by Dr R.Cummings failed to find a cause of death, so a diagnosis of Sudden Infant Death Syndrome (SIDS) was made. No further investigations were performed by police officers in relation to the death of this child.

The second child, Patrick, was born on 3.6.90. He was "a snorer from early infancy". He and remained apparently well until he allegedly had some sort of "life threatening event" at three months of age, "resulting in cortical blindness and subsequent seizures" (letter, Dr Seton). He also was found by his mother at the time of this event. The ambulance were called, he was conveyed to hospital, where he remained for a very prolonged period of time. He was discharged eventually, but was allegedly found deceased by his mother at 0930 on 13.2.91, aged about 8 months. A hospital post mortem was performed, and this confirmed hypoxic brain damage. The case was not notified to the Coroner, although the underlying cause of the child's initial "life threatening event" was never uncovered.

The third child, Sarah Folbigg, was born on 14.10.92. She had apparently been well, although "was a very loud snorer who suffered witnessed apnoea and choking episodes during sleep. Sadly none of this was investigated prior to her death" (letter, Dr Seton). She was allegedly last seen alive at 0030 on 30.8.93, and was found deceased by her mother at 0130 30.8.93, aged 11 months. The case was notified to the Coroner, and a post mortem examination was performed by Professor Hilton at the NSW Institute of Forensic Medicine. A cause of death was not found and a diagnosis of SIDS was subsequently made, although "Sarah had a very long palate and uvula. It is well known clinically that soft palates and uvulas become swollen and elongated as a secondary effect of habitual snoring" (letter, Dr Seton).

The fourth child, Laura Folbigg, was born full term at Singleton Hospital. Over the first 12 months of her life, Laura's sleeping habits were monitored by Medical Staff at Westmead Hospital. Laura was diagnosed with "central apnoea", with no evidence of obstructive apnoea. This condition was felt to be essentially of no medical significance. (per phone Dr Seton, Westmead Children's Hospital) Despite this, intensive electronic monitoring of Laura's sleep pattern was performed for quite a lengthy period of time, however at no stage were any significant sleep abnormalities ever uncovered. The father of all of the children has allegedly been diagnosed with Obstructive Sleep Apnoea.

Laura had last seen a doctor in early February for her 18 month vaccination. She had recently been unwell for approximately 1 week with cold and flu type symptoms and had been given Demazin on 27.2.99 as treatment for the symptoms. Demazin is a mixture of chlorpheniramine maleate, an anti-histamine, and phenylephrine hydrochloride, a decongestant, and can be given in syrup form to infants and children.

On the day of her death, Laura was apparently in a bad mood but did not appear to be ill. She was taken by her mother to a gym, then visited her father at work. She had allegedly fallen asleep in the car on the way home, and was then put into her own bed at home. She was heard to cough approximately 30 to 60 minutes after being placed down, and approximately 5 minutes after this the mother went into Laura's room to check on her. Laura was lying on her back on the bed. Her face was very pale, and she was noted to be not breathing. The mother then picked her up, carried her to the breakfast bar and commenced CPR whilst dialling "000" and requesting an ambulance attend. She was taken to Singleton Hospital however attempts to resuscitate her were unsuccessful.

Postmortem examination on the body of Laura Folbigg was performed later that evening on 1.3.99. There was lividity mainly on the left side of the face. There was also dorsal lividity present. There were no significant injuries externally apart from minor bruises to the lower limbs. There were no injuries to the face or in the oral cavity. There were no petechial haemorrhages on the face or on the eyelids, and re-examination the next day also failed to show petechial haemorrhages. The neck examination was normal. There were no injuries to the oral cavity. Internally, there were no significant abnormalities apart from focally haemorrhagic and collapsed lungs.

Histological examination of tissues showed an inflammatory infiltrate in the heart, consistent with myocarditis, of probable viral origin. This accords with the history of a cold/'flu-like illness for several days prior to the death of the child. There are a variety of causes for myocarditis, including some viruses, bacteria, fungi, some immune-related disorders, some drugs, and several other causative agents.

Toxicological examination of tissues and fluids showed no drugs, alcohol or poisons present. The absence of the medication (Demazin) which Laura is alleged to have been prescribed 2 days before her death suggests it had not been given for some time prior to Laura's death, possibly 12-24 hours or longer.

The death of Laura Folbigg cannot be regarded as "another SIDS"(Sudden Infant Death Syndrome). The family history of no living children following four live births is highly unusual. Laura had metabolic blood and urine tests as an infant, as part of screening for possible inherited metabolic diseases. These tests were all normal, and this would exclude a metabolic abnormality as a cause of sudden death in Laura. Obstructive Sleep Apnoea has also been excluded as a cause of death for Laura, as there was no evidence to substantiate this diagnosis despite intensive monitoring by doctors at New Children's Hospital, Westmead.

The diagnosis of SIDS should be made very sparingly after the age of 12 months. This diagnosis should only be made after a death scene investigation, post mortem examination, and various toxicological and microbiological cultures have failed to establish any other reasonable cause of death. Although there was an inflammatory infiltrate in the heart consistent with myocarditis, this may represent an incidental finding.

The possibility of multiple homicides in this family has not been excluded. If homicidal acts have been committed, it is most likely these acts have been in the form of deliberate smothering. Smothering, whether deliberately or accidentally inflicted, may leave no trace. There are no specific post mortem findings for smothering. It is usually performed by one person, in the absence of any witnesses. It is relatively easy for an adult to smother an infant or small child with a hand, pillow, soft toy or other similar object.

The bodies of all the Folbigg children have been cremated.

DOCUMENTATION AND OTHER MATERIAL AVAILABLE:

At the time of the autopsy, the following documentation and material relating to the case had been made available to me:

1. Form P79A - Report of death to the Coroner.

2. Post Mortem Report Sarah Folbigg 93/673.

The following reports relating to this case have previously been compiled:

1. Interim Autopsy Report - Date: 1 March 1999.

2. Provisional Autopsy Report - Date: 8 March 1999.

SPECIMENS RETAINED FOR FURTHER EXAMINATION:

Tissue for histology.
Brain retained for neuropathological examination.
Blood, liver, stomach, urine and bile sent for toxicological examination.
CSF sent for biochemistry.
CSF, oral, rectal, nasal, lung and spleen sent for bacterial cultures.
Vitreous sent for urea, creatinine, glucose and electrolytes.

THOSE PRESENT AT POSTMORTEM:

Professor J Hilton	-	Director of Institute of Forensic Medicine.
Detective Constable Clint Nicoll	-	Sydney Crime Scene Unit.

AUTOPSY FINDINGS

Clothing:

Clothing had been removed prior to post mortem examination and consisted of a multicoloured short pair of pants, a disposable nappy and a yellow singlet.

EXTERNAL EXAMINATION OF THE BODY:

The body was that of a deceased female infant whose appearances were consistent with the stated age of 1 year and 8 months.

Body weight	-11.52 kg.	(50th-75th percentile)
Body length	-80.5 cm.	(<25th percentile)
Head circumference	-47 cm.	(50th percentile)
Chest circumference	-49 cm.	
Abdominal circumference	-44.5 cm.	
Intercanthal distance	-2.7 cm.	
Foot length	-12 cm.	

The head hair was light brown/blonde, curly and measured approximately 10 cm.
The eyebrows were light brown.
The eyes were blue-green and natural.
The pupils were equal and measured approximately 3 mm.
There were no petechial haemorrhages on the upper or lower lids.

There was a small amount clear fluid present at the nostrils.
The lips were slightly dried and cyanosed.
The teeth were normal for age.
The frenulum was intact.
The anterior tongue appeared normal.
There were no injuries to the oral cavity.
The ears, chin and neck appeared normal.

The nipples and umbilicus were in their normal anatomical position.
The trunk was normal for age.
The external genitalia was normal.

The hymen was normal for age.
The perineal region was normal with a small amount of faecal material present around the anus.

The fingernails were slightly dirty.

Evidence of natural disease:

There was no evidence of ankle oedema, lymphadenopathy or jaundice present.

Post mortem changes:

Rigor mortis was fully developed in all major muscle groups.
The body had been briefly refrigerated.
Lividity was present dorsally but was also present on the left side of the face where lividity was rather pronounced on the left cheek and left forehead.
There was no evidence of decomposition.

Marks or scars:

Nil present.

Evidence of injury:[1]

1. An ovoid 5 x 3 mm brown bruise was present just medial to the left patella.

2. On the right anterior lower leg approximately 60 mm above the right ankle was an ovoid 12 x 10 mm brown bruise.

Evidence of medical intervention:

1. A defibrillator pad was present on the central chest.

2. Three ECG dots were present on the chest.

3. An intravenous cannula was present in the right antecubital fossa.

---

[1]**Conventions used in description of injuries:**

1. The body is described in the Standard Anatomical Position. Reference is to this position only.

2. Injuries are numbered for reference purposes only. This is arbitrary and does not correspond to any order in which they have been incurred.

3. All injuries are perimortem, unless otherwise specified.

INTERNAL EXAMINATION OF THE BODY:

Head & neck:

The scalp and skull were normal.
There were no skull fractures.
The meninges were normal and there was no extradural, subdural or subarachnoid haemorrhage.
The brain weighed 1154 g and on external examination appeared normal.
The brain was placed in formalin for further detailed examination.
The base of skull was examined and the middle ears opened.
There were no abnormalities present.
The eyes, ears, nose and mouth were normal.
The eyes were examined with an opthalmoscope prior to the internal examination.
No retinal haemorrhages or other abnormalities were present.
The neck was normal and there were no cervical spine, hyoid bone or thyroid cartilage fractures.

Cardio-vascular system:

The heart weighed 62 g.
The pericardium was normal.
The valves and atria of the heart were normal apart from an 8mm diameter area of haemorrhage on the posterior surface of the left atrium.
The free wall thickness of the right ventricle was 2 mm and that of the left ventricle was 7 mm.
The myocardium was normal on section.
The coronary arteries had a normal distribution and were free of disease.
The venous system, portal veins and hepatic portal system showed no abnormality.
The aorta and its branches were normal.
There was no evidence of congenital heart disease.

Respiratory system:

The pharynx, larynx, trachea and main bronchi were normal.
The epiglottis, vocal chords and trachea showed no abnormality.
The left lung weighed 122 g and the right lung weighed 114 g and both were focally haemorrhagic and collapsed on section.
There was no evidence of pneumonia or other pathology.
There was no free fluid in either pleural cavity.
The ribs and ribcage were intact.
The mediastinum was normal.
The thymus weighed 28 grams and was normal apart from petechial haemorrhages on the anterior aspect of the suprasternal thymus gland.
This part of the thymus measured approximately 15 x 10 x 10mm and projected superiorly beyond the suprasternal notch.

Gastro-intestinal system:

The mouth, tongue, oesophagus, stomach and duodenum were normal.
The stomach contained a small quantity of milky type fluid mixed with vegetable type material.
The gastric mucosa was normal.
The small and large intestines showed no abnormality.
There was normal intestinal material within the small intestine and semi-solid green/brown stool within the colon.
There was no free fluid in the abdominal cavity.

Hepato-biliary system:

The liver weighed 430 g and was normal on section.
No mass lesions were present.
The gallbladder and extrahepatic biliary system were normal.
The pancreas was normal.

Genito-urinary system:

Both kidneys weighed 36 g each.
The capsules of both kidneys stripped with ease to reveal normal renal parenchyma.
Both ureters were patent and of normal calibre, ending in a normal urinary bladder containing 10 ml of clear urine.
The bladder mucosa was normal.
The uterus, fallopian tubes and ovaries were normal for age.

Haemopoietic system:

The spleen weighed 46 g and was normal on section.
There was mild mesenteric lymphadenopathy.
The bone marrow, macroscopically, appeared normal.

Endocrine system:

The pituitary gland, thyroid gland and both adrenal glands were normal, both of which weighed 2 g each.

Musculoskeletal system:

Where examined as part of the internal examination, appeared normal.

Radiological examination:

The entire body was X-Rayed on 2.3.99.
There were no fractures detected.

Re-examination of the body:

The body was re-examined on 2.3.99, and shown to other medical staff at the Institute.
No additional significant findings were present.
The body was re-examined 3.3.99 and a facial dissection was performed.
Several photographs of the face were taken.
There were no bruises or any other injuries detected on facial dissection.

MICROSCOPIC EXAMINATION OF TISSUES:

Liver:	Normal.
Kidney:	Normal.
Spleen:	The splenic architecture is normal. Many germinal centres are present within white pulp areas and there is a markedly increased number of lymphocytes in red pulp. The appearances are of a probable viral infection. There is no evidence of malignancy. Nor are there any histological features to suggest any specific underlying viral infection.
Heart:	Within the myocardium is a moderately dense infiltrate of lymphocytes which have aggregated in certain areas particularly subendocardially and along the superficial surface of the myocardium, although further sections show large aggregates in the central area of the left ventricle. In these areas, there are large clusters of lymphocytes surrounding degenerate myocytes. Myocytolysis is present. No viral inclusions are seen. The appearances are of myocarditis, which is probably viral in aetiology.
Heart: **(2nd/ 3rd cuts)**	Further blocks taken confirm the presence of aggregates of lymphocytes in a similar distribution to those in the first histological examination of the heart.

Lungs:	There is an increased number of lymphocytes within the interstitium and in some alveolar spaces. There are widespread areas of haemorrhage with numerous red blood cells within alveolar spaces, which also oedema fluid, foamy macrophages and some fibrin. There is no evidence of pneumonia. The trachea is normal.
Stomach:	Autolysed, within normal limits.
Oesophagus:	Normal.
Adrenal:	Normal.
Salivary gland:	Normal.
Small and large intestine:	Normal, autolysed.
Thyroid:	Normal.
Bone marrow:	Reactive changes are present.
Pancreas:	Normal
Diaphragm/skeletal muscle:	Normal
Ovary:	Normal
Thymus:	Focal cortical haemorrhages are present.

NEUROPATHOLOGY REPORT:

See attached report.

ANALYTICAL TOXICOLOGY REPORT:

See attached report.

Quantitative procedure results:

Blood alcohol:	Not detected.	
Blood (preserved)	Chlorpheniramine:	Not detected.

The screening and quantitative tests reported by laboratory staff of the Division of Analytical Laboratories, NSW Health Department were selected by the laboratory staff with due regard to the information supplied and the Laboratory's objectives: to detect toxic levels of poisons. Furthermore, neither minor drug levels nor all specimens may have been fully examined.

A D Cala MBBS Dip RACOG FRCPA

Forensic Pathologist
NSW Institute of Forensic Medicine

13 December, 1999

NSW INSTITUTE OF
FORENSIC MEDICINE

42-50 PARRAMATTA ROAD
PO BOX 90
GLEBE NSW 2037
PHONE (02) 9660 5977
FAX (02) 9552 1613

NEUROPATHOLOGY REPORT:

Name: Laura Elizabeth FOLBIGG

Institute Case No: 99/9322

Macroscopic examination:

The brain was examined after fixation in formalin and weighs 1307 g (fresh weight 1154 g). Several irregular pieces of cranial dura are submitted for examination. These show no gross abnormalities. The leptomeninges over the convexities and at the base of the brain are thin and transparent. The vessels about the base of the brain, forming the circle of Willis are normally arranged and show no focal abnormalities. No aneurysms are identified. The external surface of the cerebrum is unremarkable and the gyri are normally arranged. The cerebrum is proportional to the size of the cerebellum. The cranial nerves, brainstem and cerebellum show no external abnormalities.

The brainstem is separated from the cerebrum through the rostral midbrain (brainstem and cerebellum 155 g; 11.8% brain weight) and the cerebral hemispheres are sectioned in the coronal plane at approximately 1.0 cm intervals. The cortical ribbon is of appropriate thickness without focal abnormality. The grey/white junction is well defined and the ratio of cortical grey matter to white matter volume is normal. The pattern of myelination is normal for age.

The ventricles are symmetric and of normal size. The striatum, thalamus, hypothalamus including the mamillary bodies, globus pallidus, amygdala and hippocampus show no gross abnormalities.

The cerebellum is separated from the brainstem and sectioned in the parasagittal plane at 0.5 cm intervals. The superior and inferior vermis and cerebellar hemispheres including the deep white matter and dentate nuclei show no gross abnormalities. The brainstem is sectioned in the transverse plane at 0.5 cm intervals. The substantia nigra and locus coeruleus are not pigmented which is normal for age. No gross abnormalities are identified.

Also submitted for examination is a 20 cm segment of spinal cord enclosed in dura. The dura is intact without focal abnormalities. The dura is opened longitudinally in the posterior midline. The leptomeninges are thin and transparent. The external surface of the cord and the ventral and dorsal roots are unremarkable. The cord is sectioned in the transverse plane at approximately 1.0 cm intervals. No focal abnormalities were identified.

Microscopic Examination:

Unless otherwise stated, sections from the left side of the brain are examined. Sections of posterior frontal and anterior parietal lobes including Brodmann areas 4&7, hippocampus, globus pallidus, medulla, cerebellar hemisphere and spinal cord, stained with haematoxylin and eosin/luxol fast blue or haematoxylin and eosin are examined and show no significant pathological abnormalities.

Diagnosis:

Brain showing no significant abnormalities (brain weight 1154 g).
Development is appropriate for age (20 months).

Dr M Rodriguez
Neuropathologist
13 December, 1999 (es)

MYOCARDITIS

NSW Police Service

EXPERT CERTIFICATE

Section 177, Evidence Act 1995 No. 25

In the matter of :	R-v- FOLBIGG
Place Statement Taken:	ADELAIDE
Date:	28 MARCH 2003

Name:	DR ALLAN DAVID CALA
Work Address:	FORENSIC SCIENCE CENTRE
	21 DIVETT PLACE ADELAIDE
	S.A. 5000 AUSTRALIA
Work Telephone	08-8226-7700
Occupation:	FORENSIC PATHOLOGIST

STATES:

1. This statement made by me accurately sets out the evidence which I would be prepared, if necessary, to give in court as a witness. The statement is true to the best of my knowledge and belief and I make it knowing that, if it is tendered in evidence, I shall be liable to prosecution if I have wilfully stated in it anything which I know to be false, or do not believe to be true.

2. I am 44 years of age.

3. I have a specialised knowledge based on the following training, study, and experience

Qualification
:MB, BS., Dip
RACOG, FRCPA

Other Study/Experience:

4. At about2pm on the 27th March 2003 at Goulburn Police Station , I examined a video.

5. Based wholly or substantially on the above knowledge, I am of the opinion that (SEE BELOW)

Witness. _____ Signature: A D Cala

ALLAN DAVID CALA

1. At approximately 2pm 27 March 2003, I viewed a video with Det. Sgt. Bernard Ryan at the Goulburn Police Station. This video was dated 28 February 1999, with the time given on the video as 3.07 to 3.11pm.

2. In this video, a small female child, identified to me by Bernard Ryan as Laura Folbigg, was seen playing around and in a backyard swimming pool. The child was wearing a swimming costume and had floatation devices on her arms and torso.

3. Laura Folbigg appeared to be in good health, and was well nourished. Her general development appeared normal for a child of her age. She responded appropriately when called, and exhibited no evidence of a cough, shortness of breath, wheeze or other form of obvious ailment or physical impairment.

4. Although not seen to be vigorously exercising, Laura Folbigg was nevertheless playing normally. At no time did I see her exhibit signs of being in any form of physical distress.

5. Had she been suffering from significant myocarditis at this time, irrespective of the cause, I would not have expected her to appear as she does. If the myocarditis was of any clinical significance at the time of the video recording, I would have expected the parents to notice Laura was unwell and they would have sought medical attention.

6. I would have expected Laura to have been disinterested in play, possibly to have been lethargic or pale, and to have exhibited poor exercise tolerance. I would have expected her to have been short of breath on even slight exertion. She may have been vomiting and been uninterested in eating, and had a fever, requiring the use of medication such as panadol.

7. That Laura Folbigg appeared in such good health less than 24 hours prior to her death makes me believe more firmly that the myocarditis which was found at autopsy played no role whatsoever in her death, and was an incidental finding.

Witness: _____ Signature: A D Cala

ALLAN DAVID CALA

2c

New South Wales Police

STATEMENT in the matter of:

Patrick FOLBIGG
DOB: 3/6/90

Date: 12 March, 1999

Name: Ian Arthur WILKINSON

Address: 115 Elder St, Lambton. Tele: 022/4952 6599

Occupation: Paediatric Neurologist

I hereby state:

1. This statement made by me accurately sets out the evidence which I would be prepared if necessary to give in court as a witness. The statement is true to the best of my knowledge and belief, and I make it knowing that, if it is tended in evidence, I shall be liable to prosecution if I have wilfully said anything which I know to be false or do not believe to be true.

2. I am 53 years of age.

3. I hereby certify that my full name is Ian Arthur Wilkinson, and my contact address is 115 Elder St, Lambton. 2299. I have specialised knowledge based upon the following training study and experience. I trained in Medicine at the University of Queensland, and subsequently trained in General Paediatrics in Sydney, before training in Neurology in Sydney and Milwaukee, U.S.A. I had a total of 4 years training in General Paediatrics and 4 years training in Neurology. I have been in practice as a Consultant Paediatric Neurologist in Newcastle for the last 18 years, and during that time I have seen something in the order of about 12,000 patients.

   I have examined, to the best of my knowledge, only one of the Folbigg children, namely Patrick. I first examined him on October 18, 1990, and lastly at the time when he was brought to the Mater Hospital in Newcastle, I think on February 13, 1991. I am enclosing a copy of my letter of November 28, 1991, which details the history of my involvement with him. Basically he presented to me with what were described as episodes of stopped breathing, subsequently had some seizures, and these were difficult to control, and I had treated him with both Tegretol and Phenobarb. He had suffered damage previously to the back part of his brain, for reasons that were never explained.

A lot of investigations were carried out to try and find the underlying cause for his problems, and I suspect that some investigations were also carried out on his siblings.

I think it is fair to say that no particular cause for the deaths of these children was ever established, despite quite a lot of work-up from various laboratories.

I have no explanation for why these children have died. It is certainly extraordinarily unlikely that this can all be blamed on "cot death", and indeed the latest child was well past the age one would expect this to happen.

Yours faithfully,

Ian Wilkinson.

MAUREEN PRICE
.....
Witness

M Price
.....
Signature

Detective B. Ryan
Singleton Police
SINGLETON NSW

29 June 1999

Dear Detective Ryan,

Re: FOLBIGG DEATHS

As you are aware, I performed a post mortem examination on the deceased body of Laura Elizabeth FOLBIGG, aged twenty months, who died at Singleton on 1 March 1999. She was the fourth child in the one family to die, the others having died over a period of 10 years from 1989 until this year. All have died at various ages, with none surviving either infancy or the toddler age. As a result of the investigations in relation to the death of Laura, I have obtained certain other information in relation to the deaths of the other children. Two of the four children have been diagnosed with "Sudden Infant Death Syndrome" (SIDS) and one other child has died months after a seizure/fit/apnoeic episode. The cause of death for Laura remains undetermined.

SIDS is diagnosed when a child (usually aged between 3 to 6 months, but usually not older than 12 months) dies and no cause of death is able to be given despite a thorough investigation of the circumstances surrounding the death of the child, including a visit to the death scene, full post mortem examination including detailed histopathological, neuropathological, microbiological, biochemical and toxicological testing of various tissues and fluids has been performed.

The first child, Caleb Folbigg, died when he was 20 days old in 1989 of "Sudden Infant Death Syndrome". He was allegedly found deceased or unresponsive but alive in his cot by his mother. This case was referred to the Coroner and a post mortem examination was performed in Newcastle. No significant disease processes were found to explain the death of this child. Toxicological analysis was unhelpful, and therefore, with consideration of all factors, a diagnosis of "Sudden Infant Death Syndrome" (SIDS) was submitted as the cause of death. No further investigation was done by police in relation to this death once the diagnosis of SIDS had been made, and this would be appropriate under the circumstances.

The second child, Patrick, died at nine months of age. He allegedly had some sort of apnoea attack (ceased breathing), or seizure at about 3 months age at home one night, having been found to be not breathing and unresponsive by his mother. He was taken to hospital, where he remained very unwell for some time. He was later diagnosed with blindness, as an area of the brain controlling vision had been affected by hypoxia (reduced blood oxygen) at the time of his initial presentation. Patrick was taken home months later, but may have suffered a further fit at home and subsequently died. Again, I believe, the mother of the child found him either deceased or unresponsive at home, following which a post mortem was performed at the John Hunter Hospital. This case was not referred to the Coroner. A death certificate was issued and the case was not investigated further.

The third child, Sarah, died at 10 months of age in 1993. She also was found unresponsive by her mother in the family home. She had allegedly been a loud snorer in life, and a possible diagnosis of Obstructive Sleep Apnoea was therefore tentatively made after her death based on her own history of snoring and a family history of snoring. A post mortem was performed by Professor John Hilton, Director of the NSW Institute of Forensic Medicine, and a diagnosis of Sudden Infant Death Syndrome was made. Again, this diagnosis was made after a negative post mortem with exclusion of natural diseases, drug intoxications, violence etc.

The fourth child, Laura, had been investigated very thoroughly by Dr Chris Seton at Westmead Hospital for many months after her birth in view of the possible family history of Obstructive Sleep Apnoea. She had been meticulously and carefully monitored during life, and no apnoeic episodes of any significance were ever uncovered. No definite diagnosis of any life-threatening condition was ever made during her life. She had been unwell for about a week prior to her death with some 'flu-like illness, and was found by her mother to be apnoeic and unresponsive in her bed after she fell asleep in the car on the way home from the gym.

This fourth child did not die of SIDS, as she was too old for this diagnosis, and had an intercurrent illness which **might** have explained her death. I do not believe that SIDS runs in families, but other conditions may do so. Many natural diseases may cause death during infancy, and these seem to have been excluded either during life or at post mortem. The deaths of **all** children in the one family in Australia in 1999 is not only extremely puzzling but clearly warrants thorough investigation.

In my opinion, the possibility of multiple homicides in this family has not been reasonably entertained, investigated and excluded. If acts of foul play have been committed, it is most likely that these acts have been in the form of deliberate smothering. Smothering, whether deliberately or accidentally inflicted, may leave no trace, there are no specific post mortem findings, and it is usually performed by one person, when alone with their victim. There are usually no other witnesses to these events. Very young children have small openings to their airways, (nose and mouth) and it is relatively easy for an adult or even a large child to deliberately smother an infant or small child using a hand, pillow or other similar object applied to the face without great force.

I believe that many causes of death such as overt physical violence (including shaken baby syndrome), poisoning, and many natural diseases can be reasonably excluded in causing the deaths of these children. There remains as an exceedingly remote possibility, and so remote as to be almost impossible, of some underlying and undiagnosed genetic or metabolic disease which has so far not been detected.

In my opinion, the deaths of the Folbigg children should be investigated as a single unit. I am greatly concerned by the deaths of these children, and I believe very vigorous efforts of enquiry should be made to exclude homicide in any or all of these children.

Please do not hesitate should you require any further assistance from me in this matter.

Yours,

Allan Cala, FRCPA
Staff Forensic Pathologist
NSW Institute of Forensic Medicine

2K

EXPERT CERTIFICATE in the matter of: Death of FOLBIGG children

Place: John Hunter Hospital Date: 6 December 1999

Name: David Michael COOPER
Address: John Hunter Hospital Tel.No: 49213000
Occupation: Paediatrician STATES:-

EXPERT CERTIFICATE
Section 177, Evidence Act 1995 No. 25

1. This statement made by me accurately sets out the evidence which I would be prepared, if necessary, to give in court as a witness. The statement is true to the best of my knowledge and belief and I make it knowing that, if it is tendered in evidence, I shall be liable to prosecution if I have wilfully stated anything which I know to be false or do not believe to be true.

2. I am 56 years of age.

3. I hereby certify:
My full name is **David Michael COOPER**
My contact address is **John Hunter Hospital,**
I have a specialised knowledge based on the following training, study and experience:-
I completed MBBS (Bachelor of Medicine, Bachelor of Surgery) at the University of Adelaide in 1965, Master of Science (Physiology) at the University of Toronto in 1974, MRACP (Membership of the Royal Australasian College of Physicians) in 1970 and FRACP (Fellowship of Royal Australasian College of Physicians)in 1973, Fellowship of the Royal Surgeons of Canada in 1974. I am currently Head Paediatrian at the Paediatric Respiratory and Sleep Service at the John Hunter Hospital. I have been performing this duty at the John Hunter combined with the Mater Hospital, Newcastle for the past eleven years. The Sleep Service is concerned with the clinical evaluation of infants and children suffering apparent sleep and breathing

See Continuation Sheet ...

Witness: _____ Signature: _____

disorders.

4. At 10.45am on Monday 6 December 1999, I spoke with Detective RYAN at the John Hunter Hospital, Newcastle. He showed me copies of a sleep study dated 15.6.90, and two EEG reports dated 18.10.90 and 5.11.90. I saw that these medical records related to a patient known as Patrick FOLBIGG born 3.6.90. I do not personally recall Patrick however from reading the above medical records I can supply the following information.

5. The sleep study was conducted on the 15th of June 1990 at the Mater Hospital in Newcastle where I was a Paediatrician at the time. I would have supervised this sleep study which was a pneumogram performed using Corometrics apparatus. This apparatus involved ECG leads being attached the chest and abdomen of the child to detect spells of apnoea and periodic breathing (PB). This device was not capable of distinguishing between central and obstructive apnoea. Central apnoea is one in which the brain does not give a signal to breath, there is no effort to breath by any of the respiratory muscles whereas an obstructive apnoea is one in which there is effort by the respiratory muscles, but due to upper airway obstruction no airflow takes place.

6. The study dated the 15.6.90 did not demonstrate any apnoeic episodes and the periodic breathing (PB%) was within normal limits for this unit. As a result, the study was recorded as normal. I believe at that time, Patrick was under the care of Doctor Richard HENRY and Barry SPRINGTHORPE's (Paediatricians).

EXHIBIT: I NOW SEEK TO PRODUCE SLEEP STUDY DATED 15.6.90

7. The EEG (Electroencephalogram) report dated 18.10.90 relates to a test performed by the Neurologist Dr J.T. Holland which appeared

normal.

EXHIBIT: I NOW SEEK TO PRODUCE EEG REPORT DATED 18.10.90

8. The EEG report dated 5.11.90 relates to a test performed by Dr Holland where he identifies asymmetry between the right and left sides of the brain both awake and asleep, and there was some seizure activity on the left side of the brain for a short period. There was excessive electrical slowing on the right side compared to the left.

9. Based wholly or substantially on the above knowledge, I am of the opinion that Patrick had no record of sleep apnoea at the age of 1.7 weeks and that later in his life he had a normal EEG and then a distinctly abnormal EEG sixteen days later.

10. Detective RYAN also showed me a sleep study report dated 5.11.92 and I saw that it related to a patient known as Sarah Kathleen FOLBIGG. I do not recall Sarah but I realise that she was Patrick's sister and this was reason for the study being completed. I supervised the study at the John Hunter Hospital which was the pheumogram with oximetry (oxygen saturation) performed on 5.11.92. The oximetry was normal; there were very few sleep apnoea recorded and some periodic breathing detected. Even so, the results were judged to be normal.

EXHIBIT: I NOW SEEK TO PRODUCE SLEEP STUDY REPORT DATES 5.11.92

11. Based wholly or substantially on the above knowledge, I am of the opinion that Sarah did not exhibit and signs of a respiratory control problem.

Witness: B. Ryan D/S/C 6.12.90 Signature:

NEWCASTLE MATER HOSPITAL - PHYSIOLOGICAL MONITORING REPORT

PATIENT DATA:	NAME	UR NUMBER	BIRTHDATE
	PATRICK FOLBIGG	360390	3/6/90
		STUDY DATE	AGE (WKS)
		15/6/90	1.7

RESULTS:	APNOEA>10	APNOEA>12	PB%
	0.00	0.00	6.04

MINUTES OF MONITORING:--- 540

SHANNON & KELLY'S DATA -

AGE (WKS)	APNOEA>10	APNOEA>12	PB%
0 - 4	1.9	0.3	3.5
4 - 8	1.2	0.2	2.5
8 - 20	1.4	0.2	1.5
20 - 52	1.6	0.6	1.0

COMMENT: NORMAL TRACE

The Royal Newcastle Hospital
Department of Clinical Neurophysiology

E.E.G. REPORT 29/10. c/D.

DATE: 18/10/90

NAME: FOLBIGG PATRICK ALAN DOB: 3/6/90 MRN: 521375

ADDRESS: 36 RAWSON ST MAYFIELD SEX: M No.: M90/1144

REFERRED BY: DR COOPER √MATER HOSP

HISTORY:

Apnoea, ? seizure.

REPORT:

The bulk of the recording is done in stage 2 sleep.

The 14 Hz sleep spindles are extremely well formed and distributed and although frequently asynchronous there is no overall asymmetry noted.

The background activities are normal and symmetrical with a very well formed frequency amplitude gradient and the appearance from time to time of cone wave posteriorly.

The arousal response at the end of the recording is normal and symmetrical.

CLINICAL INTERPRETATION:

This is a normal EEG for this age and the states of sleep.

J. T. HOLLAND

The Royal Newcastle Hospital
Department of Clinical Neurophysiology

E.E.G. REPORT

		DATE:	5/11/90	
NAME:	FOLBIGG PATRICK	DOB:	3/6/90	MRN: 521375
ADDRESS:	36 RAWSON ST, MAYFIELD	SEX:	M	No.: M90/1205-3
REFERRED BY:	DR. MORRIS	MATER		

HISTORY:

Recurrent fits, focal fits and oculogyric episodes.

REPORT:

The recording was done with the child asleep reaching stage II and subsequently waking at the end of the recording. In the early part of the recording, although there is a frequency amplitude gradient which can be identified, it is asymmetrical, with slow activities seen significantly slower on the right than on the left. In addition throughout the period of sleep, there are frequent multifocal spikes seen throughout both hemispheres. Some of these are polyspike.

In the recording stage II sleep is reached with asynchronous, but overall symmetrical and well formed 14 Hz sleep spindles.

Following arousal at the end of the recording, the record on the whole changes appropriately, but again the asymmetry is still apparent and just immediately prior to the end of the recording there is a brief repetitive seizure phenomenon seen in the left parietal region.

CLINICAL INTERPRETATION:

The record is abnormal in spite of the normal appearance of sleep spindles in stage II sleep. There is some attention drawn independently to both hemispheres. There is an excess slow on the right in comparison with the left. However, whilst there is independent potentially epileptogenic activity seen in a multifocal nature in both hemispheres, there is one further brief seizure event seen in the left parietal region.

On review there does appear to have been some deterioration in the record since the previous two. Review of the original one is again absolutely normal. The second one, I think, is borderline and this one frankly abnormal. The picture suggests an ongoing encephalopathic process.

J. T. HOLLAND

SLEEP STUDY RECORD SHEET

Date: 5/11/92.

59-54-56
FOLBIGG SARAH KATHLEEN
9 DOWER CLOSE THORNTON 2322
14-Oct-1992 F AMU.DR. R. HENRY
WD.B2 ADM.05/11/92 12

TIME STUDY COMMENCED	TIME STUDY COMPLETED
0925	1610.

REFERRING DOCTOR: HARDACRE.

DIAGNOSIS: SIBLING OF S.I.D.S.

MEDICATIONS: NIL.

PHONE NUMBER: 664489.

IF THE CHILD IS AWAKE RECORD BELOW

Time Awake	Back to Sleep	Machine On/Off
1025	1110hr.	Off
1300.	1325	Off.
1610		

EXPERT CERTIFICATE in the matter of: Death of FOLBIGG children

Place: Westmead Coroner's Court Date: 14.1.2000

Name: Bridget WILCKEN

Contact Address: The New Children's Hospital Tel.No: 02 98453654

Occupation: Clinical Geneticist (Metabolic Physician) STATES:-

EXPERT CERTIFICATE

Section 177, Evidence Act 1995 No. 25

1. This statement made by me accurately sets out the evidence which I would be prepared, if necessary, to give in court as a witness.

The statement is true to the best of my knowledge and belief and I make it knowing that, if it is tendered in evidence, I shall be liable to prosecution if I have wilfully stated anything which I know to be false or do not believe to be true.

2. I am 66 years of age.

3. I hereby certify:

My full name is **Bridget WILCKEN**

My contact address is **The New Children's Hospital, Westmead.**

I have a specialised knowledge based on the following training, study and experience:-

I completed Bachelor of Medicine at the University of Edinburgh in 1956. I am a fellow of the Royal Australasian College of Physicians and I am a certified Clinical Geneticist (Human Genetics Society of Australasia.) I have twenty nine years experience in the field of Biochemical Genetics and Metabolic Medicine. I am currently employed by the New Children's Hospital at Westmead as a Senior Staff Physician and I am the Director of the New South Wales Newborn Screening Programme and the New South Wales Biochemical Genetics Service. I am also the Chairman of the New South Wales Health Department's Genetics Services Advisory

See Continuation Sheet ...

Witness: B. Ryan D/S/C. 14.1.2000.

Signature:

Committee.

4. On the 13 February 1991 I was employed by the New Children's Hospital. Between the 13 February 1991 and 25 February 1991 an examination was made of a urine sample at the Oliver Latham Laboratory of a urine sample reference 36-03-90. The results of this examination showed a normal amino pattern and essentially normal organic acid profile. (Lactic acid was slightly elevated, a finding to be expected in a post mortem urine sample.)

EXHIBIT: I NOW SEEK TO PRODUCE N.S.W BIOCHEMICAL GENETICS SERVICE REPORT.

In December 1991, I received a letter from Doctor Alison COLLEY from the Regional Medical Genetics Unit at the Western Suburbs Hospital in Newcastle.

EXHIBIT: I NOW SEEK TO PRODUCE LETTER DATED 4 DECEMBER 1991.

On 10 December 1991 I replied to Doctor COLLEY giving my opinion on the information that I had received that there was no clear indication of a fatty acid oxidation disorder and some fairly strong evidence against it. This evidence could not positively exclude every form of fatty acid oxidation.

EXHIBIT: I NOW SEEK TO PRODUCE LETTER DATED 10TH DECEMBER 1991.

In December 1999, my staff and I at the NSW Biochemical Genetics and Newborn Screening Services conducted tests on the newborn screening blood samples for all four babies of Kathleen FOLBIGG, dates of birth 01.02.1989, 03.06.1990, 14.10.1992, and 07.08.1997 using tandem mass spectrometry. The results were entirely normal. (These tests were released after I had received permission from Mr and Mrs FOLBIGG's solicitor Mr Brian DOYLE.)

These normal results tend to exclude certain disorders of amino acid metabolism (phenylketonuria, the tyrosinaemias, maple syrup urine disease, homocystinuria due to cystathionine synthase deficiency) and a large number of organic acidurias and fatty acid oxidation defects, including methylmalonic acidaemia, propionic acidaemia, medium chain acyl CoA dehydrogenase deficiency, and several other disorders which are extremely rare. While these negative results of tests performed on cards stored for a varying number of years do not definitely exclude the disorders mentioned, there is no positive evidence indicating an inherited metabolic disorder affecting amino acid, organic acid or fatty acid oxidation pathways.

The tests performed by tandem mass spectrometry cannot be regarded as definitive because of the age of the samples and also because the sensitivity of this new type of test is not yet fully known.

We also conducted a DNA test for the common mutation seen in medium chain acyl CoA dehydrogenase deficiency, which is present in 98% of known cases of this disorder. This mutation was not present in any of the children's samples.

EXHIBIT: I NOW SEEK TO PRODUCE NSW NEWBORN SCREENING PROGRAMME REPORT RE CALEB, PATRICK, SARAH AND LAURA FOLBIGG.

Witness: _____ Signature: _____

Witness: _____ Signature: _____

Department of Health, N.S.W.
OLIVER LATHAM LABORATORY
N.S.W. BIOCHEMICAL GENETICS SERVICE

TELEPHONE ENQUIRIES:
CLINICAL GENETICIST 887 565(
SENIOR SCIENTIST 887 564
RESULTS 887 566(

3.0

REFERRED BY:MATER NEWCASTLE

FILE

Ward: 4G

36-03-90

FOLBIGG, PATRICK

Dr. WILKINSON

Male

DOB: 03-Jun-1990

TEST	REF RANGE	UNITS	KIDNEY 910427 12:30 13Feb 1991	URINE 910428 12:30 13Feb 1991
RINARY AMINO ACID PATTERN				
AMINO ACID PATTERN	... ...	.....		NAD
ETHYLMALONIC ACID SCREEN				
METHYLMALONIC ACID	... ...	.....		NEG
RINARY ORGANIC ACID PROFILE				
LACTIC ACID	... ...	.....		+
OMMENT				
LABORATORY COMMENT	... ...	.....		PMUC1
STORED FOR FURTHER ANALYSIS	... ...		STORE	.....

NAD = NO ABNORMALITY DETECTED
NEG = NEGATIVE
\+ = SLIGHT INCREASE
PMUC1 = Urine collected post-mortem.
STORE = Sample is being stored for further analyses at a future date.

SENIOR SCIENTIST:MS J HAMMOND

REPORT PRINTED 11:42 25-Feb-91

CLINICAL GENETICIST (BIOCHEMISTRY):DR B WILCKEN

LEASE RETAIN UNTIL SUPERSEDED BY A REPORT WITH 3 SAMPLES' RESULTS.

The Hunter Area Health Service

REGIONAL MEDICAL GENETICS UNIT
C/o. Newcastle Western Suburbs Hospital
Turton Road
Waratah 2298
Telephone: (049) 60 2205
(049) 60 1613
Fax: (049) 68 3904
P.O. Box 21,
Waratah 2298

Director
Dr. Michael Partington
M.B. B.S. PhD (Lond.) FRCPE FRCPC

Our Reference

AC:HB:1564

Your Reference:

4 December 1991

Dr Bridget Wilcken
Oliver Latham Laboratory
PO Box 53
NORTH RYDE NSW 2113

Dear Bridget

Re: Caleb FOLBIGG (DOB 1/2/89, DOD 20/2/89)
Patrick FOLBIGG (DOB 3/6/90, DOD 13/2/91)

Mr & Mrs Folbigg have had two pregnancies to date both resulting in the early loss of their sons. Mrs Folbigg is adopted and we do not have any past history. Mr Folbigg is 1 of 8 siblings and one of his brothers had a child die in the neonatal period cause unknown. There is no other relevant family history. I am writing to enquire whether you think specific testing for MCAD deficiency is warranted on blood spots from these boys. I will enclose all the results of metabolic results that I have, mainly on Patrick, and if you feel there is anything outstanding they should be tested for please do go ahead. The parents are considering a 3rd pregnancy and are naturally very anxious as we do not have a diagnosis for their two sons.

Caleb was born at term after a well pregnancy and weighed 7lb 7oz. He was bruised from forceps and remained overnight in a humidicrib. I understand he had phototherapy for jaundice. His parents describe him as a poor feeder with a lazy larynx. There is a note about stridor in his file. He was discharged home on day 5 gaining weight and no abnormalities were noted in the routine check by the paediatrician. I enclose his post mortem.

Patrick was born at term after a well pregnancy. He weighed 7lb loz. He did not need a humidicrib, had no problems feeding, and no stridor. He also didn't have jaundice. He went home on day 6 and a neonatal check by the paediatrician was also normal. His milestones were thought to be normal. He was about 3 months of age when his parents went into his bedroom at 3am and found him limp, pale and gasping. When he arrived at hospital he was said to be apnoeic and needed resuscitation. Subsequently he developed seizures and the question was raised as to whether he in fact

...../2

came in postictal or whether the seizures were secondary to cerebral ischaemia following this original episode. He continued to have seizures which were very difficult to control. A CAT scan showed hypodense areas in the occipital regions posteriorly. All investigations to find an infectious agent including herpes encephalitis were negative and blood samples were sent to Adelaide for lysosomal and long chain fatty acids which were normal as were his urinary MPS screen and serum carnitine. He was admitted a month later with a prolonged seizure and because of the possibility of encephalitis he was given lumbar puncture but he may well have coned and impaired the blood supply to his posterior cerebral circulation at that time as he then had cortical blindness. On his date of death he suffered a cardio-respiratory arrest at home and was unable to be resuscitated.

His brain was sent to the Royal Alexandra Hospital for Children for histopathological examination. I enclose a copy of the report which seemed only to show old necrosis presumably from ischaemia. Dr Ian Wilkinson has stored fibroblasts and approached Geoff Thompson, Murdoch Institute, regarding a disorder of lactate metabolism as there was an arterial lactate of 1.6 and in the laboratory the upper limit was 0.8. However Geoff didn't really feel happy to go ahead unless there was further support for a lactate disorder which was unable to be found.

It may be that these two boys died of unrelated causes. Naturally the parents are anxious to have every possibility explored as they are planning their 3rd pregnancy. I wondered whether the normal plasma carnitine levels ruled out the possibility of MCAD and whether you thought that there was any mileage in this as a diagnostic possibility. Thank you for your time and thoughts on these two young boys.

Yours sincerely,

ALISON COLLEY
STAFF SPECIALIST.

Encl:

New South Wales Government

Department of Health

OLIVER LATHAM LABORATORY

The Macquarie Hospital, North Ryde
Cox's Road, NORTH RYDE 2113
Phone: 887 5666
Fax: 805 1259

ADDRESS TO REPLY TO:
P.O. Box 53, North Ryde, N.S.W. 2113

Our reference:

Your reference:

Dr Alison Colley
Staff Specialist
Regional Medical Genetics Unit
C/- Newcastle Western Suburbs Hospital
WARATAH 2298

10th December, 1991

Dear Alison,

Re: Caleb and Patrick FOLBIGG

Thank you for your letter about these boys. In considering my answer I am slightly handicapped by not knowing the date of Patrick's "near miss" event. However I note with interest that a urine sample dated 20th October, 1990 was sent to us, and that that contained no suspicious dicarboxylic acids. I further notice that a plasma sample taken on 25th October and sent to Adelaide had a totally normal carnitine value including the ratio of acylated to free carnitine. Both of these would argue against a fatty acid oxidation disorder. Of course the normal urinary findings would rather depend upon the relationship between the date of the urine sample and the actual date of the episode, but if it was within a day or so I rather think it would argue against it. Also against such a diagnosis is the age of death of Caleb and the post-mortem findings which in neither child show any evidence of fatty diathesis. I am aware of a recent paper of James Leanard's but even so I feel that there are so many factors not suggesting MCAD that there is no real need to go ahead with any further analysis. If you disagree very strongly, then I suppose we could pull out the blood spots and send them for an analysis of the G mutation. A normal finding would be a further evidence against this possible diagnosis although, as you know, it would not utterly exclude it.

With kind regards,

Yours sincerely,

1569 → 18th Oct '90

Bridget Wilcken
Clinical Geneticist

The Hunter Area Health Service

REGIONAL MEDICAL GENETICS UNIT
C/o. Newcastle Western Suburbs Hospital
Turton Road,
Waratah 2298
Telephone: (049) 60 2206
(049) 60 1613
Fax: (049) 60 1968
P.O. Box 21,
Waratah 2298

Director
Dr. Michael Partington
M.B. B.S. PhD (Lond.) FRCPE FRCPC

Our Reference: AC:CG

Your Reference:

27 February 1992

Dr Bridget Wilkinson
Clinical Geneticist
Oliver Latham Laboratory
PO Box 53
NORTH RYDE NSW 2113

Dear Bridget

Re: Caleb and Patrick FOLBIGG

Thank you for your letter about these brothers. Firstly the urine sample that you had on Patrick dated 20 October 1990 was on the second day after his near-miss event which occurred on 18 October. Thank you for explanation of Patrick's investigations. I certainly agree that there are so many factors not suggesting MCAD that there really isn't enough suspicion to go ahead with any further analysis.

Mr and Mrs Folbigg have recently returned to the Clinic where I discussed with them a lack of a diagnosis for both children but that Patrick's near-miss and Caleb's SIDS may be related but as yet we do not have any evidence for this. They informed me that they are now expecting their third child, 8 weeks gestation at this Clinic. Naturally they are going to be very anxious during the pregnancy as well as after the birth of the baby and we have organised some ultrasound scans for screening and hopefully reassurance on the physical development of the baby. They will be seeing Dr Hardacre, paediatrician in Maitland for postpartum care of the infant which will undoubtedly involve the usual monitoring systems. They understand that as we do not have a diagnosis on either boy we do not have anything in the way of specific prenatal diagnosis for this baby and indeed a definite recurrence risk is elusive although 25% would be the upper mark I would think.

Kind regards, thank you again for your help with this mystery.

Yours sincerely,

ALISON COLLEY
STAFF SPECIALIST.
Copy to Dr C Marley
Dr M Holland
Dr I Wilkinson
Dr Hardacre

NSW Newborn Screening Programme
Locked Bag 2012
Wentworthville NSW 2145

Tel: (02) 9845 3255 Int + 61 2 9845 3255
(02) 9845 3659 Int + 61 2 9845 3659
Fax: (02) 9845 3800 Int + 61 2 9845 3800

Patient Results Report run on 13 Jan 2000

Dr B Wilcken
NSW Biochemical Genetics Service
The New Childrens Hospital
WESTMEAD NSW 2145

Patient Details

Patient ID : PA056649
Name : CALEB FOLBIGG
Birth Date : 01 Feb 1989 Hospital: Newcastle Western Suburbs Hosp
Mult. Code :
Patient Sex : M
Mother's name: KATHLEEN FOLBIGG

Sample Results

ACYL CARNITINES:C2,C3,C4,C5,C5OH,C6,C8,C10,:1,C14,:1,C16,CAR
Ref.Range: Intensity > 10000 units

Sample ID	Collected	Result	Comments
BJ041682	05 Feb 1989	58632	No further tests are indicated

AMINO ACID PROFILE: Ala, Cit, Gly, Leu, Met, Tyr
Ref.Range: Intensity > 10000 units

Sample ID	Collected	Result	Comments
BJ041682	05 Feb 1989	78052	No further tests are indicated

PHENYLKETONURIA: Phenylalanine
Ref.Range: <200 umol/L

Sample ID	Collected	Result	Comments
BJ041682	05 Feb 1989	11	No further tests are indicated

Medium Chain Acyl CoA Dehydrogenase
Ref.Range: Not detected [--]

Sample ID	Collected	Result	Comments
BJ041682	05 Feb 1989	--	The common A to G change was not detected at position 985 on either chromosome, thus a diagnosis of MCAD deficiency is unlikely, but still possible. See separate Biochemical

(*) = date of receipt, no collection date available

Clinical Geneticist (Biochemistry): Dr Bridget Wilcken
Principal Scientist: Dr Veronica Wiley

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Patient Results Report run on 13 Jan 2000
Patient ID: PA056649 CALEB FOLBIGG

Genetics reports for urine biochemical markers.
(See attached fact sheet for further details)

(*) = date of receipt, no collection date available

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Principal Scientist: Dr Veronica Wiley

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Patient Results Report run on 13 Jan 2000

Dr B Wilcken
NSW Biochemical Genetics Service
The New Childrens Hospital
WESTMEAD NSW 2145

Patient Details

Patient ID : PA089589
Name : PATRICK FOLBIGG
Birth Date : 03 Jun 1990 Hospital: Newcastle Western Suburbs Hosp
Mult. Code :
Patient Sex : M
Mother's name: KATHLEEN FOLBIGG

Sample Results

ACYL CARNITINES:C2,C3,C4,C5,C5OH,C6,C8,C10,:1,C14,:1,C16,CAR
Ref.Range: Intensity > 10000 units

Sample ID	Collected	Result	Comments
BJ168360	07 Jun 1990	65860	No further tests are indicated

AMINO ACID PROFILE: Ala, Cit, Gly, Leu, Met, Tyr
Ref.Range: Intensity > 10000 units

Sample ID	Collected	Result	Comments
BJ168360	07 Jun 1990	84256	No further tests are indicated

PHENYLKETONURIA: Phenylalanine
Ref.Range: <200 umol/L

Sample ID	Collected	Result	Comments
BJ168360	07 Jun 1990	14	No further tests are indicated

Medium Chain Acyl CoA Dehydrogenase
Ref.Range: Not detected [--]

Sample ID	Collected	Result	Comments
BJ168360	07 Jun 1990	--	The common A to G change was not detected at position 985 on either chromosome, thus a diagnosis of MCAD deficiency is unlikely, but still possible. See separate Biochemical

(*) = date of receipt, no collection date available

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Principal Scientist: Dr Veronica Wiley

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Patient Results Report run on 13 Jan 2000
Patient ID: PA089589 PATRICK FOLBIGG

Genetics reports for urine biochemical markers.
(See attached fact sheet for further details)

(*) = date of receipt, no collection date available

Clinical Geneticist (Biochemistry): Dr Bridget Wilcken
Principal Scientist: Dr Veronica Wiley

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Patient Results Report run on 13 Jan 2000

Dr B Wilcken
NSW Biochemical Genetics Service
The New Childrens Hospital
WESTMEAD NSW 2145

Patient Details

Patient ID : PA419632
Name : SARAH FOLBIGG
Birth Date : 14 Oct 1992 Hospital: John Hunter Hospital
Mult. Code :
Patient Sex : F
Mother's name: KATHLEEN FOLBIGG

Sample Results

ACYL CARNITINES:C2,C3,C4,C5,C5OH,C6,C8,C10,:1,C14,:1,C16,CAR
Ref.Range: Intensity > 10000 units

Sample ID	Collected	Result	Comments
BB138274	12 Jan 2000*	59152	No further tests are indicated

AMINO ACID PROFILE: Ala, Cit, Gly, Leu, Met, Tyr
Ref.Range: Intensity > 10000 units

Sample ID	Collected	Result	Comments
BB138274	12 Jan 2000*	76600	No further tests are indicated

PHENYLKETONURIA: Phenylalanine
Ref.Range: <200 umol/L

Sample ID	Collected	Result	Comments
BB138274	12 Jan 2000*	18	No further tests are indicated

Medium Chain Acyl CoA Dehydrogenase
Ref.Range: Not detected [--]

Sample ID	Collected	Result	Comments
BB138274	12 Jan 2000*	--	The common A to G change was not detected at position 985 on either chromosome, thus a diagnosis of MCAD deficiency is unlikely, but still possible. See separate Biochemical

(*) = date of receipt, no collection date available

Clinical Geneticist (Biochemistry): Dr Bridget Wilcken
Principal Scientist: Dr Veronica Wiley

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Patient Results Report run on 13 Jan 2000
Patient ID: PA419632 SARAH FOLBIGG

Genetics reports for urine biochemical markers.
(See attached fact sheet for further details)

(*) = date of receipt, no collection date available

Clinical Geneticist (Biochemistry): Dr Bridget Wilcken
Principal Scientist: Dr Veronica Wiley

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Patient Results Report run on 13 Jan 2000

Dr B Wilcken
NSW Biochemical Genetics Service
The New Childrens Hospital
WESTMEAD NSW 2145

Patient Details

Patient ID : PB556029
Name : FOLBIGG
Birth Date : 07 Aug 1997 Hospital: Singleton District Hospital
Mult. Code :
Patient Sex : F
Mother's name: KATHLEEN FOLBIGG

Sample Results

CONGENITAL ADRENAL HYPERPLASIA: 17Hydroxy Progesterone
Ref.Range: <50nmol/L :fullterm;BWt>2

Sample ID	Collected	Result	Comments
BB608364	11 Aug 1997	14	No further tests are indicated
BB611211	20 Aug 1997	12	No further tests are indicated

GALACTOSAEMIA: Galactose metabolites
Ref.Range: < 1.5 mmol/L

Sample ID	Collected	Result	Comments
BB608364	11 Aug 1997	< 1.5	No further tests are indicated
BB611211	20 Aug 1997	< 1.5	No further tests are indicated

CYSTIC FIBROSIS: Immunoreactive trypsin
Ref.Range: <100 ug/L whole blood

Sample ID	Collected	Result	Comments
BB608364	11 Aug 1997	45	No further tests are indicated
BB611211	20 Aug 1997	41	No further tests are indicated

ACYL CARNITINES:C2,C3,C4,C5,C5OH,C6,C8,C10,:1,C14,:1,C16,CAR
Ref.Range: Intensity > 10000 units

Sample ID	Collected	Result	Comments
BB608364	11 Aug 1997	55436	No further tests are indicated
BB611211	20 Aug 1997	61240	No further tests are indicated

(*) = date of receipt, no collection date available

Clinical Geneticist (Biochemistry): Dr Bridget Wilcken
Principal Scientist: Dr Veronica Wiley

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Fax: (02) 9845 3800 Int + 61 2 9845 3800

Patient Results Report run on 13 Jan 2000
Patient ID: PB556029 FOLBIGG

AMINO ACID PROFILE: Ala, Cit, Gly, Leu, Met, Tyr
Ref.Range: Intensity > 10000 units

Sample ID	Collected	Result	Comments
BB608364	11 Aug 1997	69436	No further tests are indicated
BB611211	20 Aug 1997	73656	No further tests are indicated

PHENYLKETONURIA: Phenylalanine
Ref.Range: <200 umol/L

Sample ID	Collected	Result	Comments
BB608364	11 Aug 1997	< 200	No further tests are indicated
BB611211	20 Aug 1997	< 200	No further tests are indicated

HYPOTHYROIDISM: Thyroid Stimulating Hormone
Ref.Range: <20 mIU/L whole blood

Sample ID	Collected	Result	Comments
BB608364	11 Aug 1997	1	No further tests are indicated
BB611211	20 Aug 1997	1	No further tests are indicated

Medium Chain Acyl CoA Dehydrogenase
Ref.Range: Not detected [--]

Sample ID	Collected	Result	Comments
BB608364	11 Aug 1997	--	The common A to G change was not detected at position 985 on either chromosome, thus a diagnosis of MCAD deficiency is unlikely, but still possible. See separate Biochemical Genetics reports for urine biochemical markers. (See attached fact sheet for further details)
BB611211	20 Aug 1997	--	The common A to G change was not detected at position 985 on either chromosome, thus a diagnosis of MCAD deficiency is unlikely, but still possible. See separate Biochemical Genetics reports for urine biochemical markers. (See attached fact sheet for further details)

(*) = date of receipt, no collection date available

Clinical Geneticist (Biochemistry): Dr Bridget Wilcken
Principal Scientist: Dr Veronica Wiley

Detective Snr Cnst.B.Ryan,
Singleton Detectives
Singleton Police Station
SINGLETON NSW 2330

19 June 2001

Dear Detective Ryan,

Re: R v Kathleen FOLBIGG

I acknowledge receipt of a 3 page fax from you dated 6 June 2001 in relation to this matter. I set out my responses below to some of the questions raised which have relevance to the post mortem examination I performed on Laura Folbigg.

Question (i)

> *"An examination of the literature indicates that staining from iron can indicate the presence of haemosiderin-containing macrophages (siderophages) in alveoli. In respect of any of the deceased was such a staining conducted?"*

In the case of Laura Folbigg, staining for haemosiderin-laden macrophages was performed using a Perl's stain, with a negative result. (No iron was present in any of the 10 sections of lung tissue examined)

The presence of haemosiderin-laden macrophages (siderophages) has been regarded by some pathologists to be a marker or indicator for previous attempts at mechanical airway obstruction, or deliberate smothering (1). This is a view which has not been widely accepted by the forensic pathology community. There are many causes for the presence of these macrophages, however their absence does not negate or exclude a diagnosis of deliberate smothering.

In my opinion, it is dangerous to conclude that a child has been deliberately smothered by the presence of iron in the lungs. The presence of iron in this setting is highly non-specific, and I would give the presence or absence of haemosiderin-laden macrophages no weight in assessing whether a child might or might not have died as a result of deliberate smothering.

Question (v)

> *"In relation to Laura there was found an inflammatory infiltrate in the heart consistent with myocarditis. Dr Cala indicates that this may be an incidental finding. Does he make that statement in the light of his knowledge of the family history? Could he be asked to indicate, what the result of his post mortem examination would have been, if the death of Laura was being looked at in isolation."*

The inflammatory infiltrate in the sections of heart which I examined in the case of Laura Folbigg was light in amount and patchy in distribution. There is evidence in the medical literature that this amount of inflammation could be considered of no relevance in the deaths of some children who have died as a result of, for example, choking on a foreign body or who died from motor vehicle trauma. My opinion that the inflammatory infiltrate in the heart represents an incidental finding is not based on the family history, but rather after consideration of the history provided of Laura's very sudden and most unexpected death, the post mortem findings of Laura and the histological assessment of the heart together with my own knowledge and experience of the condition of myocarditis.

In other cases I have seen where the death of a child or adult has been due to myocarditis, the inflammatory infiltrate has been much heavier in number and more diffuse in distribution throughout the heart, although the amount of inflammation is variable from case to case. There are often observable naked eye changes when examining the heart. These changes may consist of dilatation, flabbiness and pallor of the heart, and a "striped" appearance of the heart on cut section. There may be features at post mortem examination suggestive of heart failure. This may take the form of pleural effusions (straw coloured fluid in each pleural cavity) and ascites (fluid in the abdominal cavity). I should point out that these findings are not seen in every case and there are other causes for these findings. These changes were not present with Laura Folbigg, whose heart looked normal on naked eye inspection.

If I had examined the body of Laura Folbigg in isolation, without the knowledge I had at the time of previous infant deaths in the family, I might give the cause of death as Myocarditis. The question which has been asked of me is theoretical in nature and does not represent reality for this family ie there were other deaths in the family. When giving an opinion in relation to a

cause of death for Laura Folbigg, I cannot ignore any known relevant family history of severe illnesses or premature deaths, either infantile or adult. This is not to say however that such information in any way need bias or prejudice my opinion, but it is information nevertheless which may be of relevance in assessing any possible cause of death of Laura Folbigg.

Question (xi)

> *"It is noted that Dr Cala does not make a finding as to the cause of death in relation to Laura. Whilst this follows the relevant protocol, could he be asked:whether he does not subscribe to a finding of non-accidental suffocation because there are other causes of death which (on the evidence) are reasonably possible, as distinct from merely possible?"*

Question (xii)

> *"If so what are the possible causes of death in relation to Laura?"*

Non-accidental asphyxia in the form of deliberate smothering must be considered as a possible cause of death for Laura Folbigg, and as a possible cause of death for the other Folbigg children as well. I remain very suspicious that all four Folbigg children may have died as a result of deliberate smothering. The medical evidence however does not allow me to take this any higher than a **suspicion** of deliberate smothering. I set out some of my reasons for this suspicion below:

The circumstances of their deaths are remarkably similar. All four children were found deceased or cyanosed and apnoeic by the one person over a 10 year period. It seems no other witnesses were present when these children were found by the one person. All children were apparently quite warm when found, indicating their deaths occurred quite close to the time when they were found. There was an episode of cyanosis or apnoea with a previous child (Patrick Allan Folbigg) which in my view has not been satisfactorily explained, nor was his subsequent sudden death satisfactorily explained. No investigation of his death was undertaken by police as his death was not referred to the Coroner at the time.

Subsequently, the sudden death of Sarah Folbigg in 1993, following the deaths of two other children, should have raised immediate concerns to investigators that the possibility of foul play

might explain all three deaths. It seems no investigation of this sort was performed, and her death was ascribed to "Sudden Infant Death Syndrome". Sudden Infant Death Syndrome remains a diagnosis by exclusion of other causes. It replaces the cause of death as "Undetermined" with one that is acceptable to many doctors, parents, police and coroners alike without ever accurately knowing why or how the child died. It also generally avoids an inquest into the death. I would not have given the cause of death for Sarah Folbigg as "Sudden Infant Death Syndrome", but as "Undetermined" and include comments in relation to her and the other Folbigg children's deaths.

The diagnosis of Sudden Infant Death Syndrome is impossible to account for the deaths of all the Folbigg children. Caleb Folbigg was three weeks old when he died, and Laura Folbigg was almost two years when she died. The ages alone of these two children are sufficient to exclude Sudden Infant Death Syndrome as a reasonable diagnosis, especially for Laura Folbigg. Any diagnosable metabolic cause of death for these children has been more than reasonably excluded based on exhaustive biochemical tests and opinions from doctors expert in genetic and metabolic abnormalities.

Finally, I would like to add a position statement from the American Academy of Pediatrics 1994 (2), relevant in this case and with which I fully concur. It has great significance in this case, and partly reads thus:

> *"There is a small subset of infants who die unexpectedly, whose deaths are attributed to SIDS, but who may have been smothered or poisoned. Autopsy cannot distinguish death by SIDS from death by suffocation. A study of infants suffocated by their parents indicates that certain features should raise the possibility of suffocation. These include previous episodes of apnoea* (cessation of breathing) *in the presence of the same person, previous unexplained medical disorders such as seizures, age at death older than 6 months, and previous unexpected or unexplained deaths of one or more siblings or the previous death of infants under the care of the same, unrelated person."*

References:

1. Beecroft DMO, Lockett BK. Intraalveolar pulmonary siderophages in sudden infant death:a marker for previously imposed suffocation. *Pathology* 1997;29:60-3.

2. Distinguishing Sudden Infant Death Syndrome from Child Abuse Fatalities (RE9421), *Pediatrics*, Vol.94, Number 1 July 1994, p124-6 (Policy statement)

Yours,

Allan Cala

Allan D.Cala
FRCPA
Forensic Pathologist

EXPERT CERTIFICATE in the matter of: Death of FOLBIGG children
Police -v-
Place: 103 Esplanade, Hove Date: 8 December 1999

Name: Susan Mitchell BEAL
Address: 103 Esplanade, Hove. S.A. Tel.No: 08 83773455
Occupation: Paediatrician STATES:-

EXPERT CERTIFICATE
Section 177, Evidence Act 1995 No. 25

1. This statement made by me accurately sets out the evidence which I would be prepared, if necessary, to give in court as a witness. The statement is true to the best of my knowledge and belief and I make it knowing that, if it is tendered in evidence, I shall be liable to prosecution if I have wilfully stated anything which I know to be false or do not believe to be true.

2. I am 64 years of age.

3. I hereby certify:
My full name is **Susan Mitchell BEAL**
My contact address is **103 Esplanade, Hove. S.A.**
I have a specialised knowledge based on the following training, study and experience:- I graduated MBBS at Sydney University in 1958 and MD at Flinders University in 1986. I am currently employed as a Paediatrician at the Women's and Children's Hospital in Adelaide where I have been for the last thirty five years. I have been studying Sudden Infant Death Syndrome (SIDS) for over thirty years. In that time, I have published widely on SIDS, with more than fifty papers and book chapters. In 1986 I was awarded an MD for my thesis on SIDS.

I have interviewed the families of over five hundred infants who have died suddenly and unexpectedly, usually in the home on the day the baby died.

See Continuation Sheet ...

Witness: _____ Signature: _____

On the subject of recurrence of infant death in a family, I have published refereed papers (eg. Archives of Disease in Childhood). I have been invited to write book chapters on the subject, and been invited speaker on the subject in both Europe and America.

4. On Wednesday the 7th of December 1999, I had a conference with Detective Senior Constable RYAN from the New South Wales Police Service. Detective RYAN had previously forwarded a precis to me relating to the death of Caleb, Patrick, Sarah and Laura FOLBIGG.

EXHIBIT: SEE ATTACHED PRECIS MARKED ANNEXURE A

Detective RYAN showed me a quantity of medical, police and forensic records relating to Kathleen FOLBIGG, Craig FOLBIGG, Caleb FOLBIGG, Patrick FOLBIGG, Sarah FOLBIGG and Laura FOLBIGG. These records were indexed and contained within six large blue folders.

EXHIBIT: SEE LIST OF CONTENTS MARKED ANNEXURE B.

I carefully examined the files relating to the four children in the presence of Detective RYAN that day, and the files relating to Mr and Mrs FOLBIGG during the night by myself. Prior to making an assessment of those files, I would like to state my understanding about SIDS and Filicide gained from twenty five years of experience, personal research and study of literature.

When an infant dies suddenly and unexpectedly, occasionally a disease process is found. For the remainder it can be difficult to decide if the death is due to accidental suffocation, non-accidental suffocation or SIDS. The macroscopic and microscopic examination is rarely helpful but on occasion bruising or fractures or facial petechiae may point away from SIDS.

Witness: _____ Signature: _____

For a first sudden unexpectedly death in a family the infant may be found prone and the diagnosis then is most likely to be SIDS. It may be found with the face covered, and then the most likely diagnosis is accidental death. In my own experience for infants found on their back or side with the head uncovered, there is a suspicion of filicide in 20% of the cases (compared with only 2% of prone infants where filicide was suspected).

Clues to suspecting filicide if there has only been one death are:

* Abuse in other children or infants in the family.
* Apparent life threatening events (ALTE) in the index or other children, especially if commencing always in the presence of the same person.
* Munchausen syndrome in the perpetrator (usually the mother) eg. suspicion of this problem is aroused when there have been several hospital admissions during pregnancy for disorders not really related to the pregnancy, and more visits to doctors then would be expected for the health and fitness of the person.
* A reluctance to be visited by SIDS Association counsellors or occasionally obsessive involvement with such associations.
* Suspicion expressed by other family members or friends. Sometimes this presents as an unwillingness for family members to become involved or to speak about the death.
* Conflicting statements about the circumstances surrounding the death.
* A history of childhood deprivation abuse or disruption in the perpetrator.

There are a few disorders which may present as recurrent infant death. These can be excluded by appropriate investigations eg. metabolic disorders or cardiac arythmias.

Witness:_____ Signature:_____

There are two more common causes of recurrent sudden unexpected infant death. The first of these has been largely eliminated ie. leaving young infants unobserved in prone. The second is filicide. This is not only recurrent in occurring in the next child, but is likely to continue into a third or even fourth or more children.

I would agree with the pathologist who said the first unexplained death in a family may be called SIDS, the second should be labelled undetermined, and the third is murder until proven otherwise. Not how system works.

5. Based on the records I have examined in regards to the family Folbigg, I have no hesitation in saying I believe that all four children were murdered by their mother. Apart from the fact that the full story fits my previous comments made and prepared for publication by me prior to being aware of this family there are other factors which point directly towards murder by suffocation. These are:

* the wide age range of the children at the time of their initial observed events or deaths - nineteen days (Caleb) to twenty months (Laura).
* the finding of two infants (Patrick on 18.10.90 and Laura on 1.3.99) moribund rather than dead. This is extremely rare in SIDS.
* small unusual observations eg. I wonder how often the mother needed to get up at night to go to the toilet within four hours of going to bed (which is what is recorded in police report relating to Sarah).
* the reluctance of the mother to use the cardio-respiratory monitor as mentioned by the father in a letter to Margaret TANNER.

6. In support of filicide as being the cause of death of these children are the results of the study of Wolkind S, published in Acta

Paed Scand in 1993 where of forty three families with a second child dying suddenly and unexpectedly, thirty one (72%) were thought to be due to filicide. If those deaths that were partly explained were excluded thirty one out of thirty six (86%) were thought to be due to filicide. As far as I am aware there has never been three or more deaths from SIDS in the one family anywhere in the world, although some families, later proven to have murdered their infants had infants who were originally classified as SIDS.

Witness: _____ Signature: _____

In the matter of:
Police -v-
Name of expert: **Date: October 6, 2000**

Name: Janice Ophoven, M.D.

Address: 6494 Crackleberry Trail, Woodbury, MN 55129 U.S.A.

Occupation: Pediatric Forensic Pathologist **Telephone No.: 651-458-0541**

STATES: -

EXPERT CERTIFICATE
Section 177, Evidence Act 1995 No. 25

1. This Statement made by me accurately sets out the evidence which I would be prepared, if necessary, to give in court as a witness. The Statement is true to the best of my knowledge and belief and I make it knowing that, if it is tendered in evidence, I shall be liable to prosecution if I have wilfully stated anything which I know to be false or do not believe to be true.

2. I am 53 years of age.

3. I hereby certify:

My full name is: Janice Jean Ophoven, M.D.

My contact address is:

6494 Crackleberry Trail
Woodbury, Minnesota 55125
USA

I have a specialised knowledge based on the following training, study and experience:

I received my medical degree from the University of Minnesota in 1971

I completed residency training in Paediatrics at the University of Minnesota

I completed residency training in Anatomic Pathology at the University of Minnesota

I received specialty training in Paediatric Pathology at the University of Minnesota and Minneapolis Children's Hospital

I completed a fellowship in Forensic Pathology at the Hennepin County Medical Examiner's Office in 1980

I was the Associate Director and Director of Laboratories at the St. Paul Children's Hospital 1980 – 1988

Witness:_____ **Signature:**_____

I have maintained a practice in Paediatric Forensic Pathology for 20 years and have participated in the investigation of deaths and injuries in childhood in Canada and across the U.S.

I have provided courtroom testimony in deaths and injuries to children on numerous occasions.

I have been published in textbooks and peer-reviewed journals and have given educational seminars and workshops on issues pertaining to pediatric forensic pathology

4. From June 15 – August 13, 2000 I examined:

- Medical records of Kathleen Folbigg
  - Health Insurance Commision Records
  - Expert Certificate by Dr. Innis
  - Medical records supplied by Dr. Marley
  - Medical records supplied by Dr. Cash
- Medical records of Caleb Folbigg
  - Statement from Dr. Bridget Wilcken
  - Newborn Screening Blood results
  - Newcastle Western Suburbs Hospital records
  - Coroners Brief
  - Ambulance Records
- Medical records of Patrick Folbigg
  - Statement of Dr. Bridget Wilcken
  - Newborn screening blood results
  - Medical records from Newcastle Western Suburbs Hosp
  - Statement from Dr. Wilkinson
  - Medical Certificatee of cause of death
  - Cause of death certificate (hand written)
  - History, examination and progress notes
  - Report by Dr. Wilkinson to Marley
  - Report by Dr. Wilkinson to Dr. Morris
  - Adelaide Children's Hospital Pathology Report
  - Mater Hospital Pathology reports
  - Report by Dr. Challinor to Dr. Wilkinson
  - Biochemistry reports
  - Report by Dr. Wilkinson to Dr. Thomas
  - Physiotherapy report
  - Autopsy report
  - Report by Dr. Wilkinson to Dr. Bale
  - HAPS reports
  - Histopatholgy Dept Report
  - Report by Dr. Wilkinson to Folbiggs
  - Report by Dr. Colley to Dr. Wilkinson
  - Report by Dr. Marley to Dr. Holland
  - Dr. Colley to Dr. Wilcken
  - Dr. Wilckinson Dr. Colley
  - Dr. Edwards to Dr. Hardacre
  - Newcastle Mater Hospital Records June 14, 1990
  - Newcastle Mater Hospital Records October 18, 1990
  - Newcastle Mater Hospital Records November 4, 1990

- Newcastle Mater Hospital Records November 14, 1990
- Newcastle Mater Hospital Records December 22, 1990
- Statement by Dr. Marley
- Pediatric Summary
- Ambulance Records
- Beresfield Crematorium record

- Medical records of Sarah Folbigg
  - Statement by Dr. Wilcken
  - Newborn Screening Blood Results
  - John Hunter Hosptial Records
  - Statement by Dr. Marley
  - Pediatric discharge
  - Perinatal database
  - Reports: Dr. Hardacre to Dr. Marly
  - Buckner to Holland
  - Hardacre to Marley
  - Hardacre to Holland
  - Pickford to Marley
  - Edwards to Hardacre
  - Handwritten notes
  - Ambulance Records
  - Coroners Brief
- Medical records of Laura Folbigg
  - Statement of Dr. Wilcken (1.14.00)
  - Newborn Screening Blood Results
  - Statement of Christopher Seton
  - Handwritten sleep notes by Kathleen Folbigg
  - Report by Dr. Seton to Det. Ryan
  - Referral by Dr. Seon to Dr. King
  - Letter by Mr. Folbigg to Dr. Seton
  - Report by Dr. Seton to Mr. Folbigg
  - Newborn discharge summary
  - Report by Dr. Seton to Dr. King
  - Corometrics monitor supply record
  - Urine medabolic profile
  - Sleep study report (10.7.97)
  - Royal Alexandria Hospital for Children Medical History
  - Sleep study report by Seton to Sanders
  - Letter by Craig Folbigg to Margaret Tanner
  - Report by Seton to Craig Folbigg
  - Report by Seton to Dr. Sanders
  - Patient alarm traces (Corometric monitor print outs)
  - Statement of Dr. Innis
  - Information sheet
  - Progress Notes
  - Singleton Hospital Records
  - Ambulance report
  - Fairholme Surgery Records
  - Statement of Dr. Cash
  - Newborn discharge summary
  - Report by Dr. Seton to Dr. King
  - Sleep study reports 10.7.97 and 2.3.98
  - Report by Dr. Seton to Craigh Folbigg
  - Report by Dr. Seton to Dr. Sanders
  - Ambulance records
- Transcript of interview with Kathleen Folbigg
- Psychological report by Roz Garbutt

Witness:_____ **Signature:**_____

- Diary entry (19.2.89)
- Diary entries (June 4, 1996 – June 5, 1997)
- May 1992 Gibbs personal diary entry
- Diary entry July 1999
- Diary Entry June 6, 1997 – April 10, 1998
- Diary Entries January 1 – 3 1999
- Diary entry June 19, 1999
- Telephone Interception transcripts
- Listening Device Transcripts
- 26 autopsy photos of Laura Folbigg
- 53 Microscopic Slides-Laura
- 40 Microscopic Slides-Sarah
- 21 Microscopic Slides-Patrick
- 14 Microscopic Slides – Caleb
- Meeting with Constable B.Ryan on June 19, 2000

5. The following is a list of my findings

Caleb Gibson Folbigg

- Caleb was the product of a fullterm pregnancy and his 21-year-old married mother, Kathleen Folbigg, received adequate prenatal care. A "fainting" episode and a bout with the chicken pox complicated the pregnancy.
- Caleb was delivered vaginally with forceps assistance following an essentially uncomplicated labor on February 1, 1989. The baby's birth weight was 3230 grams and apgars were 9 at 1 and 9 at 5 minutes. His newborn course was complicated by a brief bout with transient tachypnea [mild respiratory distress] that resolved without difficulty. He was discharged home with his mother.
- His pediatrician, B.J. Springthorpe, at well child evaluation noted inspiratory stridor when the child was placed supine or agitated. The problem was characterised as mild laryngomalacia and no further followup was recommended.
- In the early morning of February 20, 1989, Kathleen fed Caleb [approximately 0100 hours]. Kathy checked on the baby again at 0250 hours and found him "cold' with bloody froth in his nose and mouth. Emergency medical services were called and they found the child in full cardiopulmonary arrest [essentially DOA], his skin warm to the touch, pale and cyanotic. The child was pronounced dead around 0300 hours on February 20, 1989.
- Autopsy examination was performed by Dr. R. Cummings at 1145 hours on February 20, 1989 in the City Morgue, Newcastle, New South Wales. His findings included:
  - A well developed, well nourished male infant, weight 3970 g.

- The stomach contained curdled milk.
- The lungs appeared congested and there were extravasated red blood cells in the tissue.
- There was no mention of petechial hemorrhages, specifically in the thymus.
- Routine toxicological analysis was negative.
- Cause of death: SIDS [Sudden Infant Death Syndrome]

- Bridget Widcken performed complete biochemical profile on blood samples from Caleb. The results were entirely normal.

Patrick Folbigg

- Patrick was the product of a fullterm pregnancy and his mother, Kathleen Folbigg, received adequate prenatal care. The pregnancy was uncomplicated.
- Patrick was delivered vaginally following an essentially uncomplicated labor on June 3, 1990. The baby's birth weight was 3410 grams and apgar was 8 at 5 minutes. His newborn course was uncomplicated and he was discharged home with his mother.
- His pediatricians were Richard Henry and Barry Springthorpe. He was scheduled for a sleep study for one week after his discharge. The examinations showed no GE reflux and the sleep study was normal.
- In the early morning of October 18 1990, Patrick's mother reports that she heard him coughing at approximately 0300 hours. At 0430 she was up and heard him "gasping" in his room and found him cyanotic, lifeless and making minimal respiratory effort. Emergency responders arrived around 0500 hours and provided oxygen and respiratory support. He improved spontaneously and was admitted to the hospital through the emergency department. During hospitalisation the child developed right-sided seizures that proved over time to be difficult to control and required multiple subsequent hospitalisations. Normal EEG is present in the record from October 18, 1990.
- CT scans revealed bilateral abnormalities of the brain specifically in the occipital lobes of the brain. The child also presented with severe visual deficits. The attending physicians evaluated a multitude of possible etiologies including herpes encephalitis, but eventually concluded that he suffered from encephalopathic

disorder, cause unknown. The findings were consistent with a severe hypoxic event. Despite these physical setbacks the baby continued to show satisfactory growth and development.

- On the morning of February 13, 1991, Patrick's mother put him down for a nap at ~ 0730 hours and she found him lifeless at 0930-1000 hours. Emergency responders arrived at ~1020. Emergency medical services found the child in full cardiopulmonary arrest [essentially DOA], his skin warm to the touch, pale and cyanotic. The child was pronounced dead at 1040 hours. The baby's father was apparently not present in the household at the time of his death.
- Dr. J. Bishop and Dr. G. Singh-Khaira performed autopsy examination at 1230 hours on February 13, 1991 in the Pathology Department of Newcastle Mater Hospital, Waratah. Their findings included:
  - A well developed, well nourished male infant, weight 8.57 kg.
  - The lungs showed posterior dependant congestion.
  - There was no mention of petechial hemorrhages, specifically in the thymus. The thymus was described as large.
  - Routine and special analyses were negative.
  - Neuropathology examination revealed laminar cortical necrosis of the brain with cystic degeneration in the visual cortex. This is most consistent with old infarcts occurring at the time of his arrest at age 5 months. No evidence of congenital abnormalities was present.
  - Cause of death: SIDS [Sudden Infant Death Syndrome]
  - Note: Dr. Wilkinson, the baby's paediatrician, noticed petechial hemorrhages that were interpreted as agonal. No note in the autopsy report is present.
- Bridget Widcken performed complete biochemical profile on blood samples from Patrick. The results were entirely normal.

<u>Sarah Folbigg</u>

- Sarah was the product of a fullterm pregnancy and her mother, Kathleen Folbigg, received adequate prenatal care.
- Sarah was delivered vaginally following an essentially uncomplicated labor on October 14, 1992. The baby's birth weight was 3020 grams and apgars were 9 at

1 and 10 at 5 minutes. The parents elected to take the baby home with apnea monitoring. She was discharged home with her mother.

- She underwent sleep studies November 15, 1992 and the results were interpreted as within normal limits. Overall well child visits did not indicate any reason for concern; the baby's growth and development were good. The baby had a history of snoring at sleep.
- The father reported increasing tension between the mother and Sarah.
- The baby was put to sleep in a single bed in the parent's room at about 2100 hours on August 29, 1993 without the monitor. The mother reports hearing the child "turn over" at about midnight. She got up to go to the bathroom at 0130 hours on August 30, 1993, did not hear her breathing and found her lifeless. Emergency services were summoned. Emergency medical services were called and they found the child in full cardiopulmonary arrest [essentially DOA], skin warm to the touch, pale and cyanotic. The child was pronounced dead at 0130 August 30, 1993.
- Dr. John Miller Napier Hilton performed autopsy examination at 0800 hours on August 31, 1993 in Sidney. His findings included:
  - A well developed, well nourished female, weight 9.44 kg.
  - Small scratches on the right upper arm, below the lower lip on the left and on the chin.
  - The stomach contained curdled milk.
  - The lungs showed pulmonary edema and congestion.
  - There were petechial hemorrhages present, specifically in the thymus, heart and lung.
  - Routine toxicological analysis was negative.
  - Cause of death: SIDS [Sudden Infant Death Syndrome]
- Bridget Widcken performed complete biochemical profile on blood samples from Sarah. The results were entirely normal.

<u>Laura Folbigg</u>

- Laura was the product of a fullterm pregnancy and her mother, Kathleen Folbigg, received adequate prenatal care.

Witness:_____ **Signature:**_____

- Laura was delivered vaginally following an essentially uncomplicated labor on August 7, 1997. The baby's birth weight was 3260 grams and apgars were 9 at 1 and 10 at 5 minutes. The parents elected to take the baby home with apnea monitoring. She was discharged home with her mother.
- She underwent sleep studies under the care of Dr. Chris Seton. His impression was that the child had a mild central apnea that resolved over time and were interpreted as of no medical significance. At no time did her clinical picture or studies show evidence of obstructive apnea. Her care was monitored by the medical staff at New Children's Hospital, Westmead [Ms Margaret Tanner].
- The father reported concerns about Kathleen's use of the monitor during the day when he was not present.
- Laura received her family medical care from Doctor Sanders of singleton and Dr. Innis of Singleton Heights Medical Practice.
- Laura had recently seen Dr. Innis for her 18-month routine well child visit and vaccination.
- Laura had a history of one week of cold and flu-like syndrome, and she had been administered Demazin for treatment of symptoms. She received her last dose of the medication on February 27, 1999.
- On March 1, 1999 Kathleen took the child to the gym and to her father's place of work to "visit ". Kathleen reported that she fell asleep in the car and she put her to bed upon arrival home at ~ 1100hours. Approximately 30-60 minutes later Kathleen reported hearing the child "coughing in the bedroom." She checked on her ~ 5 minutes later and found her supine and lifeless. She started CPR, emergency medical services were called, and they arrived at 1214 hours finding the child in full cardiopulmonary arrest [essentially DOA], skin warm to the touch. The child was pronounced dead at Singleton hospital at 1245 hours March 1, 1999. SIDS Death Scene Investigation Checklist was completed.
- Autopsy examination was performed by Dr. Allan David Cala at ~2100 hours March 1, 1999 at NSW Institute of Forensic Medicine, Glebe. His findings included:
  - A well developed, well nourished 20-month-old female, weight 11.52 kg.
  - There was lividity on the left side of the face and posteriorly.

- No significant physical injuries were identified on physical examination.
- The lungs showed focal hemorrhage and collapse.
- Examination of the heart showed no gross abnormalities. Microscopic examination of the tissues from the heart revealed inflammatory infiltrate in the heart, consistent with viral myocarditis.
- Toxicological analysis was noncontributory.
- There were petechial hemorrhages present in the thymus.
- Routine toxicological analysis was negative
- Cause of death: Undetermined

- My review of the autopsy materials reveals the presence of myocarditis, most probably viral in origin. Dr. Cala states in his report that his finding of myocarditis is consistent with Laura's recent illness and is probably incidental. I concur with this conclusion.
- Bridget Widcken performed complete biochemical profile on blood samples from Laura. The results were entirely normal.

6. Conclusions:

- In forming my conclusions, I have utilized all of the materials made available to me including the medical history and records, the autopsy reports and materials, the police investigative documents, the interview transcripts from Kathleen Folbigg, diary entries, witness statements, and listening device materials.
- The materials and investigative information provided in this case are of excellent quality and are sufficient for me to render an opinion to a reasonable degree of medical certainty.
- It is my opinion that these four children were all the victims of homicidal assaults that resulted in their suffocations. Suffocation is the interference with breathing by external obstruction of the nose and mouth. This process will take approximately 4 to 5 minutes to complete. During the first 1 and 1/2 to 2 minutes, while they are still fully conscious, the child will fight aggressively for their life. In small infants, this typically does not result in any external signs or physical evidence.

- I have participated in the investigation of both accidental and homicidal suffocation in children in over the course of my 20 years as a practicing pediatric forensic pathologist. Unfortunately multiple infant homicides within one family are now well documented in the literature and in forensic experience. Typically the perpetrator does not confess to the crimes but in many cases such as this the facts of the case make the diagnosis. Important facts in this case that lead to the conclusion of homicidal suffocation include:
  - The autopsy fails to identify any known natural disease or disease process that could explain the sudden deaths of these infants. All four children were growing and developing normally for their age and circumstance. Despite Patrick's handicaps he was advancing well.
  - The autopsy findings in these babies are all consistent with death by suffocation.
  - The infants were all in the care of the same person at the time of their death, their mother, and she was the last person to see each of them alive.
  - None of the deaths in this case can be attributed to SIDS [Sudden Infant Death syndrome]. It is well recognized that the SIDS process is not a hereditary problem and the statistical likelihood that 4 children could die from SIDS is in excess of 1 in a trillion.
  - The diagnosis of SIDS requires that following a complete investigation and autopsy no other cause of death is identified. Forensic standards of practice would not allow for consideration of a second diagnosis of SIDS after a second sudden death and by the time a third child has died, the death must be investigated as a homicide.
  - Patrick's sudden, profound and irreversible brain damage is consistent with and diagnosed as a hypoxic episode. Hypoxia in this case is synonymous with asphyxia and unfortunately heralds the fatal event in retrospect. No natural disease or process has been identified to explain this event. In my opinion, the cause of Patrick's cardio-respiratory arrest is the same process that killed him and his siblings.

7. <u>In my opinion the cause of death and manner of death should be listed as follows:</u>

Caleb Folbigg-	**Cause of Death:** Undetermined
	Manner of Death: Undetermined
Patrick Folbigg -	**Cause of Death:** Suffocation
	Manner of Death: Homicide
Sarah Folbigg -	**Cause of Death:** Suffocation
	Manner of Death: Homicide
Laura Folbigg -	**Cause of Death:** Suffocation
	Manner of Death: Homicide

Witness:_____ Signature:_____

Witness:_____ **Signature:**_____

I am Peter Jeremy Berry. My qualifications are BA, MB, BChir, FRCP, FRCPath and FRCPCH. I have been consultant paediatric pathologist to the Bristol Royal Hospital for Sick Children for 17 years, and professor of paediatric pathology in the University of Bristol for 9 years. I have a special interest in the investigation of sudden death in infancy and early childhood, and am author of a number of research papers and articles in that field. For many years I have carried out examinations of children found dead in suspicious circumstances, and I am regularly instructed by the Crown and defendants to provide expert evidence in the criminal courts.

At the request of Detective Senior Constable Ryan of New South Wales Police Service I have read:

1. The medical records of Kathleen Folbigg
2. The medical records of Caleb Folbigg
3. The medical records of Patrick Folbigg
4. The medical records of Laura Folbigg
5. The medical records of Sarah Folbigg

I have studied the post-mortem reports and examined microscope slides prepared from tissue samples collected during the post mortem examinations of Caleb, Patrick, Sarah and Laura Folbigg.

I have also reviewed two folders of items from the police brief of evidence including the following:

1	Statement from Mr Craig FOLBIGG
2.	Transcript of police interview with Kathleen FOLBIGG
3.	Psychological report by Roz GARBUTT
4.	Diary Entry dated 19.2.89
5.	Personal diary dated between 4.6.96 - 5.6.97 (extracts attached)
6.	Handwritten letter by Kathleen Folbigg (6 pages)
7.	Handwritten letter by Kathleen Folbigg (10 pages)
8.	Typed letter by Craig Folbigg (2 pages)
9.	Handwritten letter by Craig Folbigg (6 pages)
10.	Record of interview dated 9.12.68 (Thomas Britton)
11.	Mother's Day Card
12.	Handwritten letter by Kathleen Folbigg (2 pages)
13.	1992 May Gibbs personal diary
14.	Personal diary dated July 1999
15.	Personal diary dated between 6.6.97 - 10.4.98 (extracts attached)
16.	Diary entries dated 1.1.99 & 3.1.99.
17.	Handwritten letter by Kathleen Folbigg (2 pages)
18.	Diary entry dated 19.6.99 (8 pages)
19.	Handwritten letter by Kathleen Folbigg (3 Foolscap pages)
	Handwritten letters by Craig Folbigg (6 pages)
	Handwritten notes by Kathleen Folbigg (10 pages)
20.	Telephone Interception Transcript AT0001
	Telephone Interception Transcript AT0002
	Telephone Interception Transcript AT0003

21. Listening Device Transcript number 1- 22.7.99
Listening Device Transcript number 2 - 23.7.99
Listening Device Transcript number 3 - 23.7.99
Listening Device Transcript number 4 - 24.7.99
Listening Device Transcript number 5 - 24.7.99
Listening Device Transcript number 6 - 26.7.99
Listening Device Transcript number 7 - 27.7.99

Background

The following account of the deaths of Caleb, Patrick, Sarah and Laura Folbigg is derived from the above documents.

Mr Folbigg is 37 years of age and was one of a family of nine children, one of whom had a child who died in infancy (but not of a genetic disorder or sudden infant death syndrome).

Craig Folbigg was working as a mobile crane operator in 1985 when he suffered a back injury and was off work receiving compensation. He met Kathy Marlborough at a nightclub and began a relationship. He learned that she was adopted and had a strained relationship with her adopted mother. When he met them he found that her mother was dominating and did not get on with her.

On 26th January 1986 they began to live together. In August 1986 they became engaged. At that time he was back working, and Kathy was working in a restaurant. They bought their own home and moved in in May 1987.

They were married on 5th September 1987. In 1988 Kathy became pregnant with Caleb Folbigg. In November 1988 Craig received a compensation payout from his employer with which he paid off the mortgage to their home and other debts. He then began working as a vehicle valuer.

On 15th December 1988 while she was pregnant with Caleb his mother had been admitted following a fainting or fitting episode. The following day she had a "rigor" and felt and looked miserable. She developed a rash. Because relatives had had chickenpox she was nursed in a separate room.

Caleb Folbigg

Caleb Folbigg was born on 1st February 1989 following spontaneous onset of labour and artificial rupture of the membranes. There was meconium stained liquor and two variable decelerations with slow recovery. Delivery was by Kielland's forceps and episiotomy. His birth weight was 3.28 kilograms. Apgar scores were 9 at one minute and 9 at five minutes. In the neonatal period he had transient tachypnoea requiring oxygen. His chest X-ray was normal. Caleb was slow to suck initially, but went home with his mother well on 5th

February. Kathy used to get up at night to attend to Caleb. Craig noted that he would suck his bottle when feeding and then stop to take a breath. Kathy said that it took a little longer to feed Caleb than a normal baby as when feeding he would not breathe through his nose. He would have to stop and start feeding to catch his breath. By the time they left hospital this had improved a little bit so that it wasn't distressing him.

Caleb was seen on 17th February at two weeks of age with mild inspiratory stridor and slight costal recession, especially when upset or put on his back. His father had been concerned about his noisy breathing while feeding. His growth was on the 50th percentile. He was thought by their doctor to have congenital laryngeal stridor (floppy larynx), a common condition which resolves with time.

On 19th February 1989 he was given a feed and put down at in an adjoining room to his parents' bedroom. He was checked by Kathy Folbigg at about 10.00-10.30pm and was asleep in his cot. They went to bed leaving the door adjoining their bedroom and the sun room open with a lamp in the sun room on. His mother found him lifeless in his cot at 2:45am and noted him to be cold with a small amount of blood and froth around his mouth. Craig remembers waking up and hearing Kathy screaming that there was something wrong with the baby. She was standing over the cot in her pyjamas holding her hands on her forehead. When he picked Caleb up he noted that he was still warm. He commenced cardio-pulmonary resuscitation and she called an ambulance. The ambulance crew reported him as being warm and in asystole. He was pale around the nose and mouth (another ambulance report states that he was cold). After attempting further resuscitation the ambulance officers confirmed that he was dead. Kathy appeared to get over the death quicker than Craig and didn't like to talk about Caleb.

A further account is given in the statement of Kathy Folbigg (23 July 1999). On 19th February Caleb went to bed as usual. He had his own bedroom with a basinet. He had his early-morning feed, fell asleep in her arms and she put him back to bed. She went to the toilet before returning to her own bed. She went in to check on him putting her hand on him and he didn't seem to be moving so she flicked on the light and noticed that he wasn't breathing. Everything seemed to be panic from then on. She thinks she threw the covers off, picked Caleb up and ran through to Craig. Caleb was blue around the lips and pale. (In another statement she said that Caleb had a blood and froth around his mouth). Diary entries show that on 19th February she had trouble getting Caleb to sleep, and the last entry says "finally asleep" at 2pm and is ringed with 2 exclamation marks.

Post-mortem examination

A post-mortem examination was carried out the same day at 11:45am by Dr R Cummings at the instruction of the coroner.

External examination showed a normal baby, 3.97 kilograms, 55 centimetres long. There was posterior post-mortem staining and rigor mortis was well developed. There was no injury and the baby appeared well cared for.

Internal examination:

Normal heart weighing 25 grams.

Left lung 34 grams, right lung 53 grams with mottled pleural surfaces. The cut surfaces were moist.

The stomach contained a large quantity of curdled milk.

The liver weighed 178 grams.

The spleen weighed 15 grams and the thymus 18 grams.

Each kidney weighed 21 grams.

The brain weighed 465 grams.

There is no mention of froth or petechial haemorrhages in the post-mortem report. There is no record of any abnormality of the larynx or trachea.

Microscopic examination

The lungs showed incomplete aeration with extravasation of red blood cells and focal eosinophilic exudate.

No abnormality was described in other organs. The brain was apparently not examined histologically.

Toxicology of the liver and stomach contents was negative.

The cause of death was given as **Sudden Infant Death Syndrome**.

Patrick Folbigg

Some time that year the natural sisters of Kathy made contact with her and told her that her natural father had murdered her natural mother. He went to prison, and was later deported to England. She appeared to take this information in her stride.

After a party, and also later that year Kathy spent a night away from home possibly with a man. Shortly after this she became pregnant with Patrick Folbigg, their second child.

Patrick Folbigg was born on 3rd June 1990 after spontaneous onset of labour with artificial rupture of the membranes by vertex presentation at 39 weeks gestation.

The pregnancy was normal apart from an admission on 27th February 1990 at 25 weeks gestation with a three-day history of left groin pain. This was thought to be possibly due to a urinary tract infection, but the pain resolved spontaneously and she was discharged with a prescription for amoxil (a urine culture subsequently showed a mixed growth only).

Apgar scores were 7 at one minute and 8 at five minutes. The birth weight was 3410 grams. The placenta weighed 920 grams. His head circumference was 33.5 centimetres and crown heel length 48.3 centimetres. His mother suffered a minor perineal tear which was sutured. Her lactation was suppressed.

They went home on 8th June with Patrick being bottle-fed. They were both pleased with their new baby. He slept in a cot in the bedroom off the dining room. Craig left his job to spend all his time with his family. Again, Kathy would get up during the night to attend to Patrick because Craig was a heavy sleeper. (After about three months Craig began working again).

Sleep studies were arranged for 14th June. An electrocardiogram and serum electrolytes were normal. Thesleep study was normal.

A barium swallow showed no gastro-oesophageal reflux. Contrast in the nose suggested incoordination of swallowing.

Patrick Folbigg attended his general practitioner on a number of occasions for vaccinations, mild viral infections and other childhood illnesses. However, his significant illnesses are detailed in his hospital records.

On 17th October Kathy put Patrick to bed in his cot at about 8:30pm. At about 10:30pm Craig saw Patrick lying on his back in the cot covered with a sheet and blanket. They went to bed leaving the lamp on in his room. His mother said that Patrick had been coughing at 3am when she attended to him. She was alerted at 4:30am because she heard him gasping, and noted that he was blue around the lips, lifeless, floppy, and making minimal respiratory effort. Craig woke up to the sound of Kathy screaming. He saw Kathy standing in front of the cot with the rail in the up position and Patrick lying on his back with the covers pushed down near his feet. He appeared lifeless. Craig began resuscitation and told Kathy to call an ambulance. He heard faint laboured breathing. Kathy stated that resuscitation was not performed, and that Patrick soon gave a high-pitched cry. The ambulance arrived at 04:41 about 20 minutes later and Patrick revived slightly when paramedics gave oxygen. A paramedic recorded that the baby was having respiratory difficulties and was pale around the face and listless. The baby showed tracheal tug and intercostal recession.

On admission to hospital he was lethargic, cyanosed, and responsive only to painful stimuli. After about 15 minutes of oxygen treatment he became more alert and remained pink without high concentration oxygen. The admitting doctor noted that he was appropriately grown and arching his back. There were no signs to suggest any serious illness such as meningitis, and no evidence of trauma. Sugar in the urine in the absence of a high blood sugar with protein and blood was thought to be a response to an acute asphyxiating event. A chest X-ray was later reported to show signs which could have been due to bronchiolitis (lung fields of large

volume with increased lung markings in the peri-hilar region). Virology was subsequently negative.

By the following day, he was back to his normal self but at 9pm he developed a generalised seizure and was given diazepam. Following further fits he was started on phenobarbitone. He was seen by a paediatric neurologist and a lumbar puncture performed on 20th October which was normal. A metabolic screen was collected. A CT scan demonstrated hypodense areas in both temporal and occipital lobes. Phenytoin was added to his treatment because of further convulsions and acyclovir given to cover the possibility of herpes simplex encephalitis (investigations for herpes simplex subsequently negative). An electroencephalogram on 18th October 1990 was reported as normal. He was discharged on 29th October with a diagnosis of a seizure disorder and a respiratory tract infection.

He was readmitted on 4th November with a prolonged seizure resembling an oculogyric crisis. This resolved spontaneously after 90 minutes. He was found to be febrile and to have bilateral conjunctivitis, a fine rash, and an upper respiratory tract infection. A lumbar puncture showed normal fluid, and cultures of blood and urine were negative. An eye swab yielded adenovirus. A repeat electroencephalogram showed multi-focal epileptogenic foci. Comparison with the two previous electroencephalograms showed a progressive deterioration. A further CT scan on 5th November showed generalised loss of brain substance with patchy enhancement in both occipital lobes. High-density in the pre-contrast scan was thought to be due to dystrophic calcification. These films were subsequently seen by Professor de Silva who suggested the possibility of child abuse such as shaking injury. Patrick was discharged on 10th November with the provisional diagnosis of a seizure of disorder perhaps due to an encephalopathy.

He was admitted again on a 14th November with a generalised seizure resulting in apnoea. He had an upper respiratory tract infection. It was noted that he had lost the ability to fix on a face or to follow, and he was found to have a degree of cortical blindness. A cardiac ultrasound scan on 16th November showed no evidence of intra-cardiac thrombus. On 18th November he developed gastroenteritis. Stools collected on 20th November were positive for rotavirus. He was discharged on 22nd November.

Around this time Craig found that Kathy had been keeping a diary in which she said she was finding things too much and was going to leave Patrick and Craig. He told his sister Carol about this and they discussed it with Kathy who agreed to stay and work things out.

During this time Patrick was "a handful". Kathy had to give medication and physiotherapy. She appeared to suffer extreme anxiety and what Craig thought was depression. She became easily stressed and upset.

On 22nd December he suffered an oculogyric crisis and was admitted to hospital again and discharged the following day.

In January 1991 he was assessed and treated in the physiotherapy department.

On 12th February 1991 he had a fever during the evening and his parents wondered whether he had a seizure at that time. He slept well and played with his father early in the morning of 13th February. Craig states he left for work at about 7:30am. Patrick appeared his normal self and was eating. At 10am that morning Kathy phoned him at work screaming "it's happened again". He drove straight home when he saw the ambulances arriving. Patrick was lying on his back in his cot with the inside rail in the up position. He again began resuscitation. Patrick was limp, blue around the lips and warm to the touch. An ambulance was called at 10:03am. On the arrival of the ambulance at 10:10am Patrick was pulseless and not breathing (the respiratory rate is recorded as zero, but the ambulance man noted shallow breathing. Another officer suggests it may be an error on the case sheet, and that no respiration was present. Another is categorical that the baby was not to breathing). The baby was reported to the peripherally cyanosed with warm skin. On arrival in hospital an ECG monitor showed asystole. Despite full resuscitation no cardiac output was achieved at any stage and resuscitation was stopped after 20 minutes, death being pronounced at 10:40am on 13th February 1991.

Craig gave authorisation for a post mortem examination.

Later he asked Kathy what had happened and she said "I put him to bed for a nap. When I went to check on him I found him how he was." She said she had put him down to sleep at about 7:30am and discovered him lifeless a couple of hours later. She then called her husband at work and also the paediatric neurologist.

Kathy Folbigg gives the following account (abstracted from statement of 23 July 1999); Patrick was an unplanned pregnancy. When he was born he had no problems with breathing and no general health problems. She thought he looked like Craig. On 18th October 1990 Patrick was around three months of age. He was sleeping in a different room which they had just done up. She had fed him as usual around 12 or 1 o'clock in the morning. She found herself awake and went to check on him on the way to the toilet. She noted his breathing was laboured to and so she put on the light finding him lethargic and unresponsive with closed eyes and trying to take a breath. She called for Craig and one of them called the ambulance. She remembers that as soon as the ambulance people put oxygen on him his eyes opened although he continued to have difficulty in breathing. One moment he was lying on the bed in the hospital unresponsive, and the next minute he was awake, screaming and panicking because of all people who were there. On the second or third day he began to have a fit in Craig's arms. From October through to Christmas they were in and out of hospital trying to control the fits. On his first birthday they were told that he was blind. He needed physiotherapy to help his development. Kathy describes herself as being on auto-pilot during this period but receiving a lot of family support.

On the day that Patrick died they followed their usual routine. Kathy put him to sleep in Caleb's old room for a morning nap. She looked into the room some time later and noticed that he was on his back which was unusual because she used to lay him on his side. He was pale and wasn't breathing. She did not remember what time this was and couldn't remember if Craig was home or not. The whole day was confused. However, she remembers that she was alone with Patrick in the house that day.

Summary of metabolic and other investigations in life.

Normal rectal biopsy with no neuronal inclusions.
Normal ammonia, calcium, magnesium, and glucose.
White cell enzymes were normal ruling out adrenoleukodystrophy, Refsum's disease, Zellweger's syndrome and other generalised peroxisomopathies.
Long chain fatty acid studies normal.
Urine mucopolysaccharide screen normal
Serum carnitine was normal.
Urine amino acids, methylmalonic acid, organic acids and lactic acid values were normal.
Arterial blood lactate was slightly raised at 1.6 (normal 0.3-0 mmol per litre) on one occasion.
A repeat blood lactate level was normal.
Anti-nuclear antibodies negative.
TORCH screen negative.
No leukocyte inclusions identified.

Post-mortem examination

A post-mortem examination was carried out on 13th February 1991 at 12:30 hours.

External examination

The body was that of a normally formed a well-nourished male child weighing 8.57 kilograms, head circumference 44 centimetres, crown rump length 53 centimetres, crown heel length 77 centimetres, and foot length 10 centimetres. There was no external abnormality.

Internal examination

The skull was normal. The brain weighed 750 grams (versus 714 grams expected).

The larynx, trachea and bronchi contained frothy mucoid fluid. The right lung weighed 55 grams and the left 50 grams. Both lungs were congested posteriorly.

The heart weighed 49 grams and was structurally normal.

The thymus weighed 30 grams. It was described as an enlarged. The spleen weighed 27 grams

The liver weighed 284 grams and was congested.

The pancreas weighed 15 grams and appeared normal.

The kidneys appeared normal, the right weighing 32 grams and the left 33 grams. 10 millilitres of urine were collected for metabolic studies.

The pituitary, thyroid gland, and adrenal glands were normal, the latter weighing together 6 grams.

Numerous samples were collected for microscopy, culture, toxicology, cytogenetics, metabolic studies and electron microscopy.

Neuropathology

The brain weighed 750 grams after fixation. The gyri of both occipital lobes (visual cortex) were shrunken, thinner and more undulated than normal and the sulci were widened. On section, the cortical grey matter of the visual cortex in both hemispheres was thinner than normal and showed cystic degeneration, the cysts measuring 1 - 2 mm in diameter in a linear pattern at the junction of grey and white matter. Underlying white matter was firmer than normal and appeared expanded. Similar areas of firm white matter were present in the left frontal and both parietal lobes.

Microscopic examination showed no evidence of any neuronal storage disease or leukodystrophy. The major changes were old infarcts and gliosis of old laminar necrosis most severe in the parietal and occipital area. In the deeper parts of the cerebrum and in the cerebellar and brain stem nuclei there were neurones showing simple atrophy attributed to the baby's seizures. There was a slight lymphocytic infiltrate in the leptomeninges. There were no features suggestive of toxoplasmosis or cytomegalovirus, and the distribution of the lesions was unusual for herpes simplex. The appearance suggested the result of an episode of cardio-respiratory arrest that the baby suffered at about five months of age.

Microscopic examination of the lungs showed no significant abnormality apart from small foci of alveolar collapse in the periphery of the lung.

Microscope slides of the heart, skeletal muscle, liver, spleen, thymus, pancreas, kidneys, thyroid, adrenal glands, testes, and intestine showed no abnormality other than autolysis.

Results of additional post-mortem studies

Post-mortem blood cultures grew mixed organisms which were thought to reflect contamination. Cultures of lung tissue were negative for bacteria, viruses, and mycoplasma.

Phenobarbitone and carbamazapine levels were within the therapeutic range.

Investigation for primary lactic acidosis was not thought to be indicated.

Normal male karyotype.

Patrick was cremated.

Kathy did not want to stay on in their house and so they sold it and purchased another home. Kathy was then employed as a sales assistant at a baby clothing store. Craig could not understand how she could cope with this.

Kathy gave Craig an ultimatum that they would have another baby or she would leave him. After thinking about it for a week he agreed. She became pregnant in February 1992 with Sarah.

Sarah Folbigg

Sarah Folbigg was born on 14th October 1992 following spontaneous onset of labour at 39 weeks' gestation by scan. The birth weight was 3.02 kilograms and the head circumference 34.5 centimetres. The pregnancy had been complicated by an admission for early bleeding on 21st February. This subsided spontaneously. The placenta was delivered by controlled cord traction. Apgar scores were 9 at one minute and 10 at five minutes. She was nursed on an apnoea mattress. Her mother chose to breast feed and the parents asked for early baptism. The full neonatal examination was normal apart from mild plethora. Training in cardio-pulmonary resuscitation was given and Sarah went home with an apnoea alarm on 19th October.

She was bottle fed and slept in a crib by her parents' bed. After two or three months she went into a cot in her own bedroom adjacent to the bedroom where Kathy and Craig slept. She used to snore when she was a sleep. She slept with an apnoea blanket under the mattress. They found the apnoea mattress quite difficult to cope with.

When seen at 16 days of age she was 500 grams above her birth weight, and her general and neurological examination were normal.

A sleep study on 5th November showed very few sleep apnoeas and some periodic breathing. The results were judged to be normal. However, in a letter dated 16th November Dr David Cooper suggests that Theophylline could be of help in view of some quite long episodes of hypoventilation.

A urine metabolic screen also on 5th November showed dicarboxylic aciduria without significant ketosis. " Further investigation is indicated if child is not on MCT containing feeds".

At four weeks of age she was developing normally.

At six weeks of age she was jittery at times but within the normal range. She appeared to be thriving and gaining weight. She was vigorous, alert, and interacted well.

When Sarah was about two and a half months old Kathy went back to work on Saturdays and Sundays for financial reasons.

When seen by her paediatrician on 21st January at the age of three months she appeared well with her weight along the 75th percentile, head circumference along the 75th percentile and length between the 50th and 75th percentiles. She was neurologically and developmentally normal. A test for MCAD deficiency was said to be normal. A further sleep study and urine

metabolic screened was arranged. Her parents were described as understandably anxious, and Sarah as always wanting to be held.

At four-and-a-half months of age she was thriving and developmentally normal. When seen by her paediatrician she had a viral upper respiratory tract infection. Her mother requested a further sleep study.

She was seen five times by her general practitioner and given usual childhood vaccinations and treatment for a virus infection and a fungal skin rash. On 18th August she was prescribed Flucloxacillin for a cold like illness (this was discontinued by her parents on about 26th August because of difficulty in administration) and on 26th August 1993 she was seen for a croupy cough.

Craig noted that Kathy became easily irritated and stressed with Sarah and he badgered her to "mellow out". Kathy left her job at the baby store in the middle of 1993.

On 29th August 1993 she ate normally and was put to bed in a single bed in her parents' bedroom at about 9pm. The apnoea monitor had been discontinued for about a week. At about 9.30 or 10pm she was observed to be snoring. Her mother heard her turn over in her sleep at about 12 or 12:30am. She got up to go to the toilet at 1:30am and could not hear Sarah breathing. She turned on the bedroom light and saw that the child had a blue colour to her face and a discharge from the nose. She roused the father who commenced CPR and called an ambulance. A phone call was received at 01:25am. When an ambulance officer arrived at about 1:30am he found Craig Folbigg giving cardio-pulmonary resuscitation to Sarah on the floor. She was fully clothed in a Bond brand ski suit, blue around the mouth, and she had mucus and vomit in her mouth. She was not breathing. Full resuscitation was instituted. There was no electrical activity in her heart. The ambulance crew told her parents that she was dead. The ambulance report indicates her temperature as both normal and cold. The child was conveyed to Maitland hospital where life was pronounced extinct at 4:30am.

When a police officer attended the premises Sarah Folbigg was dressed in a yellow tracksuit, pink slippers, and wrapped in a crocheted blanket. The colour of the skin of the face and neck and hands was pale cream. He noted a small amount of semi-dried mucus material in the nostrils. An inverted U-shaped mark on the bridge of the nose was thought to be consistent with that made by an oxygen mask. He observed that both parents were "genuinely very distressed".

According to the statement of Craig Folbigg, on Sunday 29th August 1993 Sarah was suffering from a cold like illness. They went out for the day and Kathy was agitated when they got home. Sarah had a bath and they had dinner at about 5:30pm when Sarah ate solids. Sarah and Craig played normally together and watched television. At 8pm Kathy took Sarah to their bedroom to put her to sleep in a single bed which they had placed in the room for her to sleep in. He had set this bed up in their bedroom because Kathy wanted Sarah out of her cot and he felt that she would be safer in the same room as them. This bed had been in that room for the past two nights and they had stopped using the apnoea blanket.

Sarah began to cry because she didn't want to go to bed which made Kathy angry and Craig heard her making growling noises. He went up to the room to inquire what the problem was, and Kathy said "Nothing. Get out.". He went back down to the lounge and heard Kathy return carrying Sarah in her arms. She stood about three paces in front of where he was sitting and dropped Sarah on to his lap in such a way that he had to catch her.

He settled Sarah down and put her to sleep which took about half-an-hour. Kathy got into bed while he was doing this. He put Sarah in her bed between 10:30 and 11:00pm. Kathy was either asleep or ignoring him. He awoke at about 1am and saw that Kathy was not in bed with him. He could see by a street light that Sarah was in her bed. He thought that Kathy was in the bathroom and went back to sleep.

At about 1:30am he was woken by Kathy screaming and he sat up and saw that the light was on and Kathy was standing at the doorway of their bedroom with the door open. He saw that Sarah was lying on her back on her bed with the covers off. He began cardio-pulmonary resuscitation. Kathy was sitting on the floor in the hallway with her knees up to her face crying. He told her to call the ambulance which she did.

Kathy appeared to be devastated but managed better than Craig. A couple of days later he asked her what had happened before he woke up that morning. She said, "I had been to the toilet and just flicked on the light to check on the baby, and the rest you know."

Kathy Folbigg gave the following account in her statement of 23 July 1999 (summarized): The birth of Sarah was straightforward, and they stayed in hospital for about five days. She was a good feeder and the result of a planned pregnancy. She was nicknamed the catnapper because she would not sleep for longer than 15 or 20 minutes at a time. Kathy agrees that Sarah caused her more stress than Patrick.

The day before she died (29th August 1993 when she was about 10 months old they took her to the park and had a good day. That night they put her to sleep in a single bed in their own bedroom. The mattress was angled with pillows placed under it to face the wall. She slept for a couple of hours and then decided to get up and play in the lounge room. Kathy went to bed leaving Craig to play with her. He must then have put her to bed and gone to bed himself. (She denied having thrown the child down, but accepted the inconsistency between her account and Craig's concerning whether Sarah had gone to bed and then got up again).

Kathy got up to go to the toilet and on her return checked her and saw that she had moved. She was flat on her back with one of her arms hanging out. She was cool to the touch. She did not hear any breath sounds so she touched her arm and pulled back the covers. She woke Craig up straight away and he turned on the light. She called an ambulance while Craig attempted resuscitation. Two ambulances arrived, and events after that were pretty much a blur. She says that Sarah had a cold that day but was otherwise all right and was not on any medication. They were not using the apnoea mattress because they could not work out a way of using it in the single bed.

Following her death they were interviewed by the police.

Post-Mortem Examination

A post-mortem examination was ordered by the Coroner. This was carried out at 08:00 hours by Professor John Hilton at the New South Wales Institute of Forensic Medicine.

External examination.

The body was that of a well-nourished clean caucasian female with minor abrading and drying of the lips. The body temperature was 25 degrees C by the rectal route at 11:00 am on 30th August. There was generalised rigor mortis. There was posterior hypostatic staining. Minor lividity was noted on the right side of the face with blanching of the left cheek and left side of the forehead. A 1.5 centimetres scratch was present on the anterolateral aspect of the right upper arm. The frenula of the lips were normal. There were two tiny punctate abrasions present, one immediately below the the lower lip on the left side, the other slightly to the left side of the mid-line of the chin.

Internal examination

The skull and membranes of the brain appeared normal. The brain was retained intact for formal neuropathology with a portion of the upper cervical cord. The middle ears were normal. Cerebral spinal fluid was clear.

The uvula was of normal size but appeared somewhat congested on its anterior surface. The epiglottis was normal. Stomach contents were present in the trachea and major bronchi. The larynx, trachea and Major bronchi were otherwise normal.

The lungs showed focal areas of collapse with a geographic pattern. Occasional petechial haemorrhages were present and there was minor congestion and minimal oedema.

The heart was normal.

The thymus showed occasional petechial haemorrhages on its surface and within the substance of the gland but was normal in size shape and location.

The stomach contained a moderate quantity of curdled material.

The liver appeared normal.

The pancreas, spleen, bone-marrow, adrenal glands, kidneys, bladder, and genital organs were normal.

No abnormality was detected in the bones, joints, or skeletal muscles.

The pathological findings were summarised as:

1. Focal pulmonary collapse

2. Modest pulmonary congestion and minimal oedema

3. Occasional petechiae on pleura, epicardium, and on and in the thymus

4. Congested haemorrhagic uvula lying anterior to the epiglottis

5. Aspiration of gastric content (?artefactual)

The cause of death was given as:

1 A. Sudden infant death syndrome.

Subsequent microscopic examination showed marked vascular congestion of the pharyngeal aspect of the uvula.

The larynx showed a light lymphocytic inflammatory infiltrate deep to the respiratory epithelium.

Salivary gland showed two small acute inflammatory foci in the interstitium. There were no viral inclusions.

A section of diaphragm showed two foci of individual muscle fibrillary degeneration.

The spleen showed focal congestion.

The lungs showed congestion and oedema. In one section there was a light interstitial acute inflammatory infiltrate which could be seen around occasional bronchioles. A further section of lung showed multiple neutrophils within the lymphoid deposits and again some interstitial infiltration.

Examination of the brain showed no macroscopic or microscopic abnormality.

Additional post-mortem studies

Lung: no virus isolated.

Lung: bacterial culture gave a mixed growth of doubtful significance.

Spleen: bacterial culture gave a moderate growth of coliforms of three types.

Large intestine contents: bacterial culture yielded no pathogen.

Small bowel contents: bacterial culture yielded no pathogen.

Biochemical analysis of vitreous humour showed a sodium of 145, chloride 133, urea 5.5 and glucose 0.4 mmol per litre.

Comprehensive screening tests for drugs and other common poisons were negative. The blood alcohol level was nil.

Sarah was cremated.

After Sarah died Craig buried himself in his work and his relationship with Kathy suffered. They moved to another house, but began to grow apart. In 1995 Kathy gave him an ultimatum to see a counsellor which he refused and she moved out of their house. She wrote him a six-page letter explaining her feelings. Eventually they did attend a counsellor. They separated between January and April 1995 but still kept in touch. She was dieting and exercising. He asked her to resume their marriage again which they did, moving into a flat together. Kathy got a job and Craig was earning good money. They purchased another home with a swimming pool.

They moved into this house in January 1996 when Kathy appeared to be enjoying life going to the gymnasium frequently. In 1996 they planned another child and she became pregnant in November with their 4th child, Laura.

Laura Folbigg

On 3rd May 1996 Craig and Kathleen Folbigg consulted Dr Christopher Seton, a staff specialist in the sleep disorders unit at the new children's hospital at West Mead. They were seeking advice about the risk of SIDS following the loss of their three previous children. The history they supplied was considered highly suggestive of familial clustering of obstructive apnoea. This included Mr Folbigg suffering heavy snoring as did his siblings and Patrick and Sarah Folbigg. Sarah in particular was a very loud snorer who suffered witnessed apnoea and choking episodes during sleep according to Dr Seton's letter. Dr Seton noted that Caleb was facially very similar to his mother, while Patrick and Sarah were facially similar to their father.

When Mr and Mrs Folbigg were expecting the birth of their 4th child arrangements were made for a sleep study when the child was born.

Laura Elizabeth Folbigg was born at term after an uncomplicated pregnancy on 7th August 1997 by spontaneous vaginal delivery. The placenta weighed 775 grams. Apgar scores were 9 at one minute and 10 at five minutes. The birth weight was 3.26 kilograms. She was initially breast fed for the first two weeks, but then was bottle fed.

A sleep study was carried out by Dr Seton on 19th August 1997 which demonstrated mild central apnoea and no obstructive apnoea. Laura also underwent a full biochemical, blood and metabolic investigations which were normal. In particular, urine amino acids, methylmalonic acid screen, lactate and organic acid profile were normal. She was discharged on 21st August

1997 with a home monitoring device designed to record and download breathing and heart information. They were instructed on cardio-pulmonary resuscitation and how to operate the monitor. (Laura was monitored on the machine for approximately 12 months without complication.)

Kathy started going to the gymnasium again and would give Laura to a neighbour to look after until Craig got home. He did not agree with this and she stopped. She used to leave Laura with others when she began to going to the gym regularly.

A sleep study on the 2nd October 1997 showed mild improvement. The mild central apnoea had improved and was totally normal on 3rd 1998. Laura did not at any time show signs of suffering from obstructive apnoea which is "a potentially inherited breathing disorder associated with SIDS".

When Laura was about three months old they moved her into her own bedroom adjacent to their bedroom. At first she slept in a cot. Kathy and Craig were sleeping in separate rooms and appeared to grow apart as they were both just living for Laura.

In March 1998 Craig Folbigg expressed concerns that his wife was not utilising the monitor as diligently as she should, and wrote to Margaret Tanner a nurse consultant with the sleep study unit. "strangely though I feel that Kathy finds it all tedious and frustrating and would probably rather not use it at all, merely entrusting Laura's survival to fate! You would think that after all she had been through as a mother she of all people would be more diligent with the monitoring". The monitor was eventually returned to the new children's hospital around Laura's first birthday.

On 9th March 1998 she attended the accident and emergency department for vomiting and diarrhoea. A diagnosis of a viral gastroenteritis was made.

On 22nd June 1998 she was seen with croup following an upper respiratory tract infection over the previous two days. She was allowed home the same day after a period of observation and oxygen treatment.

Laura was seen by Dr Paul Innis her general practitioner on several occasions over seven months. On 14th August 1998 she had flu-like symptoms. On 19th October 1998 she presented with a burn on her left forearm and palm which was treated over the next eight days with daily dressings. On 19th January she presented with a rash which was initially thought to be allergic, but was subsequently accompanied by fever and a sore throat. On 5th February she was well and attended for her 18 month immunisation.

In the middle of February 1999 Kathy gave Craig another letter to read stating that she was thinking of leaving. They talked about their relationship and she agreed to stay.

At about this time they purchased a single bed for Laura.

On Monday morning the 1st March 1999 Craig woke Laura up at about 6:30am. She was suffering from a runny nose and congestion of her chest. He fed her as usual and they watched television.

Kathy got out of bed at about 7:00am and Craig began to get ready for work. Laura became upset and Kathy and Craig had an argument. Kathy became very agitated.

Craig drove to work, but at about 8:15am received a telephone call from Kathy when she apologised and Craig agreed to try harder.

At about 11am that morning Laura and Kathy went to his place of work and she played in his office for about half an hour. Craig told them that he would be home for lunch as usual. At around lunchtime he received a phone call to go to hospital because there was something wrong with Laura. Kathy was sitting in a waiting room and Laura was lying on her back in an adjoining room being attended to by hospital staff. Kathy said "Laura fell asleep on the way home. I put her into bed. I went to feed the dog. I had a play with the dog. I heard Laura coughing on the monitor but didn't check on her straight away. About 10 minutes later, I checked on her and found her."

An ambulance had attended at 12:14pm. Officers found Laura's mother giving cardio-pulmonary resuscitation on a breakfast bar. She stated that child and had been heard coughing, and when checked approximately five minutes later was found not breathing. She said that Laura had been suffering from a runny nose and cough for a couple of days (she had been giving her Demazin (chlorpheniramine and and phenylephrine) for the symptoms, last taken on 27th February). The baby was warm, blue and dressed in a pair of floral tights and a small top. There was no blood, vomit or foreign object in the child's mouth. An ECG monitor was attached and registered asystole or bradycardia (the accounts of the ambulance crews differ). The crew continued resuscitation and Laura was transferred to hospital at 12.35 pm. where she was found to be cyanosed with no heart beat, no respiration, and fixed dilated pupils. Despite full resuscitation death was declared at 12:45 pm.. No bruise, mark, or other abnormality in physical appearance was found on examination. The parents were recorded as showing great anguish and anger.

Subsequent history from her mother was that Laura and had been in a bad mood, but did not appear to be seriously ill. She was last fed at 7 am. She took her to a gymnasium and to visit her father at his place of work. Laura fell asleep while travelling home in the car, and she put the sleeping child to bed in her own room at 11am, placing her on her side on top of the bed and then covering her with a woollen rug. At about 11:30am Kathleen heard the child coughing. At about 11:35am she checked on Laura and found her lying on her back and pale. She carried her to the breakfast bar in the kitchen where she rang for an ambulance and commenced cardio-pulmonary resuscitation.

Examination of the bedroom showed some small dark stains on the pillow of her bed. A screening test was positive for blood.

Kathy Folbigg gave the following account in her statement of 23 July 1999 (summarized); It was a difficult decision to have Laura. The birth itself was straightforward and she was born in good condition. They stayed in hospital for five days and she breast fed for about the first week. At about 10 days of age she spent a night in hospital undergoing sleep studies. They were given a more advanced monitor and she stayed in the bedroom with them for the first couple of months. There were many false alarms. After about three months they moved her into her own room, initially in a basinet, and later in a cot. Kathy describes her as a good sleeper and a really good baby. At about six months of age they began to use the monitor less during the day. Kathy says this was probably more her decision then Craig's because it was she who responded to the alarms. When she had left Laura previously with friends, she did not give them the monitor.

They had a huge party for her first birthday. They were advised to discontinue using the monitor.

About 10 days before Laura's death Kathy wrote Craig a letter explaining she was thinking of leaving them. In interview she agreed to this proposition. She also agreed that there was some kind of argument between her and Craig on the morning before Laura died.

On the day of her death her mother took it to the gym as usual. She fell asleep in the car and her mother carried her into the house and put her to bed. She went out to check on her dogs, and around 11am about 15 to 20 minutes later she checked on Laura and found her flat on her back. She went to put her back on her side and there was no reaction. She may have been a little pale and was cool but not cold. She grabbed her and ran out into the kitchen to the breakfast bar and began resuscitation. She dialled the ambulance and continued until it arrived. She phoned Craig at work but spoke to someone else, so that when Craig finally came home she was angry with him for having taken so long.

During this interview Kathy Folbigg says that she had stopped writing diaries and had thrown them away.

Post-mortem Examination

A post-mortem examination was carried out at 9pm on 1st March 1999 by order of the Coroner.

External examination

The body was that of a female infant consistent with the stated age of one year and eight months. The body weighed 11.52 kilograms, crown heel length 80.5 centimetres, head circumference 47 centimetres, Chest circumference 49 centimetres, abdominal circumference 44.5 centimetres, and foot length 12 centimetres. The pupils were equal and measured approximately 3 mm. There was a small amount of clear fluid present at the nostrils. The lips were slightly dried and cyanosed. The frenulum was intact. The external genitalia were normal. Rigor mortis was fully developed. There was posterior lividity, and lividity was also

present on the left side of the face where it was pronounced on the left cheek and left forehead.

An ovoid 5 by 3 millimetre brown bruise was present just medial to the left kneecap. An ovoid 12 by 10 millimetre brown bruise was noted on the right anterior lower leg.

There were marks of medical intervention.

Internal examination

The scalp and skull were normal. The brain weighed 1154 grams and was saved for further examination. No retinal haemorrhages were present. The neck and cervical spine were normal.

The heart weighed 62 grams and was normal apart from an eight millimetre diameter area of haemorrhage on the posterior surface of the left atrium.

The airways were normal. The left lung weighed 122 grams and the right lung or 114 grams. Both showed focal haemorrhage and collapse on their cut surfaces.

The thymus weighed 28 grams and was normal apart from petechial haemorrhages on the anterior aspect of the suprasternal thymus.

The stomach contained a small quantity of milky-type fluid mixed with vegetable-type material. The intestines were otherwise normal.

The liver weighed 430 grams and was normal.

Each kidney weighed 36 grams. The bladder contained 10 mls. of clear urine.

The spleen weighed 46 grams. Lymph nodes in the mesentery were mildly enlarged. The bone-marrow and ribs were normal.

The pituitary gland, the thyroid gland, and both adrenal glands were normal, the latter weighing two grams each.

No abnormality was found in the musculoskeletal system.

The body was re-examined on 2nd March and no additional significant findings were present. A facial dissection was carried out on 3rd March 1999 and no bruises or any other injuries were detected.

Microscopic examination

The heart showed a moderately dense infiltrate of lymphocytes with degenerate muscle cells interpreted as evidence of a viral myocarditis.

The spleen showed a markedly increased number of lymphocytes in the red pulp.

The lungs showed an increased number of lymphocytes within the interstitium and in some alveolar spaces. There were widespread areas of haemorrhage with numerous red blood cells within alveolar spaces which also contained oedema fluid, foamy macrophages, and fibrin.

Focal cortical haemorrhages were present in the thymus.

Microscope slides of the liver, kidney, stomach, oesophagus, adrenal, salivary gland, small and large intestine, thyroid, bone-marrow, pancreas, diaphragm, skeletal muscle, and ovary were all essentially normal.

Neuropathology report

The brain weighed 1307 grams after fixation. The brain stem and cerebellum weighed 11.8 per cent of the total brain weight. No macroscopic abnormality was seen in the brain or 20 cms segment of spinal cord examined.

No microscopic abnormality was seen in the multiple sections. Development was appropriate for age.

Additional post-mortem studies

Cerebrospinal fluid: no growth

Rectal swab: normal bowel flora

Lung: profuse post-mortem contaminants.

Spleen: mixed growth.

Toxicological analysis showed no alcohol in blood, and no chlorpheniramine in blood. Screening tests for various other common drugs were negative.

Craig was overcome with grief. Kathy went back to the gymnasium the following Monday, 8th March. Craig went back to work on Wednesday 10th March. Craig wanted to talk about Laura, but Kathy did not. Kathy moved out of their house a few weeks later and moved into a flat.

Sometime after this Craig found an A5 size Diary of Kathy's at his house in a box. He found some entries which concerned him which he took to the police.

He went to his wife's flat to speak to her, but she did not return to him. He was deeply hurt and contacted detective Ryan and told him some things which she says were not true. He told him that on the night Sarah died, Kathy and Sarah were not in the room when he woke up at

1am. He also said that after Laura died he heard Kathy talking to herself using a different accent. He said that he told Detective Ryan these things out of spite and because he was hurt that Kathy wouldn't return to him.

Examination of Microscope slides

I have been supplied with 14 stained microscope slides of tissue taken from the body of **Caleb Folbigg** (99/11208)

Lung. Sections show moderate to marked pulmonary haemorrhage which is focally confluent. There is alveolar oedema, pleural haemorrhage and occasional intra-alveolar squames and macrophages. The lung is normally developed and shows no evidence of infection or aspiration. There is possible interstitial emphysema attributable to resuscitation.

Sections of lung stained by Perls' method for ferric iron show dark blue pleural and interstitial macrophages. In addition there are moderate numbers of pale blue intra-alveolar macrophages, some with haemosiderin granules.

Heart. Normal

Kidney. Mature kidney with occasional sclerotic glomeruli. There is focal extra-medullary haemopoiesis. The renal pelvis and vessels are normal, as is perirenal brown fat.

Liver. Normal architecture and portal tracts. Hepatocytes appear pale, but their cytoplasm is granular rather than vacuolated. Residual extramedullary haemopioesis is noted.

Adrenal. Foci of calcification are noted in the fetal cortex and medulla.

Thymus. There are no petechial haemorrhages, and no acute stress reaction.

Spleen. Normal with normal lymphoid tissue.

Mesentery. Normal with normal immature lymph nodes.

I have been supplied with 21 slides labelled 99/11207 prepared from tissue taken from the body of **Patrick Folbigg**.

These comprise 19 sections of brain and spinal cord, and a single section each of striated muscle and anterior pituitary.

The brain shows extensive old laminar cortical necrosis with loss of grey matter at the depths of sulci, cysts, gliosis, gitter cells, and focal mineralization. The cerebellum shows probably artefactual separation of the cortex, and reduced white matter.

I have been supplied with 41 slides labelled 93/1673 prepared from tissues taken from the body of **Sarah Folbigg**.

Lung. There is mild focal intra-alveolar haemorrhage and oedema. Aspirated food does not reach alveolar spaces. There is no significant haemorrhage.

There is vascular congestion of the uvula, larynx and epiglottis. Normal tongue. Salivary gland contains groups of lymphocytes.

Trachea. There is a small number of subepithelial and intra-epithelial lymphocytes.

Heart. Normal

Liver. Nucleated cells present in sinusoids. There are occasional lipid droplets in hepatocytes, but no marked fatty change in haematoxylin and eosin stained sections.

Kidney. Congested Medulla.

Lymph node and spleen. Normal B and T cell areas.

Thymus. Focal parenchymal haemorrhage. No acute stress reaction.

Adrenal, thyroid, pancreas: normal.

Colon. Autolysed, but otherwise normal.

Diaphragm. Normal.

Brain and spinal cord, normal.

I have been supplied with 53 microscope slides ;labelled 99/9322 prepared from tissue taken from the body of **Laura Folbigg.**

Myocardium. Heart muscle shows a patchy but widespread interstitial mononuclear infiltrate in the right and left ventricles. There is no definite myocyte necrosis.

Lung. There is focal intra-alveolar haemorrhage. Post-mortem proliferation of bacteria is noted in airways. There is aspiration of stomach contents with post-mortem acid digestion. Peribronchial and subepithelial lymphocytes are increased, and there is a possible interstitial infiltrate. Focal alveolar hyperinflation is attributable to resuscitation.

Trachea. A few subepithelial lymphocytes only.

Kidney. Normal.

Liver. Mononuclear cells in portal tracts and in groups in sinusoids. No fatty change in haematoxylin and eosin stained section.

Spleen. Prominent germinal centres with mononuclear cells in the sinusoids (reactive).

Thymus. Focal parenchymal haemorrhage. No starry sky reaction.

Pancreas. Normal endocrine and exocrine tissue.

Oesophago-gastric junction. No evidence of reflux.

Adrenal. Normal

Small bowel and colon. Autolysed, but otherwise normal.

Salivary gland. Occasional lymphoid aggregates.

Costochondral junction. Normal bone marrow and growth plate.

Striated muscle and diaphragm. Normal.
Middle ear. No acute inflammation.

Brain and spinal cord. There is no significant pathology. Occasional perivascular haemorrhages are noted in the brain, and one or two possible lymphocytes are seen in the leptomeninges.

Additional studies

Attempts to further characterize the lymphoid cells in the myocardium of Laura Folbigg, and to stain for common viruses by immunohistochemistry failed for technical reasons.

Perls' Prussian blue stains of the lungs of Sarah and Laura Folbigg showed no excess of intra-alveolar siderophages.

COMMENT

I will first consider these deaths in isolation, and then together.

Caleb Folbigg

1. Caleb Folbigg was the result of a normal pregnancy complicated only by an intercurrent maternal infection, possibly chicken pox. Delivery was by Kielland's forceps, there was meconium staining of the liquor, and he suffered transient tachypnoea. I am unable to relate his death to any of these events.
2. He was thought to have a floppy larynx, a generally benign and self-limiting condition. While "near miss" SIDS events have been attributed to this condition in the

medical literature, it is not a recognised cause of infant death, and there is nothing to suggest that the condition was responsible for death in this case.

3. The post-mortem examination showed a normally grown baby with no natural disease to account for death.
4. There were no marks of violence, and no features to suggest suffocation such as facial petechial haemorrhages.
5. The mother's observation of blood stained froth around the nose and mouth is a common finding in sudden infant deaths and accidental or deliberate suffocation.
6. Abundant milk in the stomach was consistent with the mother's account that she had recently fed him.
7. Petechial haemorrhages were not described on the thymus and other thoracic organs.
8. The lungs showed quite marked intra-alveolar haemorrhage.
9. The presence of haemosiderin as shown by Perls' stain is a very unusual finding in infant deaths, and is so unusual that in my view it excludes the death from the SIDS category.
10. Haemosiderin in the alveolar spaces indicates previous haemorrhage into the lungs. There are many causes which would generally be obvious from the medical history and post-mortem examination. One recently described cause is repeated imposed upper airway obstruction, but in these cases there have been previous events which in retrospect could be attributed to deliberate suffocation. Such events were not noted in Caleb's case.
11. The mother's diary entry is, on the face of it extremely worrying.

Faced with a similar case today, I would not give the cause of death as SIDS because of the finding of haemosiderin in the lungs. That and the diary entry would lead me to suspect suffocation, and I would recommend a full police investigation. (This is in no way critical of those involved at the time who did not have these two pieces of information).

Patrick Folbigg

1. Patrick Folbigg was the result of a normal but unplanned pregnancy complicated by a possible maternal urinary tract infection. The delivery was normal and he was of normal birth weight.
2. Sleep studies and an electrocardiogram were normal. A barium study showed no gastro-oesophageal reflux (there was a suggestion of in-coordination of swallowing).
3. Patrick was well while his father remained at home spending all his time with his family.
4. At about 4 months of age Patrick was found apparently lifeless by his mother in the early hours of the morning. He recovered rapidly with oxygen, and there appeared to have been an acute asphyxiating event.
5. Following this episode he developed signs of severe brain damage including epilepsy for which no other cause was found.
6. Extensive laboratory tests ruled out inherited metabolic disorders.
7. Diary entries found by Craig Folbigg indicated that Kathy was under considerable strain.

8. At the age of about 9 months he was found lifeless by his mother who phoned her husband and the paediatric neurologist.
9. Permission for post-mortem examination was given by the baby's father.
10. There were no external signs of injury or asphyxia.
11. Detailed examination of the brain showed changes consistent with a previous episode of cardio-respiratory arrest, and no evidence of a metabolic or infectious disorder.
12. Levels of anti-convulsants were in the therapeutic range.

Patrick's initial collapse was never explained. Such "near-miss" events resulting in brain damage are a cause for concern because the window of opportunity to find a child in extremis and affect resuscitation is very short, probably a matter of only a few minutes. This raises the question that the person who finds the baby may have been present when the collapse occurred, and may have been its cause. Such "acute life threatening events" are not part of the usual natural history of SIDS.

Taking this case in isolation I would have given the cause of death as "not ascertained", ascribing it to brain damage following an unexplained collapse, also noting that the child's mother found him both on that occasion and when he subsequently died. Her action in calling her husband and neurologist before the ambulance is unusual.

Sarah Folbigg

1. Sarah Folbigg was delivered normally after a planned pregnancy complicated by an admission for bleeding in the first trimester.
2. Her development was normal. She had a sleep study which was also judged to be normal. A test for MCAD deficiency (an inherited chemical disorder that can cause sudden death in infancy) was normal.
3. Craig noted that Kathy became easily irritated and stressed.
4. Although they had been given an apnoea mattress, this was not in use at the time of Sarah's death.
5. On the evening before Sarah's death Kathy had apparently been angry with her.
6. Sarah was found lifeless by her mother in the early hours of the morning.
7. Post-mortem examination showed relatively trivial scratches, but no other marks of significant trauma.
8. Internal examination showed congestion of the uvula and thymic, pleural and epicardial petechial haemorrhages.
9. The cause of death was given as sudden infant death syndrome.
10. Subsequent histological, microbiological, biochemical and toxicological examination failed to give a cause for her death.

Taken in isolation, the death of Sarah resembles many ascribed to the "sudden infant death syndrome". The post-mortem findings were consistent with that diagnosis. However, at 10 months of age she was older than most SIDS, the majority having occurred by 6 months of age. That alone is reason for closely scrutinizing the circumstances. It is of concern that Craig's account indicates considerable tension in Kathy on the evening that Sarah died.

Nevertheless, in these circumstances and after careful investigation I would probably give the cause of death in isolation as SIDS, but with misgivings.

Laura Folbigg

1. Laura Folbigg was born after a normal pregnancy and delivery.
2. Laura also underwent sleep studies on three occasions which were normal without obstructive sleep apnoea at any time.
3. Investigations for inherited biochemical disorders were negative.
4. Craig was concerned that Kathy was not using the apnoea monitor diligently.
5. At 14 months Laura suffered a burn to her left forearm and palm. I have been told that this was a result of a corroborated witnessed incident.
6. In February 1999 Kathy considered leaving Craig.
7. On March 1st they had an argument. Laura was found lifeless by her mother later that morning.
8. A post-mortem examination showed no marks of injury other than brown bruises on the legs. These are common findings in toddlers and of little significance.
9. Detailed examination of the soft tissues of the face showed no bruising.
10. Microscopic examination of the heart showed the heart muscle to be infiltrated by inflammatory cells, an appearance attributed to viral myocarditis.
11. Microbiological and toxicological studies were negative.

Inflammation of the heart may cause sudden death in two ways. Firstly, extensive damage to the muscle of the heart can result in failure of the heart muscle as a pump shown at post-mortem by dilatation of the heart and accumulation of fluid in the lungs and elsewhere. This was not found in Laura. Secondly, it is generally agreed that even quite minor inflammation can result in abnormal heart rhythms and sudden death. In Laura's case the infiltrate in the heart was quite extensive, and most pathologists would have accepted it as the cause of death, although I was unable to convince myself of actual damage to heart muscle cells.

However, it is recognised that an inflammatory infiltrate in the heart muscle is also quite commonly found in those who die of other causes, for example in road traffic accidents. It has been described as an incidental finding in suffocation. An inflammatory infiltrate in the heart must therefore be quite common in the general population, and probably accompanies some common childhood illnesses. The finding of an inflammatory infiltrate in the heart does not necessarily mean it was responsible for death.

Nevertheless, taken in isolation I would have ascribed this death to myocarditis recognising that although the infiltrate was quite extensive, I could not see actual damage to heart muscle.

The four deaths together

Sudden death of four infants in the same family who were previously well (in the case of Patrick before his initial collapse) due to natural disease is unprecedented in my experience, and I know of no substantiated examples in the literature. Nevertheless, it is important to explore this possibility.

All four children were seen to be well shortly before they were found lifeless. With the exception of Laura, no disease process was found to account for their collapse.

In the case of Patrick, Sarah and Laura sleep studies and tests for inherited biochemical disorders were negative. No electrocardigraph abnormality was noted in any of these children in life. It is therefore very unlikely that these children died as a result of an inherited metabolic, cardiac, or airway disorder. In particular, the suggestion that there may have been familial obstructive sleep apnoea was not substantiated by the sleep studies.

There are as many theories of SIDS as there are cot death researchers, but I know of no substantiated cause of sudden and unexpected death of previously well infants that is applicable to these cases.

If death was not natural, then it may have been accidental. I could identify no recognised hazard common to these deaths. I note that the family moved on several occasions ruling out some unrecognised environmental toxin such as carbon monoxide or toxic fungi.

There were no definite pathologic features of deliberate injury. If these children died as a result of the action of a carer, then the most likely mechanisms are shaking or suffocation. I note that a suggestion was made on the basis of radiological findings that Patrick Folbigg might have been shaken, but there was no evidence from pathology that this was the case for the other children.

Suffocation in young children is often unaccompanied by any external signs such as pressure marks or facial petechial haemorrhages, and there are no diagnostic internal findings. Petechial haemorrhages beneath the capsule of the thymus, the pleura and the pericardium are commonly found but are also described in SIDS. The deaths of Caleb, Patrick, and Sarah were entirely compatible with suffocation as the cause.

In many cases, a diagnosis of suffocation can only be made from the history and circumstances.

1. All of these children were previously well.
2. All were found by their mother.
3. There was a short interval between them being seen alive and being found lifeless.
4. Haemosiderin was found in the lungs of Caleb Folbigg, although I place no great weight on this in the absence of previous collapses.
5. Patrick's initial collapse and rapid response to oxygen is consistent with near suffocation.
6. Kathy Folbigg appears to have been under particular stress on the day that Caleb, Patrick, Sarah and Laura died.
7. Most babies who die as cot deaths are found lifeless when the parents get up in the morning. Caleb was found at 2.45 am., Patrick at 4.30 am., Sarah at 1.30 am. and Laura at about midday.

8. In the case of Laura her father was worried that Kathy Folbigg was not using her monitor. Monitors were not in use at the time of the death of any of these children.
9. Kathy Folbigg's actions in calling her husband and neurologist before an ambulance is unusual.
10. I am not professionally qualified to comment on the content of the diary kept by Kathy Folbigg other than to say that it appears worrying, and requires expert review.

CONCLUSION

The sudden and unexpected death of three children in the same family without evidence of a natural cause is extraordinary. I am unable to rule out that Caleb, Patrick, Sarah, and possibily Laura Folbigg were suffocated by the person who found them lifeless, and I believe that it is probable that this was the case.

P.J. Berry

November 2000

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December 1, 2001

Det. Bernie Ryan
Singleton Police Station - Criminal Investigation Branch
22 Hunter Street
Singleton 2330
NSW AUSTRALIA

Re: Kathleen Folbigg

Dear Det. Ryan,

This correspondence is in response to your request for a summary of my opinions regarding the deaths of Caleb Folbigg on February 20, 1989, Patrick Folbigg on February 13, 1991, Sarah Folbigg on October 30, 1993 and Laura Folbigg on March 1, 1999. In preparation of this report I have reviewed the following materials:

Materials

Medical records of Kathleen Folbigg	Medical records of Laura Folbigg
Health Insurance Commision Records	Statement of Dr. Wilcken (1.14.00)
Expert Certificate by Dr. Innis	Newborn Screening Blood Results
Medical records supplied by Dr. Marley	Statement of Christopher Seton
Medical records supplied by Dr. Cash	Handwritten sleep notes by Kathleen Folbigg
Medical records of Caleb Folbigg	Report by Dr. Seton to Det. Ryan
Statement from Dr. Bridget Wilcken	Referral by Dr. Seton to Dr. King
Newborn Screening Blood results	Letter by Mr. Folbigg to Dr. Seton
Newcastle Western Suburbs Hospital records	Report by Dr. Seton to Mr. Folbigg
Coroners Brief	Newborn discharge summary
·mbulance Records	Report by Dr. Seton to Dr. King
ical records of Patrick Folbigg (Vol I & II)	Corometrics monitor supply record
Statement of Dr. Bridget Wilcken	Urine medabolic profile
Newborn screening blood results	Sleep study report (10.7.97)
Medical records from Newcastle Western Suburbs Hosp	Royal Alexandria Hospital for Children Medical History
Statement from Dr. Wilkinson	Sleep study report by Seton to Sanders
Medical Certificatee of cause of death	Letter by Craig Folbigg to Margaret Tanner
Cause of death certificate (hand written)	Report by Seton to Craig Folbigg
History, examination and progress notes	Report by Seton to Dr. Sanders
Report by Dr. Wilkinson to Marley	Patient alarm traces (Corometric monitor print outs)
Report by Dr. Wilkinson to Dr. Morris	Statement of Dr. Innis
Adelaide Children's Hospital Pathology Report	Information sheet
Mater Hospital Pathology reports	Progress Notes
Report by Dr. Challinor to Dr. Wilkinson	Singleton Hospital Records
Biochemistry reports	Ambulance report
Report by Dr. Wilkinson to Dr. Thomas	Fairholme Surgery Records
Physiotherapy report	Statement of Dr. Cash
Autopsy report	Newborn discharge summary
Report by Dr. Wilkinson to Dr. Bale	Report by Dr. Seton to Dr. King
HAPS reports	Sleep study reports 10.7.97 and 2.3.98
Histopathology Dept Report	Report by Dr. Seton to Craigh Folbigg
Report by Dr. Wilkinson to Folbiggs	Report by Dr. Seton to Dr. Sanders
Report by Dr. Colley to Dr. Wilkinson	Ambulance records

Records of Patrick Folbigg continued...	
Report by Dr. Marley to Dr. Holland	Statement from Craig Folbigg
Dr. Colley to Dr. Wilcken	Transcript of Kathleen Folbigg interview
Dr. Wilckinson Dr. Colley	Psychological report by Roz Garbutt
Dr. Edwards to Dr. Hardacre	Diary Entry (2.19.89)
Newcastle Mater Hospital Records June 14, 1990	Diary Entry (6.4.96 - 6.5.97)
Newcastle Mater Hospital Records October 18, 1990	Handwritten letters by Kathleen Folbigg
Newcastle Mater Hospital Records November 4, 1990	Letters by Craig Folbigg
Newcastle Mater Hospital Records November 14, 1990	Interview of Thomas Britton (12.9.68)
Newcastle Mater Hospital Records December 22, 1990	Mother's Day Card
Statement by Dr. Marley	May 1992 Gibbs personal diary entry
Pediatric Summary	Diary entry July 1999
Ambulance Records	Diary entry (6.6.97 - 4.10.98)
Beresfield Crematorium records	Diary entry (1.1.99 - 1.3.99)
Medical records of Sarah Folbigg	Diary entry (6.19.99)
Statement by Dr. Wilcken	Telephone interception transcripts
Newborn Screening Blood Results	Listening device transcripts
John Hunter Hosptial Records	
Statement by Dr. Marley	26 - Autopsy photos (Laura)
Pediatric discharge	53 - Microscopic Slides (Laura)
Perinatal database	40 - Microscopic Slides (Sarah)
Reports: Dr. Hardacre to Dr. Marly	21 - Microscopic Slides (Patrick)
Buckner to Holland	14 - Microscopic Slides (Caleb)
lacre to Marley	
dacre to Holland	
Pickford to Marley	
Edwards to Hardacre	
Handwritten notes	
Ambulance Records	
Coroners Brief	

CALEB GIBSON FOLBIGG

Findings

Caleb Folbigg was the product of a full-term pregnancy and his 21-year old married mother, Kathleen Folbigg, received adequate prenatal care. Caleb was delivered vaginally with forceps assistance following an essentially uncomplicated labor on February 1, 1989. Birth weight 3280 grams. Birth length 42.2 cm. Apgar scores 9 and 9 at 1 and 5 minutes. He demonstrated slight jaundice. His newborn course was complicated by a brief bout with transient tachypnea (mild respiratory distress) that resolved without difficulty. A chest x-ray was performed and was interpreted as clear. Caleb was discharged to home on February 5, 1989 with his mother.

Caleb slept in a white cane bassinet in the sun room, a room adjacent to the parents bedroom. It was reported that he always slept in this bassinet. He was characterized as a quiet baby who slept fairly well. He was formula fed.

pediatrician, Dr. BJ Springthorpe, at well child evaluation on February 17, 1989 (at 2-weeks of age), noted inspiratory stridor n the child was placed supine or agitated. The problem was characterized as mild laryngomalacia and no further follow-up was recommended.

The father, Craig Folbigg last saw Caleb asleep on his side or his back in his bassinet between 2200 and 2230 hours. In the early morning of February 20, 1989, Kathleen fed Caleb (~0100 hours). Kathleen checked on the baby again at 0250 hours and found him "cold" with bloody froth in his nose and mouth.

Paramedics arrived at the Folbigg residence at ~0259 hours. They found the infant pale, cyanotic, not breathing, unconscious, pulseless and warm to the touch, in apparent cardiac arrest. It is unclear whether paramedics went into a bedroom and gathered the child or whether Kathleen brought the infant to the paramedics. The child was laid down on the loungeroom floor in the supine position. Airway was cleared with oral suction and airway was placed. Mouth to mouth resuscitation was commenced, followed by intermittent positive pressure ventilation. Caleb remained asystole. At ~0338 hours Caleb was transported to Newcastle Mater Hospital. At 0400 hours on February 20, 1989 Dr. Sandy Chapman, Newcastle Mater Hospital, pronounced Caleb to be dead.

Autopsy was performed on February 20, 1989 by Dr. R. Cummings. Findings include:

- A well-developed, well-nourished male infant, weight 3970 grams.
- Lungs: are congested and in places show incomplete aeration, in other sections their alveoli contain extravasated red blood cells and a small amount of eosinophillic exudate.
- Stomach contained a large quantity of curdled milk
- No mention of thymic or other intrathoracic petechiae
- Routine toxicological analysis was negative

- Cause of Death: SIDS

Dr. Bridget Widcken performed a complete biochemical profile on blood samples from Caleb. The results were entirely normal. There is no positive evidence indicating an inherited metabolic disorder affecting amino acid, organic acid, or fatty acid oxidation pathways. These normal results tend to exclude certain disorders of amino acid metabolism (phenylketonuria, the tyrosinaemias, maple syrup urine disease, homocystinuria due to cystathionine synthase deficiency) and a large number of organic acidurias and fatty acid oxidation defects, including methylmalonic acidaemia, propionic acidaemia, medium chain acyl CoA dehydrogenase deficiency, and several other disorders which are extremely rare. The laboratory also conducted a DNA test for the common mutation seen in medium chain acyl CoA dehydrogenase deficiency, which is present in 98% of known cases of this disorder. This mutation was not present in any of the children's samples.

My review of the autopsy includes the following in addition to the findings described by Dr. Cummings:

- Extensive pulmonary hemorrhage (significantly greater that 10%)
- Absence of intrathoracic petechiae, specifically the thymus, either grossly or microscopically
- Absence of risk factors commonly recognized in the epidemiology of Sudden Infant Death Syndrome. These include young age, prone sleeping position, inadequate prenatal care, low birth weight/premature birth, maternal smoking, co-sleeping, ethnicity, low socioeconomic status, young maternal age, multiple births.

Opinions

In forming my conclusions I have utilized all the materials made available to me including all the medical history and records, the autopsy reports and materials, the police investigative documents, the interview transcripts from Kathleen Folbigg as well as witness statements, diary entries, and listening device materials.

The materials and investigative information provided in this case are of excellent quality and are sufficient for me to render an opinion to a reasonable degree of medical certainty.

It is my opinion to a reasonable degree of medical certainty that Caleb Folbigg did not die of the condition known as Sudden Infant Death Syndrome. It is also my opinion that Caleb's death is most consistent with death by suffocation. It is my opinion that Caleb Folbigg was the victim of probable homicidal assault that resulted in his suffocation.

I have participated in the investigation of both accidental and homicidal suffocation in children over the course of my 20 years as a practicing pediatric forensic pathologist. Unfortunately multiple infant homicides within one family are now well documented in the literature and in forensic experience. Typically the perpetrator does not confess to the crimes but in many cases such as this the facts of the case make the diagnosis. Important facts in this case that lead to the conclusion of homicidal suffocation include:

- The autopsy fails to identify any known natural disease or disease process that could explain the sudden death of Caleb. Caleb was growing and developing normally for his age and circumstance.
- The findings at autopsy that are consistent with the determination of death by suffocation are the absence of thymic petechiae, the presence of extensive pulmonary hemorrhage and the description of the mom that blood was present on her child's face.
- Caleb was in the care of his mother at the time of his death and she was the last person to see him alive.
- All of the Folbigg infants were all in the care of their mother at the time of their death and she was the last person to see each of them alive.
- None of the deaths in the Folbigg case can be attributed to SIDS. It is well recognized that the SIDS process is not a hereditary problem and the statistical probability that 4 children in one sibship could die from SIDS would be infinitesimally small. If you calculate the risk of this event occurring 4 times in one family, using routine statistical probability for a random event occurring 4 times in one family (with an occurrence of <1/1000 live births) it would be less than 1 in one trillion.
- The diagnosis of SIDS requires that following a complete investigation and autopsy no other cause of death is identified. After the deaths of subsequent Folbigg children, suddenly and unexpectedly, without explanation, Caleb's death cannot be considered sudden infant death syndrome and should be investigated as a homicide.
- Absence of risk factors commonly recognized in the epidemiology of Sudden Infant Death Syndrome. These include young age, prone sleeping position, inadequate prenatal care, low birth weight/premature birth, maternal smoking, co-sleeping, ethnicity, low socioeconomic status, young maternal age, multiple births. There is no history of infant apnea or significant breathing problems; no evidence of hyperthermia; no recent history of illness.

In my opinion the cause of death and manner of death should be listed as follows:

- Cause of Death: Undetermined
- Manner of Death: Undetermined

SIDS, also called crib or cot death, is the sudden death of an infant under 1-year of age that remains unexplained after thorough case investigation, including the performance of a complete autopsy, examination of the death scene, and a review of the clinical history. The SIDS diagnosis should not be applied unless all of the following are true:

- A complete autopsy is done, including the cranium and cranial contents, and autopsy findings are compatible with SIDS
- There is no gross or microsopic evidence of trauma of significant disease process
- There is no evidence of trauma on a skeletal survey
- Other causes of death are adequately ruled out and
- There is no evidence of current alcohol, drug, or toxic exposure; and thorough death scene investigation and review of the clinical history are negative

The SIDS diagnosis has been applied variably during the latter half of this century. What we know now is that some children who were diagnosed as SIDS in the past were actually murdered and families where there were multiple infant deaths attributed to SIDS were actually the victims of serial killings. The current practice is to carefully apply the diagnosis of SIDS only in cases that fulfill the current diagnostic criteria. It is also now common practice for the medical examiner to reconsider the deaths of children originally thought to have died of SIDS when subsequent infants die suddenly, unexpectedly, and without explanation. This is certainly the circumstance that applies in Caleb's case. At this time it would be more appropriate to consider the cause of death to be undetermined and the manner of death undetermined. There have been no cases of metabolic disorders reported where multiple children in one sibship have died suddenly and without explanation following complete medical evaluations and autopsy with essentially negative findings. Comprehensive medical testing and genetic consultation have failed to reveal any abnormalities in the Folbigg family.

As a routine practice, I continue to review the world literature in my practice. I have included some references in my SIDS research that I consider relevant to this case, but I did not base my opinions on them.

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PATRICK ALLAN FOLBIGG

Findings

Patrick Folbigg was the product of a full-term pregnancy and his mother, Kathleen Folbigg, received adequate prenatal care. The pregnancy was uncomplicated. He was delivered vaginally following an uncomplicated labor at 1106 hours on June 3, 1990 at Newcastle Western Suburbs Hospital. Birth weight 3410 grams. Birth length 48.3 cm. Apgars 7 and 8 at 1 and 5 minutes. Patrick's newborn course was uncomplicated and he was discharged home on June 8, 1990 with his mother.

Patrick slept in a cot in a bedroom separate from his parents, off the dining room. Craig Follbigg spent full-time with the family for Patrick's first 3 months at which time Craig returned to work.

His pediatricians were Richard Henry and Barry Springrthorpe. He underwent a sleep study 1-week after his discharge from the hospital on June 15, 1990. The examinations showed no GE reflux and the sleep study was normal.

On October 18, 1990 Kathleen stated that she heard Patrick coughing at ~0300 hours. At 0430 hours she was up and heard him "gasping" in his room and found him cyanotic, lifeless, and making minimal respiratory effort. Craig woke to the sound of Kathy screaming. He ran into Patrick's room and found Kathy standing over his bed. Caleb was supine with his covers apparently "kicked off." Craig then began CPR. Emergency responders arrived at the Folbigg residence at ~0500 hours and provided oxygen and respiratory support. He improved spontaneously and was admitted to the hospital through the emergency department. Upon presentation at the hospital, Patrick was lethargic, cyanotic, responding only to painful stimuli. At the emergency department, Patrick received oxygen and he became more alert and regained color. Dr. Dezordi did not see any signs of upper airway obstruction or of aspiration. There were no signs suggestive of serious illness nor evidence of trauma or injury. EEG report interpreted as normal. Chest x-ray did not demonstrate signs of aspiration.

ing hospitalization the child developed right-sided seizures that proved over time to be difficult to control. CT scan (October 24, 1990) revealed bilateral abnormalities of the brain specifically in the occipital lobes. The child was diagnosed with severe visual deficits. The attending physicians evaluated a multitude of possible etiologies none of which identified a cause for Patrick's sudden episode, but eventually concluded that Patrick suffered from an encephalopathic disorder, of unknown cause. The findings are consistent with a severe hypoxic event. Despite these physical setbacks, Patrick continued to show satisfactory growth and development. Patrick was discharged home on October 29, 1990.

On November 4, 1990 Patrick was again admitted to Newcastle Mater Hospital presenting with fever, vomiting, conjunctivitis, cough, rash, and seizure. CT scan (November 5) reveals same abnormalities, worsening. The cause of this "loss of brain substance" was not clear. EEG report (November 5) reveals asymmetry between the right and left sides of the brain both awake and asleep. Some seizure activity on the left side of the brain was noted for a short period of time. There was excessive electrical slowing on the right side compared to the left. He was discharged on November 10, 1990.

Dr. Merl DeSilva, Royal Hobart Hospital, reviewed CT scans dated October 23 and November 5, 1990. It is his opinion that the findings were compatible with brain injury from shaking of an infant, in the sub-acute phase. The injury may have occurred some days prior to the scans. Dr. DeSilva felt the injury was not a result of a direct blow to the head.

On November 14, 1990 Patrick was again admitted to Newcastle Mater Hospital presenting with cough, rhinorrhea, stridor, vomiting, and seizures. He was discharged on November 22, 1990 at 1000 hours only to be readmitted at 2100 hours in occulogyric crisis. He was discharged November 23, 1990.

November 21, 1990 Patrick was seen by Dr. C.J. Challinor, ophthalmic surgeon. Dr. Challinor found that Patrick demonstrated an ility to fix appropriately. On examination he did not fix and follow. His ocular movements seem full but he had continual ocular movements that were not nystogmoid or roving but consisted in of changes of conjugate gaze direction in a random manner. Dr. Challinor's impression was that of cortical visual impairment.

On the morning of February 13, 1991 Kathleen put Patrick down for a nap at ~0730 hours. She reportedly found him lifeless at 0930-1000 hours. After calling for an ambulance, Kathy telephoned Craig at work screaming "it's happened again." Craig immediately left work for home. Emergency responders arrived at ~1020 hours. Paramedics found the child in full cardiopulmonary arrest, his skin warm to the touch, pale and cyanotic.

Dr. Chris Walker examined Patrick upon arrival at Newcastle Mater Hospital. The child was not breathing, was receiving ventilator support with oxygen, resuscitation continued although Patrick remained asystole. Patrick developed a broad agonal rhythm after adrenalin was administered intravenously. No cardiac output was noted. Resuscitation ceased after 20 minutes. Dr. Chris Walker pronounced Patrick dead at 1040 hours February 13, 1991.

Dr. Wilkinson noticed petechial hemorrhages that were interpreted as agonal. No note in the autopsy report is present.

Autopsy was performed on February 13, 1991 at 1230 hours by Dr. J. Bishop and Dr. G. Singh-Khaira. Autopsy findings include:

- A well-developed, well-nourished male infant, weight 8.57 kg.
- Lungs show posterior dependant congestion
- No mention of petechial hemorrhages, specifically in the thymus
- Thymus described as large
- Routine and special analyses were negative

- Neuropathology examination revealed laminar cortical necrosis of the brain with cystic degeneration in the visual cortex. This is most consistent with old infarcts occurring at the time of his arrest at age 5 months. No evidence of congenital abnormalities was present.
- Cause of death: SIDS

My review of the autopsy does not include any additional comments with the exception that in my opinion, the cause of death is inconsistent with sudden infant death syndrome. In my opinion the cause of death is consistent with suffocation, manner homicide.

Dr. Bridget Widcken performed a complete biochemical profile on blood samples from Patrick. The results were entirely normal. There is no positive evidence indicating an inherited metabolic disorder affecting amino acid, organic acid, or fatty acid oxidation pathways. These normal results tend to exclude certain disorders of amino acid metabolism (phenylketonuria, the tyrosinaemias, maple syrup urine disease, homocystinuria due to cystathionine synthase deficiency) and a large number of organic acidurias and fatty acid oxidation defects, including methylmalonic acidaemia, propionic acidaemia, medium chain acyl CoA dehydrogenase deficiency, and several other disorders which are extremely rare. The laboratory also conducted a DNA test for the common mutation seen in medium chain acyl CoA dehydrogenase deficiency, which is present in 98% of known cases of this disorder. This mutation was not present in any of the children's samples.

Opinions

In forming my conclusions I have utilized all the materials made available to me including all the medical history and records, the autopsy reports and materials, the police investigative documents, the interview transcripts from Kathleen Folbigg as well as witness statements, diary entries, and listening device materials.

The materials and investigative information provided in this case are of excellent quality and are sufficient for me to render an opinion to a reasonable degree of medical certainty.

Patrick's sudden, profound and irreversible brain damage is consistent with and diagnosed as a hypoxic episode. Hypoxia in this case is synonymous with asphyxia and unfortunately heralds the fatal event in retrospect. No natural disease or process has been identified to explain this event, nor was there a recurrence of an acute life threatening event observed by anyone except his mother. In my opinion, the cause of Patrick's cardio-respiratory arrest is the same process that killed him.

I have participated in the investigation of both accidental and homicidal suffocation in children over the course of my 20 years as a practicing pediatric forensic pathologist. Unfortunately multiple infant homicides within one family are now well documented in the literature and in forensic experience. Typically the perpetrator knows not confess to the crimes but in many cases such as this the facts of the case make the diagnosis. Important facts in this case that lead to the conclusion of homicidal suffocation include:

- The autopsy fails to identify any known natural disease of disease process that could explain the sudden death of Patrick. He was growing and developing normally for his age and circumstance. Despite his handicaps he was advancing well.
- The autopsy findings are consistent with death by suffocation.
- It is my opinion that Patrick's death is not consistent with a seizure or the presence of a seizure disorder.
- Patrick was in the care of his mother at the time of his death and she was the last person to see him alive.
- All of the Folbigg infants were all in the care of their mother at the time of their death and she was the last person to see each of them alive.
- Absence of risk factors commonly recognized in the epidemiology of Sudden Infant Death Syndrome. These include young age, prone sleeping position, inadequate prenatal care, low birth weight/premature birth, maternal smoking, co-sleeping, ethnicity, low socioeconomic status, young maternal age, multiple births. There is no history of infant apnea or significant breathing problems; no evidence of hyperthermia; no recent history of illness.
- None of the deaths in the Folbigg case can be attributed to SIDS. It is well recognized that the SIDS process is not a hereditary problem and the statistical probability that 4 children in one sibship could die from SIDS would be infinitesimally small. If you calculate the risk of this event occurring 4 times in one family, using routine statistical probability for a random event occurring 4 times in one family (with an occurrence of <1/1000 live births) it would be less than 1 in one trillion.

In my opinion the cause of death and manner of death should be listed as follows:

- Cause of Death: Suffocation
- Manner of Death: Homicide

SIDS, also called crib or cot death, is the sudden death of an infant under 1-yeare of age that remains unexplained after thorough case investigation, including the performance of a complete autopsy, examination of the death scene, and a review of the clinical history. The SIDS diagnosis should not be applied unless all of the following are true:

- A complete autopsy is done, including the cranium and cranial contents, and autopsy findings are compatible with SIDS
- There is no gross or microsopic evidence of trauma of significant disease process

- There is no evidence of trauma on a skeletal survey
- Other causes of death are adequately ruled out and
- There is no evidence of current alcohol, drug, or toxic exposure; and thorough death scene investigation and review of the clinical history are negative

The SIDS diagnosis has been applied variably during the latter half of this century. What we know now is that some children who were diagnosed as SIDS in the past were actually murdered and families where there were multiple infant deaths attributed to SIDS were actually the victims of serial killings. The current practice is to carefully apply the diagnosis of SIDS only in cases that fulfill the current diagnostic criteria. It is now common practice for the medical examiner to reconsider the deaths of children originally thought to have died of SIDS when subsequent infants die suddenly, unexpectedly, and without explanation. This is certainly the circumstance that applies in Patrick's case. At this time it would be more appropriate to consider the cause of death to be undetermined and the manner of death undetermined. There have been no cases of metabolic disorders reported where multiple children in one sibship have died suddenly and without explanation following complete medical evaluations and autopsy with essentially negative findings. Comprehensive medical testing and genetic consultation have failed to reveal any abnormalities in the Folbigg family.

As a routine practice, I continue to review the world literature in my practice. I have included some references in my SIDS research that I consider relevant to this case, but I did not base my opinions on them.

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SARAH KATHLEEN FOLBIGG

Findings

Sarah Folbigg was the product of a full term pregnancy and her mother, Kathleen Folbigg, received adequate prenatal care. She born via spontaneous vaginal delivery following essentially uncomplicated labor on October 14, 1992 at 0543 hours at John Hunter Hospital. Birth weight 3020 grams. Length 52 cm. Apgars of 9 and 10 at 1 and 5 minutes. Sarah Folbigg's newborn course was uncomplicated. The parents elected to take the baby home with apnea monitoring. She was discharged home on October 19, 1992 on a micromotion monitor.

She under went sleep studies on November 5, 1992. The oximetry was normal; there were very few sleep apneas recorded and some periodic breathing detected. Even so, Dr. Cooper judged the results to be normal.

Dr. Geoff Hardacre examined Sarah at 16-days of age. At this time, Dr. Hardacre determined that Sarah was neurologically and developmentally normal, feeding well, and thriving (already 500 grams above birthweight). He notes that Mrs. Folbigg describes Sarah as being totally different from her two boys. In the 10 days Sarah had been on the sleep monitor there had been 3 alarms with shallow breathing, no stimulation was required.

Dr. Hardacre examined Sarah at 4-weeks of age. At this time, Sarah was growing and developing normal to advanced (length and head circumference ~50%, weight 75%). She was neurologically normal, alert, vigorous, and thriving.

Dr. Hardacre examined Sarah at 6-weeks of age. Dr. Hardacre felt Sarah was a well looking, thriving baby - vigorous and alert. Her weight had progressed from the 50% to almost the 90%.

Dr. Hardacre examined Sarah at 3-months of age. At this time, Sarah was growing and developing normal (length between 50 - 75%, ight 75%, head circumference 75%). She was determined to be neurologically and developmentally normal. Dr. Hardacre agreed .. arrange a repeat sleep study and urine metabolic screen to ensure Mr. and Mrs. Folbigg that all was within normal limits.

On March 9, 1993 Mrs. Folbigg brought Sarah to Dr. Elizabeth Pickford's office reporting that Sarah had been lethargic and sleeping poorly for the last few days. She had not been drinking well that day and had a little diarrhea and vomiting. Dr. Pickford determined that Sarah was suffering a probably viral upper respiratory tract infection. She was afebrile, happy and playful, thriving and developmentally normal. Height 65 cm. Weight 6950 grams.

Overall well child visits did not indicate any reason for concern, the baby's growth and development were gook. Sarah had a history of snoring at sleep. Although the apnea blanket seemed to stress Kathleen the pediatrician commented in his record that the number of false alarms was few.

In reviewing witness statements, there are conflicting stories about the night of August 29, 1993. What is clear is that the baby was put to bed in a single bed in the parents' bedroom without the apnea monitor. On the evening of Sarah's death, Craig Folbigg reports that Kathy appeared to be unhappy with her daughter. The mother reports hearing the child "turn over" at about midnight. She awoke again ~0100 hours August 30, 1993 and reports that she could not hear Sarah breathing. She found the child cyanotic and awoke her husband who commenced CPR while she called for an ambulance. In another statement given to Det. Glen Ward the parents state that Sarah was put to bed ~2130 hours but at some time the monitored was removed from her and she was moved into bed with the parents. Mrs. Folbigg reportedly awoke ~0130 hours, noticed that Sarah was not breathing and found fluid coming from her mouth. In yet another statement, Craig Folbigg states that he awoke at ~0100 hours and found Kathy not in the room, and Sarah still sleeping in her bed. About 0130 hours he was awoke by Kathy's screaming. Kathy was standing in the bedroom doorway; Craig turned and ked at Sarah and saw that the covers were off her body, she was laying on her back and her arms resting along her sides. He set ah on the floor, noting that she was still warm to the touch, and commenced CPR.

Upon arrival at ~0130 hours, emergency medical responders found Craig Folbigg performing CPR on Sarah. Emergency responders found Sarah fully clothed, cyanotic, warm to touch, not breathing, with mucus and vomit in her mouth. At ~0210 hours resuscitation efforts were discontinued as the infant remained asystolic. Sarah was transported to Maitland Hospital where Dr. John Stanger pronounced her dead at 0430 hours.

Autopsy was performed by John Millar Napier Hilton at the New South Wales Institute of Forensic Medicine at 0800 hours on August 31, 1993. Autopsy findings include:

- A well-developed, well-nourished female; weight 9.44 kg
- Small scratches on the right upper arm, below the lower lip on the left and on the chin
- Stomach contained curdled mild
- Lungs showed pulmonary edema and congestion
- Petechial hemorrhages were present, specifically in the thymus, heart, and lung
- Routine toxicological analysis was negative
- Cause of death: SIDS

My review of the autopsy does not include any additional comments with the exception that in my opinion, the cause of death is inconsistent with sudden infant death syndrome. In my opinion the cause of death is consistent with suffocation, manner homicide.

Dr. Bridget Wilcken performed a complete biochemical profile on blood samples from Sarah. The results were entirely normal. There is no positive evidence indicating an inherited metabolic disorder affecting amino acid, organic acid, or fatty acid oxidation pathways. These normal results tend to exclude certain disorders of amino acid metabolism (phenylketonuria, the tyrosinaemias, maple syrup urine disease, homocystinuria due to cystathionine synthase deficiency) and a large number of organic aciduria s and fatty acid oxidation defects, including methylmalonic acidaemia, propionic acidaemia, medium chain acyl CoA dehydrogenase deficiency, and several other disorders which are extremely rare. The laboratory also conducted a DNA test for the common mutation seen in medium chain acyl CoA dehydrogenase deficiency, which is present in 98% of known cases of this disorder. This mutation was not present in any of the children's samples.

Opinions

In forming my conclusions I have utilized all the materials made available to me including all the medical history and records, the autopsy reports and materials, the police investigative documents, the interview transcripts from Kathleen Folbigg as well as witness statements, diary entries, and listening device materials.

The materials and investigative information provided in this case are of excellent quality and are sufficient for me to render an opinion to a reasonable degree of medical certainty.

It is my opinion to a reasonable degree of medical certainty that Sarah Folbigg did not die of the condition known as Sudden Infant Death Syndrome. This child was within 2 weeks of approaching her first birthday. In my opinion she does not fall within the age range associated with SIDS and would not be considered for the diagnosis of SIDS for that reason in and of itself. Although the classic classification of SIDS includes children under 1 year of age, this is not the age range accepted by most forensic pathologists and a sudden unexpected infant death, greater than 6-months from the SIDS condition would be considered atypical and by essentially ear of age would be excluded. It is my opinion that Sarah's death is most consistent with death by suffocation. It is my opinion that Sarah Folbigg was the victim of probable homicidal assault that resulted in suffocation.

I have participated in the investigation of both accidental and homicidal suffocation in children over the course of my 20 years as a practicing pediatric forensic pathologist. Unfortunately multiple infant homicides within one family are now well documented in the literature and in forensic experience. Typically the perpetrator knows not confess to the crimes but in many cases such as this the facts of the case make the diagnosis. Important facts in this case that lead to the conclusion of homicidal suffocation include:

- The autopsy fails to identify any known natural disease of disease process that could explain the sudden death of Sarah. She was growing and developing normally for her age and circumstance.
- The findings at autopsy that are consistent with the determination of death by suffocation.
- Sarah was in the care of her mother at the time of his death and she was the last person to see her alive.
- All of the Folbigg infants were all in the care of their mother at the time of their death and she was the last person to see each of them alive.
- None of the deaths in the Folbigg case can be attributed to SIDS. It is well recognized that the SIDS process is not a hereditary problem and the statistical probability that 4 children in one sibship could die from SIDS would be infinitesimally small. If you calculate the risk of this event occurring 4 times in one family, using routine statistical probability for a random event occurring 4 times in one family (with an occurrence of <1/1000 live births) it would be less than 1 in one trillion.
- The diagnosis of SIDS requires that following a complete investigation and autopsy no other cause of death is identified. Sarah's death cannot be considered sudden infant death syndrome and should be investigated as a homicide.
- Absence of risk factors commonly recognized in the epidemiology of Sudden Infant Death Syndrome. These include young age, prone sleeping position, inadequate prenatal care, low birth weight/premature birth, maternal smoking, ethnicity, low socioeconomic status, young maternal age, multiple births. There is no history of infant apnea or significant breathing problems; no evidence of hyperthermia; no recent history of illness.

In my opinion the cause of death and manner of death should be listed as follows:

- Cause of Death: Suffocation
- Manner of Death: Homicide

SIDS, also called crib or cot death, is the sudden death of an infant under 1-year of age that remains unexplained after thorough case investigation, including the performance of a complete autopsy, examination of the death scene, and a review of the clinical history. The SIDS diagnosis should not be applied unless all of the following are true:

- A complete autopsy is done, including the cranium and cranial contents, and autopsy findings are compatible with SIDS
- There is no gross or microscopic evidence of trauma of significant disease process
- There is no evidence of trauma on a skeletal survey
- Other causes of death are adequately ruled out and
- There is no evidence of current alcohol, drug, or toxic exposure; and thorough death scene investigation and review of the clinical history are negative

The SIDS diagnosis has been applied variably during the latter half of this century. What we know now is that some children who were diagnosed as SIDS in the past were actually murdered and families where there were multiple infant deaths attributed to SIDS were actually the victims of serial killings. The current practice is to carefully apply the diagnosis of SIDS only in cases that fulfill the current diagnostic criteria. It is now common practice for the medical examiner to reconsider the deaths of children originally thought to have died of SIDS when subsequent infants die suddenly, unexpectedly, and without explanation. This is certainly the circumstance that applies in Sarah's case. There have been no cases of metabolic disorders reported where multiple children in one sibship have died suddenly and without explanation following complete medical evaluations and autopsy with essentially negative findings. Comprehensive medical testing and genetic consultation have failed to reveal any abnormalities in the Folbigg family.

As a routine practice, I continue to review the world literature in my practice. I have included some references in my SIDS research that I consider relevant to this case, but I did not base my opinions on them.

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LAURA ELIZABETH FOLBIGG

Findings

Laura Folbigg was the product of a full term pregnancy and her mother, Kathleen Folbigg, received adequate prental care. She was delivered vaginally following an essentially uncomplicated labor on August 7, 1997 at Singleton Hospital. Pregnancy and delivery were essentially uncomplicated. Birth weight 3260 grams. Length 49 cm. Apgars 9 and 10 at 1 and 5 minutes. Mr. and Mrs. Folbigg elected to take Laura home with apnea monitoring. Laura was discharged home with her mother.

Dr. Christopher Seton was contacted in January of 1997 and notified by Craig Folbigg that he and his wife were expecting the birth of their fourth child. Dr. Seton agreed to provide a full investigation by the sleep disorder unit at West Mead at the time of their baby's birth. On Tuesday August 19, 1997, Laura was submitted to a comprehensive evaluation which included sleep study, full biochemical, blood and metabolic investigation. Dr. Seton's impression was that the child had a mild central apnea that resolved over time and were interpreted as of no medical significance. At no time did her clinical picture or studies show evidence of upper airway obstruction or bradycardia. Her care was monitored by the medical staff at New Children's Hospital, West Mead (Ms. Margaret Tanner). She was discharged home on August 21, 1997.

On February 3, 1998 Laura underwent a sleep study under the care of Dr. Seton. Dr. Seton's impression is that Laura's sleep breathing had at that time normalized with no evidence of upper airway obstruction. It was Dr. Seton's impression that home monitoring should continue but that no further sleep studies were required without evidence or change in breathing patterns.

In March 1998, Craig Folbigg, in a letter to Margaret Tanner, expresses concern regarding Kathleen's diligence in monitoring Laura's sleep during the day. "Strangely though feel that Kathy finds it all tedious and frustrating and would probably rather not use it at all." I'm more than sure that Kathy does not monitor Laura during her daytime sleeping.

April 30, 1998 Laura again saw Dr. Seton. At that time, she was considered to be meeting all developmental milestones with optimal weight gain. Dr. Seton found no physical evidence of sleep disordered breathing.

Laura received her primary care from Dr. Sanders of Singleton and Dr. Innis of Singleton Heights Medical Practice. The child's last visit to a doctor was with Dr. Innis in early February 1999 for her 18-month vaccination.

Laura had a one-week history of cold and flu-like symptoms at the time of her death of March 1, 1999. She had been administered Demazin for treatment of symptoms. She received her last dose of medication of February 27, 1999.

According to statements, on March 1, 1999 Kathleen took Laura to the gym and to Craig Folbigg's place of work to visit. Laura was apparently in a bad mood but did not appear to be ill. Kathleen reported that Laura fell asleep in the car on the way home and she put her to bed upon arrival at home so that she was lying in her crib on her right side. ~30 to 60 minutes later Kathleen heard Laura coughing "in the bedroom". She checked on her 5 minutes later and found her supine, pale, and not breathing. She took Laura to the breakfast bar in the kitchen where she called an ambulance and commenced CPR. Emergency personnel arrived at the Folbigg residence at 1214 hours to find Kathleen alone with Laura who was laying in the supine position on a breakfast bar inside the house, pulseless, not breathing, warm to the touch and in full cardiopulmonary arrest (essentially DOA). No blood, vomit, or foreign object was appreciated in Laura's mouth, although cyanosis was present. At 1229 hours the infant was transferred to Singleton Hospital. Laura arrived at Singleton Hospital ER at 1235 hours in full cardiac arrest. Upon presentation in the ER, Laura was cyanotic, pupils fixed and dilated, pulseless, not breathing. Resuscitation attempts were discontinued 10 minutes after arrival in the ER. Laura was pronounced dead at 1245 hours on March 1, 1999. Dr. Tuan Au, treating physician, noted no bruises, marks or abnormalities in Laura's physical appearance. Dr. Au suspected that Laura's death was not from SIDS but from homicide.

opsy examination was performed by Dr. Allen David Cala at ~2100 hours on March 1, 1999 at New South Wales Institute of Forensic Medicine. Autopsy findings included:

- A well-developed, well-nourished 20-month old female, weight 11.552 kg.
- Lividity was noted on the left side of the face and posteriorly.
- No significant physical injuries were identified on physical examination.
- Lungs showed focal hemorrhage and collapse
- Heart showed no gross abnormalities. Microscopic examination of the tissues from the heart revealed inflammatory infiltrate in the heart, consistent with viral myocarditis.
- Petechial hemorrhages were present in the thymus.
- Routine toxicological analysis was negative.
- Cause of death: Undetermined
- Dr. Cala has not excluded the possibilitiy of multiple homicides in this family. "if homicidal acts have been committed, it is most likely these acts have been in the form of deliberate smothering. Smothering, whether deliberately or accidentally inflicted, may leave no trace. There are no specific post-mortem findings for smothering. It is usually performed by one person, in the absence of any witnesses. It is relatively easy for an adult to smother an infant or small child with a hand, pillow, soft toy or other similar object."

My review of the autopsy includes the following additional comments:

- The microscopic sections of heart from Laura Folbbig reveal the presence of myocarditis, most probably viral in origin. Dr. Cala states in his report that his finding of myocarditis is consistent with Laura's recent illness and is probably incidental. I concur with this conclusion. Microscopic foci of inflammation of the heart is related to her cold symptoms and are not the cause of her death
- Examination of the many sections of lung reveal striking and extensive hemorrhage into the lung tissue. This finding is consistent with the reports in the literature that suggests that extensive pulmonary hemorrhage is suspicious for suffocation.

Dr. Bridget Wideken performed a complete biochemical profile on blood samples from Laura. The results were entirely normal. There is no positive evidence indicating an inherited metabolic disorder affecting amino acid, organic acid, or fatty acid oxidation pathways. These normal results tend to exclude certain disorders of amino acid metabolism (phenylketonuria, the tyrosinaemias, maple syrup urine disease, homocystinuria due to cystathionine synthase deficiency) and a large number of organic aciduria and fatty acid oxidation defects, including methylmalonic acidaemia, propionic acidaemia, medium chain acyl CoA dehydrogenase deficiency, and several other disorders which are extremely rare. The laboratory also conducted a DNA test for the common mutation seen in medium chain acyl CoA dehydrogenase deficiency, which is present in 98% of known cases of this disorder. This mutation was not present in any of the children's samples.

Opinions

In forming my conclusions I have utilized all the materials made available to me including all the medical history and records, the autopsy reports and materials, the police investigative documents, the interview transcripts from Kathleen Folbigg as well as witness statements, diary entries, and listening device materials.

The materials and investigative information provided in this case are of excellent quality and are sufficient for me to render an opinion reasonable degree of medical certainty.

It is my opinion to a reasonable degree of medical certainty that Laura Folbigg did not die of the condition known as Sudden Infant Death Syndrome. In my opinion she does not fall within the age range associated with SIDS and would not be considered for the diagnosis of SIDS for that reason in and of itself. It is my opinion that Laura's death is most consistent with death by suffocation. It is my opinion that Laura Folbigg was the victim of probable homicidal assault that resulted in his suffocation.

I have participated in the investigation of both accidental and homicidal suffocation in children over the course of my 20 years as a practicing pediatric forensic pathologist. Unfortunately multiple infant homicides within one family are now well documented in the literature and in forensic experience. Typically the perpetrator knows not confess to the crimes but in many cases such as this the facts of the case make the diagnosis. Important facts in this case that lead to the conclusion of homicidal suffocation include:

- The autopsy fails to identify any known natural disease of disease process that could explain the sudden death of Sarah. She was growing and developing normally for her age and circumstance.
- The findings at autopsy are consistent with the determination of death by suffocation.
- Laura was in the care of her mother at the time of his death and she was the last person to see her alive.
- All of the Folbigg infants were all in the care of their mother at the time of their death and she was the last person to see each of them alive.
- None of the deaths in the Folbigg case can be attributed to SIDS. It is well recognized that the SIDS process is not a hereditary problem and the statistical probability that 4 children in one sibship could die from SIDS would be infinitesimally small. If you calculate the risk of this event occurring 4 times in one family, using routine statistical probability for a random event occurring 4 times in one family (with an occurrence of <1/1000 live births) it would be less than 1 in one trillion.
- The diagnosis of SIDS requires that following a complete investigation and autopsy no other cause of death is identified. After the deaths of the Folbigg children, suddenly and unexpectedly, without explanation, none of the infant deaths can be considered sudden infant death syndrome and should be investigated as homicides.
- Absence of risk factors commonly recognized in the epidemiology of Sudden Infant Death Syndrome. These include young age, prone sleeping position, inadequate prenatal care, low birth weight/premature birth, maternal smoking, ethnicity, low socioeconomic status, young maternal age, multiple births. There is no history of infant apnea or significant breathing problems; no evidence of hyperthermia.

- In my opinion the cause of death and manner of death should be listed as follows:
  - Cause of Death: Suffocation
  - Manner of Death: Homicide

SIDS, also called crib or cot death, is the sudden death of an infant under 1-year of age that remains unexplained after thorough case investigation, including the performance of a complete autopsy, examination of the death scene, and a review of the clinical history. The SIDS diagnosis should not be applied unless all of the following are true:

- A complete autopsy is done, including the cranium and cranial contents, and autopsy findings are compatible with SIDS
- There is no gross or microscopic evidence of trauma of significant disease process

- There is no evidence of trauma on a skeletal survey
- Other causes of death are adequately ruled out and
- There is no evidence of current alcohol, drug, or toxic exposure; and thorough death scene investigation and review of the clinical history are negative

The SIDS diagnosis has been applied variably during the latter half of this century. What we know now is that some children who were diagnosed as SIDS in the past were actually murdered and families where there were multiple infant deaths attributed to SIDS were actually the victims of serial killings. The current practice is to carefully apply the diagnosis of SIDS only in cases that fulfill the current diagnostic criteria. It is now common practice for the medical examiner to reconsider the deaths of children originally thought to have died of SIDS when subsequent infants die suddenly, unexpectedly, and without explanation. This is certainly the circumstance that applies in this case. There have been no cases of metabolic disorders reported where multiple children in one sibship have died suddenly and without explanation following complete medical evaluations and autopsy with essentially negative findings. Comprehensive medical testing and genetic consultation have failed to reveal any abnormalities in the Folbigg family.

As a routine practice, I continue to review the world literature in my practice. I have included some references in my SIDS research that I consider relevant to this case, but I did not base my opinions on them.

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Summary Opinions

It is my opinion, to a reasonable degree of medical certainty, that the four Folbigg children were all the victims of homicidal assaults that resulted in their suffocation. The process of suffocation will take ~4 to 5 minutes to complete. During the first 1.5 to 2 minutes, while they are still fully conscious, the child will fight aggressively for their life. In small infants, this typically does not result in any external signs or physical evidence.

Important facts in this case that lead to the conclusion of homicidal suffocation include the following:

- The autopsy fails to identify any known natural disease or disease process that could explain the sudden deaths of these infants. All four children were growing and developing normally for their age and circumstance. Despite Patrick's handicaps he was advancing well.
- The autopsy findings in these babies are all consistent with death by suffocation.
- The infants were all in the care of the same person at the time of their death, their mother, and she was the last person to see each of them alive.
- None of the deaths in this case can be attributed to SIDS [Sudden Infant Death syndrome]. It is well recognized that the SIDS process is not a hereditary problem and the statistical likelihood that 4 children could die from SIDS is in excess of 1 in a trillion.

- The diagnosis of SIDS requires that following a complete investigation and autopsy no other cause of death is identified. Forensic standards of practice would not allow for consideration of a second diagnosis of SIDS after a second sudden death and by the time a third child has died, the death must be investigated as a homicide.
- Patrick's sudden, profound and irreversible brain damage is consistent with and diagnosed as a hypoxic episode. Hypoxia in this case is synonymous with asphyxia and unfortunately heralds the fatal event in retrospect. No natural disease or process has been identified to explain this event. In my opinion, the cause of Patrick's cardio-respiratory arrest is the same process that killed him and his siblings.

The medical literature from the 70s and 80s that supported multiple cases of SIDS in one family have come under dispute because many if not all of the cases could have been homicides. There are no verified or substantiated cases of 3 or more SIDS deaths in one family. The current epidemiology of SIDS has been revised and there is no hereditary risk for the event.

Risk factors for SIDS have been identified from epidemiological data. Epidemiology is the study of distribution and determinants of health related states or events in specified populations, and the application of this study to control health problems. Such factors as time of year or time of day are epidemiological data and cannot be applied to an individual case or cases for diagnosis.

Current concepts of risk factors for SIDS are changing. However the historically recognized risk factors for SIDS include:

Maternal smoking
Prone sleeping condition
2-4 month age group
Poor prenatal care
Low socioeconomic status
Young maternal age
Multiple births
Higher incidence in males
Low birth weight / preterm delivery

These factors can obviously be present in a second or third child in the same family. The incidence of SIDS varies by region and race around the world. Vital statistics for age, race, and location in the US demonstrate that the incidence of SIDS has declined over the last two decades from 1-2 /1000 live births to < 1/1000 live births.

If you have any additional questions, or should you need additional information, please do not hesitate to contact me.

Sincerely,

Janice Ophoven, M.D.
Pediatric Forensic Pathologist

PROFESSOR PETER B HERDSON

MB, ChB, B Med Sc, PhD, FRCPA, FRANZCR (Hon)

Consultant Forensic Pathologist

EXPERT CERTIFICATE in the matter of:

Police vs Kathleen Megan Folbigg

Place: 18/51 Musgrave St, Yarralumla, Canberra, ACT, 2600 Date: 17 January 2002

Name: PETER BARRIE HERDSON

Contact Address: 18/51 Musgrave St., Yarralumla, ACT, 2600

Occupation: Consultant Forensic Pathologist Tel. No: 02 6260 3576

STATES: -

EXPERT CERTIFICATE
Section 177, Evidence Act 1995 No. 25

1 This statement made by me accurately sets out the evidence which I would be prepared, if necessary, to give in court as a witness.

The statement is true to the best of my knowledge and belief and I make it knowing that, if it is tendered in evidence, I shall be liable to prosecution if I have wilfully stated anything which I know to be false or do not believe to be true.

2 I am 69 years of age.

3 I hereby certify, I am a duly qualified and registered Medical Practitioner, Professor of Pathology and Consultant Forensic Pathologist. I have a specialised knowledge based on my training, study and experience as a Pathologist for the past 40 years, and as a Forensic Pathologist for the past 31 years. I hold the qualifications of Member of the Pharmaceutical Society of New Zealand; Bachelor of Medical Science (1956); and Bachelor of Medicine, Bachelor of Surgery (1959) from the University of Otago, Dunedin, New Zealand; Doctor of Philosophy in Pathology (1965) from Northwestern University, Chicago, Illinois, United States of America; Fellow of the Royal College of Pathologists of Australasia (1969); Honorary Fellow of the Royal Australasian and New Zealand College of Radiologists (1985). As a Forensic Pathologist, I have dealt with a large number of infant deaths over the past 30 years or so.

4 On Monday November 12, 2001 I met with Detective Sergeant Bernie Ryan in my office in Canberra, having had several telephone conversations with Sergeant Ryan over previous weeks. We discussed aspects of the deaths of Caleb, Patrick, Sarah and Laura Folbigg and Sergeant Ryan left me with five large dossiers of material relating to the case together with a total of

128 histopathological slides relating to the post mortem examinations on the four infants.

Subsequently, I have examined all of this material and offer the following comments.

I will first consider each of these four deaths in isolation, and then together.

(i) The death of Caleb Gibson Folbigg, DOB 1.2.89, died 20.2.89.

I concur with the detailed clinical summaries, assessment of the post mortem examination performed Dr Roy Cummings on February 20, 1989 in the City Morgue, Newcastle, NSW, and the assessment of the subsequent histopathology and toxicologic analysis provided by Professor Peter Jeremy Berry of Bristol, England and Dr Janice Jean Ophoven of Woodberry, Minnesota, United States of America.

In my opinion, the findings taken in isolation leave the cause of death undetermined, but apparently consistent with Sudden Infant Death Syndrome.

(ii) The death of Patrick Folbigg, DOB 3.6.90, apparent life threatening episode 18.10.90 with subsequent epilepsy and blindness, died 13.2.91.

I concur with the detailed clinical summaries, assessment of the post mortem examination performed by Dr Jan Bishop and Gurpret Singh Khaira at the Newcastle Mater Hospital, Newcastle, NSW, whose final diagnosis was that of a normally formed male infant of approximately 8 months age with old infarcts and gliosis in the parieto-occipital area of both cerebral hemispheres probably secondary to the cardio-respiratory episode suffered by the infant when aged approximately 5 months, and the assessment of the subsequent histopathology and toxicologic analysis provided by Professor Peter Jeremy Berry of Bristol, England and Dr Janice Jean Ophoven of Woodberry, Minnesota, United States of America.

In my opinion, the history of a life threatening episode with subsequent abnormalities would be most unusual for a death to be due to so-called Sudden Infant Death Syndrome and the cause of death in this case is more accurately undetermined.

(iii) Sarah Folbigg. DOB 14.10.92, died 30.8.93.

I concur with the detailed clinical summaries, assessment of the post mortem examination performed by Associate Professor John M N Hilton at the Glebe Forensic Institute in Sydney, NSW, where the findings taken in isolation could be diagnosed as Sudden Infant Death Syndrome, and the assessment of the subsequent histopathology and toxicologic analysis provided by Professor Peter Jeremy Berry of Bristol, England and Dr Janice Jean Ophoven of Woodberry, Minnesota, United States of America.

(iv) Laura Folbigg. DOB 7.8.97, died 1.3.99.

I concur with the detailed clinical summaries, assessment of the post mortem examination performed by Dr Allan D Cala at the Glebe Forensic Institute in Sydney, NSW where the cause of death was undetermined, and the assessment of the subsequent histopathology and toxicologic analysis provided by Professor Peter Jeremy Berry of Bristol, England and Dr Janice Jean Ophoven of Woodberry, Minnesota, United States of America. In this case, I agree that histopathology of the heart reveals a myocarditis which is most probably of viral origin and I further agree with Dr Cala that his finding of myocarditis is consistent with Laura's recent illness and is probably incidental.

Laura died when aged approximately 19 months, and this is significantly older that the usual age range for Sudden Infant Death Syndrome.

Considering these four infant deaths together, I would draw attention to the comments of other Pathologists (and in agreement with my own experience) that the first unexplained death of an infant in a family may be attributed to Sudden Infant Death Syndrome, the second should be labelled undetermined and the third should be considered homicide until proven otherwise. I am unaware that there have ever been three or more thoroughly investigated infant deaths in one family from Sudden Infant Death Syndrome.

5 Based on all the material that I have reviewed relating to these four infant deaths, in my opinion all four infants probably died from intentional suffocation.

- In drawing this conclusion, apart from my comments above I would draw attention to the wide age range of the children at the time of the initial observed events or deaths, twenty days for Caleb to approximately 19 months for Laura.

- The fact that two infants, Patrick on 18.10.90 and Laura on 1.3.99, were found moribund rather than dead is not the pattern associated with Sudden Infant Death Syndrome.

- The pattern of the mother's actions and reactions over the ten year period is not typical of so-call Munchausen Syndrome by proxy.

Respectfully submitted.

PETER B HERDSON
MB, ChB, B Med Sc, PhD,
FRCPA, (Hon) FRANZCR
Consultant Forensic Pathologist

Witness: _____ Signature: _____

Forensic Science

Annexure "A"

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18/10/02

To: Mr Peter Krisenthal,
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PO Box K847,
Haymarket, NSW, 2000

Re: the deaths of Caleb, Patrick, Sarah and Laura Folbigg

I have been asked by Mr. Peter Krisenthal in two letters dated 8/8/02 and 16/9/02 to provide an opinion as to the causes of death of these infants. In preparing this report I have based my opinions on:

1) An autopsy report on Caleb Gibson Folbigg by Dr. R. Cummings dated 9/5/89;
2) An autopsy report on Patrick Allan Folbigg by Dr. J. Bishop and Dr. G. Singh-Khaira dated 14/2/91
3) A neuropathology report on Patrick Allan Folbigg by Dr. A. Kan dated 24/6/91;
4) An autopsy report with associated ancilliary testing on Sarah Kathleen Folbigg by Assoc. Prof. J.M.N. Hilton dated 25/11/93;
5) A neuropathology report on Sarah Kathleen Folbigg by Dr. R. Pamphlett undated;
6) An autopsy report with associated ancilliary testing on Laura Elizabeth Folbigg by Dr A.D. Cala dated 26/7/99;
7) A series of 48 glass slides from Laura Elizabeth Folbigg;

8) A series of 29 colour autopsy photographs of Laura Elizabeth Folbigg;
9) A blue folder of medical records of Caleb Folbigg;
10) A black folder of medical records of Patrick Folbigg;
11) A blue folder of medical records of Sarah Folbigg;
12) A blue folder of medical records of Laura Folbigg;

I have also received reports and statements of expert opinions of:

1) Dr B. Wilcken dated 10/12/91 & 14/1/00;
2) Dr. A. Colley dated 4/12/91 & 27/2/92;
3) Dr. S.M. Beal dated 8/12/99;
4) R. Garbutt dated 4/2/00;
5) Prof. J. Berry dated November 2000;
6) Dr. J. Ophoven dated 6/10/00 & 1/12/01;
7) Prof. P. Herdson dated 17/1/02.

BACKGROUND:

I am currently employed by the Forensic Science Centre in Adelaide as a Specialist Forensic Pathologist and have been there since May 1999; prior to that I was a Senior Consultant Histopathologist at the Women's and Children's Hospital, with a position of Visiting Consultant Pathologist at the Forensic Science Centre. I hold Clinical Professorships with the Departments of Pathology and Paediatrics at the University of Adelaide. I am also a Consultant Paediatric Forensic Pathologist to the Child Protection Unit at the Women's and Children's Hospital, Adelaide.

I qualified in medicine in Australia in 1978 (University of Tasmania (MBBS) and in Canada in 1982 (LMCC). I hold fellowships in Anatomical Pathology in three countries: Canada (FRCPC), the United Kingdom (FRCPath) and the United States (FCAP). I also hold a fellowship in Family Medicine with the Canadian College of Family Physicians (CCFP). I have a specific interest in sudden infant and childhood death and have published or have in press over 270 papers in peer-reviewed journals, many of which deal with natural, accidental and homicidal causes of sudden infant death. I have also presented or coauthored over 200 papers that have been presented at national and international meetings. I regularly direct or codirect workshops for pathologists, police officers and lawyers on issues in paediatric forensic pathology and have been invited to present such material in Australia, the United States, the United Kingdom, parts of Europe, South Africa, Israel, Canada, Indonesia and Japan. I have coauthored a text on sudden childhood death (the second edition of which is pending), have edited another text on sudden infant death syndrome, and am at present coediting an Encyclopedia of Forensic and Legal Medicine. I have two higher degrees: a Doctor of Medicine (MD) and a Master of Medical Science (MMedSci), both from the University of Adelaide. The theses for these degrees both deal with aspects of sudden death in infants and children. I have performed over 600 autopsies on children, infants and fetuses and appear regularly in court. I also regularly receive paediatric medicolegal cases for opinion from colleagues in Australia and New Zealand, and occasionally the United States. I have enclosed a copy of my full CV for your information.

OPINION AND COMMENTS:

The current cases are exceedingly complex raising many issues in paediatric forensics that are either not clear cut, or in some cases are not completely understood. Important information from the death scene or from tissue examination was sometimes not available as these examinations were not always performed. For this reason it is often difficult to make definitive statements about possible diagnoses. I will not summarise the medical and social histories of the children as this has already been done in some detail in several of the reports that I have referred to.

It should be stated at the outset that sequential deaths of four young children in the same family are exceedingly rare, are of great concern and must always raise the possibility of homicide or an inherited abnormality. For this reason it is vital at the time of autopsy to check for any evidence of underlying disease. Unfortunately the pathological findings following suffocation in infants and young children are often completely nonspecific[1] and so the family history and social circumstances must also be considered in formulating an autopsy diagnosis. While I found diary entries by Kathleen Folbigg concerning, I would not feel qualified to comment on their psychiatric significance. They require expert assessment.

The most likely causes of multiple infant deaths in a family with no abnormalities clinically or at autopsy are inflicted suffocation or rare inherited disorders of metabolism. However, this refers to cases where no abnormalities are detected, whereas the current cases are quite different in that unequivocal abnormal findings were present. i.e.:

1) Patrick had chronic brain damage and epilepsy that was difficult to control;

2) Laura had established myocarditis.

These are well recognised and accepted causes of death in children[2,3]. Thus, while I would agree that suffocation cannot be excluded in any of these children, I would also not be able to exclude underlying organic illness as a cause of death in two of the four children (Patrick and Laura). There was also clinical evidence of an organic disorder that may be related to airway compromise and respiratory arrest in a third child[4] (Caleb), and autopsy evidence of airway narrowing in the remaining child (Sarah).

If these children presented as <u>individual isolated deaths in separate families</u> I would have listed the major issues and causes of death as:

1) **Caleb, aged around 19 days, DOB 1/2/89.**

Caleb was allegedly found deceased in his bassinette by his mother on 20/2/89.

Sudden infant death syndrome or SIDS is defined as 'the sudden death of an infant under one year of age which remains unexplained after a thorough case investigation, including performance of a complete autopsy, examination of the death scene and review of the clinical history'[5,6]. A death scene examination by a pathologist or a trained person is therefore required before a diagnosis of SIDS can be made. This is in part to exclude the possibility of accidental asphyxia. As I was unable to find a formal death scene examination for Caleb I would not be able to exclude the possibility of a sleeping accident and so would not be able to make a diagnosis of SIDS. I will not use the term SIDS if there has not been formal assessment and recording of the death scene findings.

Another point of concern is the issue of Caleb having episodic respiratory difficulties with a diagnosis of a floppy larynx (voice box). I would not diagnose SIDS in any infant who has had a history of airway narrowing with breathing difficulties as I could not say that this was not involved in the fatal episode. As no histologic examination was conducted of the larynx at the autopsy (not a routine examination), it is uncertain whether there were any structural abnormalities of cartilage present. Laryngomalacia has been associated with airway obstruction and recurrent apnoea of infancy with some infants requiring resuscitation. Three infants with laryngomalacia in one study had episodes of collapse during hospitalisation observed by medical personnel[4]. Two infants in another family who died suddenly have also been reported with a similar condition (softening of the airway below the larynx, the bronchi) raising the possibility of this being involved in their deaths[7].

Another significant omission in this case was that the brain did not appear to have been examined histologically.

Subsequent examination of lung sections by Prof. Berry revealed scattered iron containing macrophages (scavenger cells). While this has been claimed to be highly suggestive of an asphyxial episode[8] I have found in a separate study that nearly one in five infants who die of SIDS have this finding, in addition to infants dying of nonasphyxial disorders; i.e. it is not specific for asphyxia[9].

Given the above points and omissions I would have to label the cause of death as 'undetermined', noting a history of breathing problems involving a floppy larynx (laryngomalacia).

2) **Patrick, aged around 8 months, DOB – 3/6/90**

Patrick was allegedly found deceased by his mother in his cot on 13/2/91. His medical history included an episode of previous severe brain damage resulting in a seizure disorder.

In isolation, the cause of death would appear to be reasonably clear cut given the history of frequent seizures. Dr. A. Kan in his neuropathology report found changes of scarring, atrophy and inflammation that were in keeping with seizures, previous cardiorespiratory arrest and possibly

treated encephalitis. The changes were however, chronic and relatively nonspecific and could have arisen from a variety of quite different diseases and conditions.

The frequency of sudden death in epilepsy (known as SUDEP) in children is unknown, however in general epileptic populations estimates have ranged from one in 200 to one in 680 patients. The typical case of unexpected death encountered in paediatric autopsy practice is of an epileptic child, often with mental retardation, who is found dead in bed with minimal external or internal findings.

The association of sudden death with sleep is noteworthy and most likely relates to reduction in seizure threshold, with an increase in epileptic discharges. A variety of theories have been proposed to explain the occurrence of sudden death in epilepsy including suffocation from bedding, asphyxia, pulmonary fluid overload (edema) and cardiac arrhythmia. Suffocation and aspiration of food or foreign material are considered unlikely in most cases.

The most popular theory to explain why apparently stable epileptic children are at increased risk of sudden death involves nervous system instability with abnormal cardiac rhythms during seizure activity.

The absence of death scene and autopsy findings of disturbed bedding, urinary or faecal incontinence, bite marks on the tongue and foam in the mouth or trachea, does not mean that an epileptic episode did not occur, as these features have been absent in fatal episodes that have been witnessed. As any type of fit may precede sudden death, not just generalized tonic/clonic convulsions this could explain minimal external findings. Autopsy investigations may show pre-existing chronic brain damage or developmental malformations with loss of nerve cells (neuronal depopulation) and scarring (gliosis) of the hippocampus secondary to past hypoxic episodes, usually with no evidence of an acute lesion[2].

In Patrick's case the event that provoked the episode of oxygen deprivation to the brain is less clear. However a CT scan from the Newcastle Mater Hospital dated 23/10/90 stated that the image was 'compatible with encephalitis' and a follow-up scan dated 5/11/90 noted 'generalised loss of brain substance' which 'could be related to post inflammatory change'. There was no mention of intracerebral or retinal haemorrhage or diffuse cerebral oedema to suggest possible inflicted injury. Although Dr. M. DeSilva considers that the findings were compatible with shaking, they were relatively nonspecific, without any of the characteristic features of shaking-impact syndrome such as bleeding and tears within the brain or its coverings being identified on admission.

With such an abnormal brain and history, I would have attributed death to epilepsy against a background of possible encephalitis. There was no clinical documentation of features to support a diagnosis of shaking-impact syndrome[10].

3) **Sarah, aged around 10.5 months, DOB – 14/10/92.**

Sarah was allegedly found dead in her bed by her mother on 30/8/93.

Again I could find no evidence of a death scene examination performed by, or involving, a pathologist.

Prof. Hilton has commented on an unusually congested uvula which produced an 'obstructive element in the airway'. I am not sure of the significance of this finding as it is not something that I have personally seen, however, I do not think that the observation of upper airway narrowing by such an experienced pathologist should be discounted. Sudden and unexpected death is well-recognised in infants with narrowing of the upper airways due to a variety of cysts, tumours and malformations[11].

Given the above points, with no other abnormal findings present at autopsy, I would have to label the cause of death as 'undetermined', with an autopsy finding of narrowing of the upper airway.

4) **Laura, aged around 19 months, DOB – 7/8/97.**

Laura was allegedly found not breathing by her mother on 1/3/99. She had a recent history of an apparent upper respiratory tract infection.

I would agree with Dr. Cala and Prof. Berry that the slides from the heart demonstrated myocarditis. Myocarditis is a well-known cause of sudden and unexpected death in children of all ages and may be found in infants who present in a similar manner to SIDS. Although some children may have symptoms and signs of heart failure a significant number of cases will have nonspecific clinical features giving no indication of a primary cardiac problem prior to autopsy.

Myocarditis is most commonly caused by microbiological agents, in particular to coxsackie B viruses. Other viruses such as coxsackie A, polio, Echo, influenza A, adeno, cytomegalovirus HIV and parvovirus may also cause myocarditis and death due to cardiac involvement. I could not find any evidence that confirmatory viral studies were performed at the time of autopsy, presumably because the inflammation was not detected until microscopic examination was performed[3].

Given the finding of extensive myocardial inflammation with no other abnormalities present I would have attributed the death to myocarditis. An identical conclusion would be drawn by 'most pathologists' according to Prof. Berry. This is with the recognition that myocarditis may be found coincidentally at autopsy in children dying of a wide range of other conditions.

The autopsy findings, however, cannot be taken in isolation and with the occurrence of 4 deaths within the same family and police concerns I would list the causes of death as follows:

1) Caleb: Undetermined, with laryngomalacia;

2) Patrick: Undetermined, cannot exclude epilepsy;

3) Sarah: Undetermined, with narrowing of the upper airway;

4) Laura: Undetermined, cannot exclude myocarditis.

CONCLUSIONS:

In my view the critical issue in the pathology of these cases is the presence of underlying conditions which are known to cause sudden death in young children and babies. I am certainly concerned that there may have been inflicted suffocation but could not state unequivocally that this had occurred, and could not agree that their autopsies have failed to 'identify any known natural disease or disease processes that could explain the sudden deaths', as has been stated. by Dr Ophoven.

Although these cases are discussed in several of the expert reports as SIDS deaths they cannot, by definition, be regarded as such, either on their own or together. Thus, comments on the significance of the presence or absence of SIDS risk factors and use of statistics derived from SIDS deaths are not applicable.

The unusual background of this family with many issues of concern does not negate the fact that potentially significant organic illness was present in these children. Upper airway narrowing, epilepsy and myocarditis may have been coincidental to their deaths, but alternatively may have been causative or contributory; unfortunately this issue cannot be clarified from the autopsy records. Given the information that I have been provided with I simply cannot see how the significance of these conditions can be down-played as potential causes of death, no matter how worrying the circumstances are.

Clinical Professor Roger W. Byard
BMedSci, MB, BS, MMedSci(Paed), MD,
CCFP, MACLM, FCAP, FRCPC, FRCPath

Robert Ouvrier
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28th October 2002

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Criminal Investigations
NSW Police Service
274 Sloane Street
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ATT: Detective Sergeant B Ryan

EXPERT CERTIFICATE IN THE MATTER OF : POLICE – v KATHLEEN MEGAN FOLBIGG

28th October 2002

Robert Ouvrier, Department of Neurology, The Children's Hospital at Westmead
Occupation: Paediatric Neurologist states:-

EXPERT CERTIFICATE

Section 177 : Evidence Act 1995 No. 25

1. The statement made by me accurately sets out the evidence which I would be prepared, if necessary, to give in court as a witness.

The statement is true to the best of my knowledge and belief and I make it knowing that, if it is tendered in evidence, I shall be liable to prosecution if I have wilfully stated anything which I know to be false or do not believe to be true.

2. I am 62 years of age.

3. I hereby certify that I am a paediatric neurologist. I have specialised knowledge based on my training study and experience as a paediatric neurologist for the past 30 years. I hold the following qualification:

MBBS, BSc (MED), FRACP. I was formerly Head of the Department of Neurology and Neurosurgery at The Children's Hospital at Camperdown and Westmead, Sydney and I am

currently Head of the Institute for Neuromuscular Research at The Children's Hospital at Westmead. I am the Petre Professor of Paediatric Neurology in the University of Sydney.

4. I am asked to provide an expert opinion in regard to the apparent life threatening event (ALTE) that occurred 4 months prior to Patrick Folbigg's death on 18/10/1990, his seizures which followed the ALTE and his "generalised loss of brain substance" as reported in his file CT scan conducted at the Mater Hospital on 05/11/1990.

5. To my knowledge I have never seen the patient Patrick Folbigg nor any other member of his family. I base my comments on the records provided by Detective Ryan and listed numbered 1-196 in the brief provided to me with his letter of 17/10/02. I have read the documents in question. I have not seen any X-rays or other pathological material relevant to the case. I note from these records that Patrick is 1 of 4 siblings who have died unexpectedly in infancy. One of these, Caleb Folbigg born 01/02/1989 died on 20/02/1989. He was said to have died at 20 days of age of unexplained causes and to have had a "floppy larynx". (Letter of Dr Ian Wilkinson 30/10/1990). I also note reference to the fact that a further two of Patrick's siblings died following his death (Statement of Dr Ian Wilkinson 08/10/1999).

6. The clinical notes with regard to Patrick Folbigg may be summarised briefly as follows. Patrick was born on June 3, 1990, a normal vertex vaginal delivery following a normal pregnancy. The birth weight was 3410 grams at 39 weeks. The 5 minute Apgar score was 8 and there were no apparent neonatal problems. The length at birth was 48.3cm and the head circumference 33.5cm. He was considered to be a normal newborn by the paediatrician, Dr Morris. He was discharged to the care of his parents on June 8. Because of the history of the previous sudden infant death in Caleb, a sleep study was organised for 14 June 1990. This study was carried out during an admission to The Mater Hospital from 14/06/90-15/06/90. The infant was considered to be well by the examining resident. The study did not demonstrate any apnoeic episodes and the periodic breathing was within normal limits for this unit. As a result, the study was recorded as normal (Expert Certificate of David Michael Cooper 6/12/99). The infant Patrick also had a barium swallow on 15/06/1990, the report being: "Apart from some incoordination of swallowing no significant pathology was demonstrated".

   On 18 October 1990 at 06:00, the child was admitted for the second time to The Mater Hospital. The history stated that the infant had been snuffly for the previous 3 days with some vomiting after bottle feeds. He was seen by his mother at 3am because she heard him coughing. At 4:30 she heard him gasping and he was found to be blue around the lips, lifeless, floppy and making minimal respiratory effort. CPR was not performed. He later made a high pitched cry and revived slightly when paramedics administered oxygen about 20 minutes later. Initial examination showed a temperature of 35°PR. Pulse rate 160, respiratory rate 60, blood pressure 90/50, peripheral cyanosis, lethargy, responding only to painful stimuli. Dilated pupils but reactive, pulse oximetry 88% in air. After about 15 minutes he became much more alert and pink in air. He was moving his head freely and arching the back with the comment "that he always does this". There was a large anterior fontanelle, not tense, not bulging. Snuffly nose. CVS:- pulses initially difficult to feel, later good pulses, head circumference 44cm (75^{th} centile), right fundus normal, left not well visualised. Supporting weight on legs, moving all limbs against gravity and resistance. Liver span was 7cm. The chest examination revealed widespread wheezing with scattered crackles. 4+ glucose was noted on urinalysis with a small amount of blood and positive 2+ protein. Fever developed on 19 October. Red cells were noted in the urine and an abdominal ultrasound was normal. Frequent seizures commenced at approximately 21:00 on 19/10/1990.

   An examination by Dr Wilkinson on 18/10/1990 revealed that the infant was "subdued" with little spontaneous movement. Tendon reflexes in the lower limbs were somewhat brisk. The left optic disc was indistinct. Retinal haemorrhages were not observed. Various diagnoses were

considered including viral encephalitis. CSF was normal. CT scan revealed hypodense areas in the temporal and occipital lobes. Chest X-ray showed slight hyperinflation with increased markings. EEG showed left frontal lobe discharges. Urine metabolic screening, rectal biopsy and serum lactate, ammonia, calcium, magnesium and glucose were all normal. The child was treated with phenytoin and phenobarbitone and discharged on 29 October.

The CT scan has apparently been lost but a resident at the time, Dr Joseph Dezordi, recalls having sent the films to Dr DeSilva, then Director of Radiology at The Children's Hospital, Camperdown, who was said to have raised the possibility that the appearance was caused by child abuse. (Statement of Dr Dezordi 17/03/2000, page 5). This evidence was corroborated by Dr DeSilva himself in his Expert Certificate dated 27/04/2001, paragraph 5, in which he comments on two reports of CT scans on Patrick Folbigg dated 23/10/1990 and 05/11/1990: "After reading these reports I can categorically state that the findings described are commonly seen in cases of child abuse from violent shaking injury and associated ischaemic brain damage. In the circumstances described to me, I feel strongly that this child's findings are due to non-accidental injury". The actual report of Dr J Lau for the CT scan done on 23/10/1990 is as follows: "In the pre contrast scant (sic!) there is a decrease in attenuation seen in both occipital lobes, temporal lobe and left frontal lobe. The grey/white matter differentiation is lost. Ventricular system not dilated. No haemorrhage seen. Minimal widening of the peripheral cerebral sulci is seen in the frontal and the parietal lobes.

Post contrast scan with thin cuts over the posterior cranial fossa and temporal lobe shows the hypodense areas involving both posterior parts of the temporal lobes and occipital lobes Abnormal enhancement demonstrated. The intra-cranial vessels are well enhanced. No abnormal fluid collection seen. Impression: The picture is compatible with encephalitis involving both temporal lobes, occipital lobes and left frontal lobe. Herpes encephalitis has to be considered".

Patrick Folbigg was readmitted to the hospital on 4 November 1990 until 10 November with an episode resembling an oculogyric crisis. He was febrile on admission, 40°C, distressed and crying. Soon after arrival the eyes were noted to be deviated upward and to the right – fixed, no jerking. The pupils were unable to be seen. He was not floppy, not stiff and no jerking of limbs or head was noted and he was still able to move the arms and legs. The eyes remained deviated for approximately 1 hour and returned to normal shortly after Panadol was administered. A lumbar puncture revealed a normal CSF with no cells. Rectal biopsy, white cell enzymes, very long chain fatty acids and plasma carnitine were reported as normal. CT scan on 05/11/1990 was reported as showing: "In the pre contrast scan there is mild generalised widening of the subarachnoid space. Ventricular system not dilated. There is some increased density seen in both occipital lobes. The grey/white matter differentiation is intact otherwise. In the post-contrast scan with thin cuts over the posterior cranial fossa, the 4th ventricle is not dilated. Some abnormal enhancement is seen in both occipital lobes, patchy in areas and distributed in both grey and white matter. There is generalised loss in brain substance. The patchy enhancement seen in both occipital lobes could be related to the post inflammatory changes. The high density seen in the pre-contrast scan may be due to dystrophic calcification". Dr J Lai

A further admission occurred on 14/11/1990-22/11/1990. There was a history of worsening rhinorrhoea and cough with little sleep. His mother left him for 5 minutes on a couch. On return the eyes were looking up into the head, there was stridulous breathing and vomit++. The infant was alert on arrival in Casualty. It was noted that the infant did not fix nor follow although he reacted to sound. Further staring attacks occurred during the hospitalisation. An EEG report by Dr J T Holland on 05/11/1990 was as follows: "There is an excess slow on the right in comparison with the left. However, whilst there is independent potentially epileptogenic activity seen in a multifocal nature in both hemispheres, there is one further brief seizure event seen in the left parietal region.

On review there does appear to have been some deterioration in the record since the previous two. Review of the original one is again absolutely normal. The second one, I think, is borderline and this one frankly normal. The picture suggests an ongoing encephalopathic process".

A further admission occurred from 22-23/12/1990 following episodes of prolonged upward gaze without generalised seizures in association with viral illness and a temperature of 39.1°.

On 08/02/1991, a Genny Dwyer reported considerable improvement in visual responses in the previous 2 weeks. On the day in question the infant was fixing, following 180° and reaching towards the visual stimulation.

The infant died on 13/02/1991. According to the expert certificate of Dr Christopher Walker, Director of the Emergency Department at The Mater Hospital, dated 18/01/2000, "The child had been found by the mother some time prior to the ambulance being called that morning (13/02/1991). I was told that Mrs Folbigg had then called her husband at his work in Kotara ... I was also told that Mr Folbigg had driven from his workplace in Kotara to their home in Maylield and had commenced bystander CPR. I was told that this had occurred prior to the arrival of the NSW Ambulance Service at the child's home at 10:10am. The ambulance officers reported to me that on arrival at the home they found the child to be pulseless and not breathing. The child was reported by the ambulance officers as peripherally cyanosed. The child was also reported to have warm skin temperature. Basic life support was continued by ambulance officers. Bag mask ventilation with oxygen and external cardiac compression was performed until arrival at the hospital at 10:18am.

I examined the child on arrival at the hospital. The child was not breathing and received ventilator support with oxygen by hospital emergency staff. The child was placed on an ECG monitor. The monitor showed asystole. .....Resuscitation ceased after 20 minutes and death was pronounced by me at 10:40am on 13 February 1991."

Autopsy was performed on Patrick Folbigg on 13 February 1991. The <u>macrosopic diagnosis</u> was:

1. Normally formed male infant of approximately 8 months of age.
2. Brain and spinal cord fixed for later examination.
3. Hepatic congestion.
4. Congested postero-basal dependant segments – both lungs.
5. Enlarged thymus.

The <u>final diagnosis</u> was: Normally formed male infant of approximately 8 months of age. Old infarcts and gliosis in the parieto-occipital area (both cerebral hemispheres), which are probably secondary to the cardio-respiratory (sic) suffered at about 5 months of age. (Dr J Bishop).

The brain sections were subsequently reviewed by Dr Alex Kan of the Histopathology Department of The Royal Alexandra Hospital for Children who commented, "... I believe that the small amount of linear cortical calcification in the occipital region is just part of the laminar cortical necrosis. I can see no suggestive features of toxoplasmosis or cytomegalo-virus infection, and the distribution of the lesions is unusual for herpes simplex encephalitis and they certainly appear far more likely to be the result of the episode of cardiorespiratory arrest this baby suffered at about 5 months of age".

7. **Comment**: Patrick Folbigg appeared to have been normal at birth and to have been well up until the time of his second admission to The Mater Misericordiae Hospital at Newcastle on 18 October. The clinical history and findings at admission on that occasion, coupled with the early onset of seizures which became intractable would be in keeping with an encephalopathy, due most likely, in my opinion, to an asphyxial episode. This pattern of delayed seizures is common in my experience in acute life threatening events of whatever cause. The first CT scan

description appears to be more in keeping with such an asphyxial insult than with a shaking or striking injury to the head since there was no evidence of intracranial haemorrhage. The subsequent evolution of the case with episodic tonic upgaze, seizures and decrease in visual attention would have been consistent with brain damage suffered during the event leading to the admission on 18/10-30/10/1990. The second CT Scan findings would be in keeping with brain atrophy secondary to necrosis of portions of the brain and disappearance of cerebral oedema resulting from the original cerebral insult. There was no supportive evidence for an underlying metabolic or degenerative disease of the brain. The pathological findings at autopsy would have been consistent with damage due to a serious hypoxic event suffered at the age of 4 months but I cannot exclude the possibility that the findings could have possibly been caused by shaking or trauma since this may sometimes cause apnoea. As to why the visual detcrioration was not noted until the admission of 14-22/11/1990, this may simply have been due to the difficulties of evaluating the visual function in an unwell infant as would have been the case during the admission of 18-30/10/1990 and 4-10/11/1990. It is possible that further asphyxial events had occurred during that time interval but I think that is less likely than a lack of observation. The final event appears to have been a further asphyxial episode without clear explanation.

The most plausible explanation for the series of events is that there was an acute asphyxical event on the morning of 18/10/1990. Such an event could have been a "near miss" SIDS (ALTE) or could have been due to deliberate suffocation of the infant. A series of such events in four siblings with exclusion of other underlying pathological states (such as metabolic disorders, cardiac conditions or epilepsy) would be more likely to be due to deliberate suffocation than any other cause. Furthermore, the failure of the mother to administer cardio-pulmonary resuscitation immediately on discovery of the infant's moribund state on 18/10/1990 and on the day of death argue strongly in favour of deliberate neglect.

I enclose a paper by Constantinou et al. Describing a similar evolution of the clinical picture to that of Patrick Folbigg as seen in several infants with "near miss SIDS" studied intensively in our unit. The reports of the CT scans are similar to those of Patrick Folbigg. I also attach a relevant article by Dr R Meadow (J Paeds, 1990;117:351-7).

Signed

4.11.02

Robert Ouvrier
Encl (2)

I certify that I have read the Expert Witness Code of Conduct contained in Schedule 1 of the District Court Rules. I agree to be bound by the Code. To the best of my ability, this report has been prepared in accordance with the code.

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Hypoxic-ischaemic encephalopathy after near miss sudden infant death syndrome

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SUMMARY Between 1982 and 1985, 14 infants aged 3–26 weeks presented with severe hypoxic episodes as a result of the 'near miss' sudden infant death syndrome (SIDS). They all had metabolic acidosis, cardiovascular instability, acute renal failure, ischaemic colitis, or acute neurological dysfunction. Investigation of the cause excluded infection and trauma, or a primary metabolic, pulmonary, cardiac, or seizure disorder. Seven infants were deeply comatose on admission, never regained consciousness, and died within 60 hours. A characteristic evolution of hypoxic ischaemic encephalopathy not previously clearly described after near miss SIDS was seen in the seven who lived. Five of the seven were conscious within one hour of resuscitation and showed a striking interval of near normality before neurological deterioration that was characterised by status epilepticus, deep coma, and brain stem dysfunction from 36–96 hours after the event. A biphasic course was not apparent in the remaining two, each of whom was comatose on admission, though refractory seizures did develop. Computed tomograms of the brain more than a week after the event showed cortical infarction or cerebral atrophy. Six of the survivors, followed up from 16–55 months, have serious residual deficits including spastic quadriplegia, delayed development, cortical blindness, or infantile spasms.

'Near miss' sudden infant death syndrome (SIDS) refers to the recognised clinical phenomenon of the rescue of an apparently healthy infant from a life threatening event. The infant is found limp, apparently unconscious, not breathing, pale or cyanotic, and requires stimulation or more vigorous resuscitation. Once specific toxic, metabolic, infectious, traumatic, and other identifiable causes for this life threatening event have been excluded, there remains a set of infants who for unknown reasons have experienced an inexplicable episode of near death.[1]

The vast number of publications on near miss SIDS suggests that infants either die or recover rapidly following such an event.[2–5] At most, subtle neurological abnormalities may occur.[6,7] Only a single report suggests that significant neurological dysfunction may occur.[8]

In order to clarify whether near miss SIDS is associated with considerable morbidity, a retrospective review was conducted of infants with a history suggestive of near miss SIDS and clinical evidence of hypoxic derangement of a number of organ systems, including the central nervous system, admitted to the intensive care unit of the Royal Alexandra Hospital for Children, Sydney.

Patients and methods

The case histories of all infants admitted to the intensive care unit of The Royal Alexandra Hospital for Children, Sydney, for the period 1 June 1982 to 30 September 1985 were reviewed to identify those infants who had sustained a serious hypoxic injury as a result of near miss SIDS.

All subjects fulfilled the accepted criteria for the diagnosis of near miss SIDS. They had been found pale, cyanosed, limp or not breathing, and had required vigorous stimulation or cardiopulmonary resuscitation because death was thought to be imminent. In all infants, there was a lack of pathological events during pregnancy and delivery, including prematurity, and their neurological state seemed normal before the life threatening event. There was neither a history of seizures nor of fever.

Evidence of a hypoxic insult during admission consisted of metabolic acidosis (arterial pH 7·0 or less on presentation) or of cardiovascular instability (hypotension—that is, systolic blood pressure less than 40 mm Hg associated with poor peripheral perfusion), acute renal failure (absent or urinary output decreased to less than 1 ml/kg/hour, or serum creatinine concentration of over 100 umol/l),

ischaemic colitis (profuse watery bloodstained diarrhoea with no other bacteriological, toxic, or surgical cause), or acute neurological dysfunction (deep coma or convulsions). Deep coma was diagnosed when the infant did not open his eyes (either spontaneously or in response to noise), made no sound, and made no purposeful response to localise or resist painful stimuli.

Infants were included in the study only if no obvious cause (sepsis, trauma, metabolic disorder, or primary pulmonary, abdominal, or cardiac abnormality) had been found for the life threatening event after extensive investigation during the hospital admission. Apart from detailed physical examination this investigation included full blood count, estimations of the serum concentrations of electrolytes, ammonia, glucose, calcium, and magnesium, liver function tests, urinary amino acid screen, and organic acid profile by gas liquid chromatography. Cerebrospinal fluid was analysed and viral and bacteriological cultures of blood, urine, stool, and cerebrospinal fluid were made. Chest radiography, electrocardiograms, and skeletal surveys were also carried out. Necropsies on those who died failed to establish a cause of death other than the primary hypoxic event.

The hospital records of those who lived were reviewed with particular reference to neurological state in the acute period. Serial electroencephalographic traces and cerebral computed tomograms were also studied. The neurodevelopmental outcome in the long term survivors, followed up clinically by the attending paediatrician, was recorded.

Results

Fourteen infants from 3–26 weeks of age (mean 13 weeks) satisfied criteria for the diagnosis of a near miss SIDS event with a significant hypoxic insult. There were seven boys and seven girls. One child (case 1) has been previously reported and was found to have an incidental stage I neural crest tumour that resolved spontaneously without treatment. Urinary catecholamine excretion was normal.[9] As a comparison over the same period of time 35 other infants were admitted to the general hospital wards having had a near miss SIDS event that was not associated with hypoxic sequelae.

All infants were seen in hospital within 90 minutes of the life threatening event, six of the infants died within 24 hours. A further infant was withdrawn from life support systems after 60 hours because of brain death. Seven lived. All the children had previously been well and had not given cause for medical concern before the near miss SIDS event. Of those who lived, two had been slightly irritable and two had rhinorrhoea for a day or two before admission. Of those who died, two had been slightly irritable and one had had rhinorrhoea.

Of the seven who died, six were judged to have had cardiorespiratory arrest either at home by paramedical personnel, or in a hospital emergency department. Resuscitation consisted of intubation, artificial ventilation, and adrenaline given either intravenously or through the endotracheal tube. Estimated duration of arrest before resumption of cardiac output varied from 10–35 minutes. One other infant who died (case 13) presented with agonal gasping and unrecordable blood pressure. All these infants remained hypotensive and required volume expansion and inotropic support. Four developed signs of acute renal failure, and four ischaemic colitis.

After resuscitation at home all seven who lived had established spontaneous respirations by the time that they arrived in the hospital emergency department. Resuscitation had consisted of vigorous stimulation, mouth to mouth resuscitation or cardiac massage. One infant required resuscitation with a bag and mask (case 7). Three of these infants required intravenous colloid and inotropic support to maintain adequate cardiac output. Three developed ischaemic colitis, and two acute renal failure (one of whom required peritoneal dialysis).

Table 1 shows the evidence of hypoxic derangement during admission in the two groups. Metabolic acidosis on presentation, continued cardiovascular instability, acute renal failure, or ischaemic colitis occurred more often among those who died. In two infants (cases 2 and 4) acute neurological dysfunction was the only evidence of an hypoxic injury. Liver function tests showed a mild increase in the transaminase activity in two cases (cases 1 and 6). Three infants (cases 1, 3, and 4) developed hyponatraemia secondary to inappropriate antidiuretic hormone secretion during admission.

Acute neurological course

All seven who died were in deep coma at their initial presentation and remained so until the time of death. In all but one the pupils did not react to light at initial presentation. Three patients (cases 8, 10, and 13) developed seizures before death.

Two distinct patterns of neurological dysfunction were seen in the seven survivors. All had normal pupillary reactions at the initial assessment; details of their neurological course and outcome are shown in table 2.

Five of the infants (cases 1–5) showed a biphasic evolution of neurologic dysfunction characterised by

Table 1 *Clinical signs of hypoxic insult*

Case No	Sex	Age (weeks)	Initial pH <7·0	Cardiovascular instability	Acute renal failure	Ischaemic colitis
Those who lived:						
1	Female	7	Not measured	No	No	Yes
2	Male	17	No	Yes	No	No
3	Male	26	No	No	No	Yes
4	Female	12	Not measured	No	No	No
5	Male	13	Not measured	No	Yes	Yes
6	Male	4	Not measured	No	Yes	No
7	Female	8	Yes	Yes		
Those who died:						
8	Female	9	Yes	Yes	Yes	Yes
9	Female	3	Yes	Yes	Yes	Yes
10	Female	17	Yes	Yes	No	Yes
11	Male	14	Yes	Yes	Yes	Yes
12	Male	16	Yes	Yes	Yes	Yes
13	Female	11	No	Yes	No	No
14	Male	36	Not measured	Yes	No	No

Table 2 *Features of neurological course and outcome of those who lived*

Case No	Neurological state on admission	Onset of seizures (hours after event)	Onset of deep coma (hours after event)	Duration of deep coma (hours)	Outcome
1	Irritable, did not fix and follow light, abnormal tone, reflexes	2	48	48	Spastic quadriplegia, severe cortical blindness, infantile spasms
2	Awake, alert, active, vision fixed momentarily	36	48	4	Moderate developmental delay, moderate cortical blindness, infantile spasms
3	Alert, active, vision fixed momentarily	51	96	48	Cortical blindness on discharge from hospital
4	Alert at times, occasionally lethargic	11	72	48	Moderate developmental delay, moderate cortical blindness, infantile spasms
5	Lethargic, few spontaneous movements	3	72	120	Spastic quadriplegia, mild cortical blindness, infantile spasms
6	Slow withdrawal to pain	5	At presentation	120	Spastic quadriplegia, severe cortical blindness
7	Floppy, no response to painful stimuli	42	At presentation	96	Mild developmental delay, severe cortical blindness, infantile spasms

an initial period of near normality and subsequent neurological deterioration. These five infants were conscious and rousable within one hour of resuscitation. They were awake, alert and active, or irritable or lethargic with only minor abnormalities of tone and deep tendon reflexes. In three cases there were concerns about their ability to fix their gaze. This striking interval of near normality continued until the onset of seizures from 2–51 hours (mean 21 hours) after the event. In one patient (case 1) isolated myoclonic jerks, evident at initial presentation, did not recur until 18 hours after the event. The seizures (initially tonic or multifocal and clonic) increased in frequency and duration with eventual secondary generalisation from 28–72 hours (mean 51 hours) after the event. Status epilepticus, defined as a generalised convulsion which lasted more than 30 minutes or as serial convulsions between which there was no return of consciousness, occurred in all five.[9] Two patients in this group required an infusion of thiopentone to control the seizures. Associated with increasing severity of the convulsions there was overall deterioration in the infants' neurological condition, with progression to deep coma from 48–96 hours after the event. Deep coma, as defined previously, persisted from 4–120 hours (mean 58 hours). Signs of brain stem dysfunction—that is, no oculocephalic responses

(cases 3 and 4), pupillary inequality (case 1), and divergent eyes (case 5)—occurred in four of the patients in association with deep coma. The fontanelle was bulging in four.

The other two survivors (cases 6 and 7) were deeply comatose on admission. A biphasic course was not apparent though refractory seizures did develop. One of these patients (case 7) also required an infusion of thiopentone.

Cranial computed tomograms were obtained in all except one of the patients (case 6). Early scans (obtained within eight days of the life threatening event) were normal (cases 3 and 4), or showed focal (case 5), or generalised (case 1) hypodensity, suggestive of cerebral oedema. Cranial ultrasound in the first case showed increased density in the subarachnoid space suggestive of blood, but this was not confirmed on computed tomography. Cranial ultrasound of case 6 showed no abnormality. Computed tomograms obtained after the first eight days were normal in only one case (case 3), or showed changes of cerebral atrophy in one patient (case 5) and frontal temporal or occipital low attenuation areas consistent with watershed infarction in three (cases 1, 2, and 7).

Ictal activity on an abnormal background rhythm of low amplitude slow waves was seen in five of the six patients in whom electroencephalographic traces were obtained within one week of the life threatening event. This activity consisted of either rhythmic spike and slow wave resembling paroxysmal lateralising epileptiform discharges in three cases (cases 2, 6, and 7), or rhythmic spikes in two (cases 4 and 5). The discharges characteristically arose unilaterally in alternate hemispheres, or occasionally synchronously in both. The one patient in whom ictal activity was not seen (case 3) was receiving an infusion of thiopentone at the time of the record. Electroencephalographic records after the first week showed persistence of the abnormal background rhythm with the disappearance of ictal activity.

OUTCOME

Six of the seven infants (cases 1, 2, 4, 5, 6, and 7) have been followed up for periods of one year four months to four years seven months (mean two years eight months) and they all show evidence of serious handicap. One child (case 3) was unfortunately lost to follow up, but was said to be developmentally normal one year after the event when reviewed by his local doctor.

Three of the survivors (cases 1, 5, and 6) are severely handicapped by spastic quadriplegia and live in institutions. The remaining three (cases 2, 4, and 7) are mildly or moderately developmentally delayed as assessed with the Reynell-Zinkin Developmental scales for young visually handicapped children and the Maxfield-Bucholz scale of social maturity.

All survivors are cortically blind. Three of them (cases 1, 6, and 7) are severely impaired and have light perception only. One (case 5) can distinguish large objects at one metre, and two (cases 2 and 4) can do so at three metres. Visual function in these two is characterised by visual inattention and variability. Case 3 was cortically blind at discharge from hospital. He was said to have normal vision one year after the event.

Five infants developed infantile spasms with electroencephalographic changes of true or modified hypsarrhythmia.

Discussion

The exact association between near miss SIDS and SIDS is unclear. Most series emphasise a possible overlap between the two (similar age distribution and more male and preterm infants and siblings of SIDS victims than in the general population). Babies referred as 'near miss' infants probably are, as a group, at increased risk for sudden death.[1-4 12-14]

Near miss SIDS may represent a pathophysiological disorder characterised by prolonged central or mixed apnoea, the end result of which may be sudden death.[13 14] It has been suggested that a child either dies from SIDS, or recovers from near miss SIDS after a life threatening apnoea, and that this recovery is complete.[4 5] The literature does not address the possibility that recovery from near miss SIDS is not always an 'all or nothing' event. Significant hypoxic sequelae are not reported.

This study identifies a selected group of infants who were resuscitated from a 'near miss' event and who also showed clinical evidence of hypoxic derangement of at least one, and often many, organ systems. Necropsies showed findings consistent with a diagnosis of SIDS. Careful clinical and laboratory investigations in the surviving infants excluded specific toxic, metabolic, infective, and other causes. All the infants were neurologically normal before the event. The infants did not fulfil the criteria for the diagnosis of the syndrome of haemorrhagic shock and encephalopathy which, in addition to encephalopathy and convulsions, is also characterised by a high fever, severe hepatic dysfunction, and a haemorrhagic diathesis.[15 16]

Mortality in this selected group was high and the survivors developed serious neurological complications. In most of the survivors the acute neurological course started with a normal period, and coma and convulsions developed later. The long term outlook

was poor, with cortical blindness, infantile spasms, and varying degrees of developmental delay.

The findings presented here contrast with those that suggest that infants either die or recover rapidly after a 'near miss' event. By selecting a group with hypoxic derangement, many infants who do recover rapidly have been excluded. It remains surprising, however, that reports of significant neurological deficits after near miss SIDS are infrequent. Aubourg *et al* described 12 infants with status epilepticus in eight of whom a history of an unrecognised life threatening event 36 hours to four days before the onset of epilepsy was retrospectively obtained.[8] These infants shared many features with the babies reported here including an interval of near normality, similar electroencephalographic features, and a high frequency of handicap including cortical blindness, a well known manifestation of hypoxic insult.[10]

Interestingly, Kelly *et al* described a number of infants who required bag and mask resuscitation from a near miss episode. These infants developed transient lethargy, poor fixation on objects, hyperreflexia, and clonus.[4] In addition the same group reported that some infants who had repeated, severe near miss episodes requiring resuscitation or vigorous stimulation later developed a seizure disorder.[11]

The poor prognosis of this selected group of infants is not surprising. Deep coma for even a few hours may be associated with poor neurological recovery in older children.[18] Fixed, dilated pupils, the continued need for cardiopulmonary resuscitation, and severe metabolic derangement at initial assessment all predict a poor prognosis in studies of near drowning.[19 20] Snyder *et al*, however, reported that normal consciousness after an anoxaemic episode and before the onset of seizures is associated with a comparatively favourable outcome.[21 22] It is apparent that in the babies described here the period of near normality after the hypoxic-ischaemic insult was not associated with a good prognosis.

As hypoxic-ischaemic encephalopathy after near miss SIDS has not previously been clearly delineated, the most interesting aspect of this study is the clearly biphasic course of five of the survivors. The interval of near normality before the secondary neurological deterioration is similar in many ways to the early period of hyperalertness that typically gives way to increasing stupor and seizures in severe perinatal hypoxic-ischaemic encephalopathy.[23]

The phenomenon of delayed neurological deterioration after anoxia, first described by Plum and Posner, should be clearly distinguished from the delayed onset of seizures and coma in the infants described in this report.[24 25] The former condition predominantly affects white matter, is thought to be due to demyelination, usually occurs from two to eight days after the anoxic event, and is characterised by pyramidal and extrapyramidal signs and symptoms without seizures. The condition reported in this paper probably reflects acute hypoxic-ischaemic encephalopathy, a triad of multifocal necrosis, vasogenic oedema, and raised intracranial pressure affecting primarily the grey matter.[26]

The results of recent experimental work have suggested that the clinical characteristics of acute hypoxaemic encephalopathy—that is, the hyperalert state and the seizure activity, may relate to hyperactivity of excitatory synapses that are highly concentrated in those regions of the developing brain vulnerable to hypoxic-ischaemic damage. Excessive release of neurotransmitters (particularly glutamate) triggers a cascade of biochemical reactions and potentially lethal ionic shifts with eventual neuronal death taking place over hours or days.[27]

Specific treatment, including glutamate antagonists, may soon become available so that this cascade of biochemical events can be interrupted. Flunarizine hydrochloride, a commercially available calcium channel blocker, has been shown to block the effects of brain perinatal hypoxaemia-ischaemia in an in vivo model and offers exciting prospects for the reduction of the serious consequences of hypoxaemic-ischaemic encephalopathy.[28] It is possible that the early use of anticonvulsants may prevent refractory seizures that coincide with deteriorations in mental state.

Before such treatments are available knowledge of the present poor prognosis of this unfortunate group of infants may be of value as a guide to acute treatment regimens, in parental counselling, and in the anticipation of long term rehabilitation needs.

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ORIGINAL ARTICLES

Suffocation, recurrent apnea, and sudden infant death

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We reviewed the cases of 27 young children from 27 different families who were suffocated by their mothers. The certainty, or near certainty, of suffocation was based on reliable observation or recording of the suffocation, maternal confession, or successful prosecution in a criminal court. Eighteen of the children are alive, although one has severe brain damage; nine are dead. Twenty-four were reported to have had previous episodes of apnea, cyanosis, or seizure, and 11 had had 10 or more such episodes that were either invented or caused by the mother. Repetitive suffocation usually began between the ages of 1 and 3 months and continued until it was discovered, or the child died, 6 to 12 months later. The 27 children had 15 live older siblings and 18 who had died suddenly and unexpectedly in early life; 13 of the dead siblings had had recurrent apnea, cyanosis, or seizures, and, although most of them at the time of death were certified as having sudden infant death syndrome, it is probable that some were suffocated. Repetitive suffocation has a characteristic clinical presentation that should allow identification before brain damage or death occurs. The characteristics should also allow the cause of death of some cases of sudden infant death to be established more accurately. (J PEDIATR 1990;117:351-7)

Suffocation, caused by smothering the child, is considered to be an uncommon form of child abuse and an uncommon cause of childhood death. Despite some reports of the presentation of suffocation as "near miss" sudden infant death,[1-3] the suggestion that a proportion of children with recurrent apnea, or certified as having died from sudden infant death syndrome, have been suffocated tends to produce either outrage or skepticism. This article records the findings relating to families in which suffocation of a child has occurred, the clinical presentation, its relationship to Munchausen syndrome by proxy, and its relevance to other sudden, unexplained deaths of young children.

SIDS Sudden infant death syndrome

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METHODS

The children were considered to have been suffocated with certainty, or near certainty, if one or more of the following criteria were fulfilled:

1. The mother was filmed suffocating her child or she was observed doing so by a reliable, impartial observer.
2. The mother confessed to suffocation and there was strong circumstantial evidence supporting that confession. (For the surviving children, useful corroboration of the mother's confession came from the fact that separation of the child from the mother led to immediate cessation of the child's apneic episodes.)
3. The mother was found guilty in the criminal court of suffocating her child.

Eleven of the cases fulfilled all three criteria, 12 fulfilled two of the criteria, and four had a single criterion.

The families came to my attention from different sources, including consultant pediatric specialists, child care agencies and social service departments, the police, prosecution services, and courts of justice concerning custody of children.

The data include information relevant to the time when the suffocation was discovered. I collected information at the time of my involvement with these cases, which ranged from seven cases for which colleagues consulted me when suffocation was first suspected, to others in which I was involved as long as 8 months after the suffocation had come to light, when the main problems concerned the safest long-term placement for the surviving children or the siblings.

The information was obtained from the medical records, the reports of social service departments and other child care agencies, the affidavits prepared for court purposes, and the statements of witnesses to the police. The covert video surveillance was usually established and monitored by the police surveillance team for the area served by the investigating hospital. Both parents were interviewed at length on at least one occasion, and seven parents had repeated interviews or a follow-up interview 1 to 7 years later. The living children were assessed, as were their siblings in the same home. Further information was obtained from interviews with the physicians, social workers, and police officers involved with the families, and from records of the child care and criminal court proceedings.

RESULTS

The 27 children came from 27 different families living in different part of Britain. Ten girls and eight boys survived the suffocation and are alive, although one girl has severe brain damage. Three girls and six boys are dead.

The clinical features relating to the 18 live children and the nine dead children were similar. Thus most of the data are presented for the combined group of 27 suffocated children: separation into those who are dead and those who are alive is undertaken only when the characteristics of the two groups are different.

Siblings. In the 27 families there had been 33 siblings born before the index child who was suffocated (Table I). The 18 dead siblings, who died suddenly and unexpectedly in early life, included 13 boys: the group of live siblings and half-siblings comprised six boys and nine girls. There was a significant excess of dead boys in both the suffocation and sibling groups ($p < 0.05$).

The 18 siblings who had died suddenly and unexpectedly before the index child was known to have been suffocated had been subject to autopsy and the findings certified as SIDS or findings "consistent with SIDS" (or similar phraseology) for 13 children, asphyxia from choking with vomitus or foreign body in 3, and not ascertained in 2.

Clinical presentation. The age of the child at the time of the initial episode ranged from 2 weeks to 18 months (median 2 months). The age at which the suffocation was discovered, either in a live child or at the child's death, ranged from 2 to 48 months of age (median 9 months).

Twenty-four of the suffocated children were initially seen because of unexplained attacks of either apnea, cyanosis, or seizure or because of "near miss cot death." These episodes had been extensively investigated by methods that included repeated observation in the hospital; electroencephalographic, electrocardiographic, and polygraphic recordings; upper gastrointestinal tract x-ray studies; esophageal pH monitoring; and a wide range of biochemical and other tests. The investigations had not revealed a cause for the attacks. Five children had petechiae about the face or mouth, and another two had bruises on the neck. In at least 14 cases, careful search of the child revealed no external marks of suffocation.

Eleven of the children had had between 1 and 10 such episodes; 13 had had more than 10 episodes, including one child who was believed to have had more than 520 episodes. Twenty-one of the children who were alleged to have recurrent apnea had had apnea alarms issued by pediatricians, but at least 10 had problems when using the alarm, ranging from inexplicable alarm failure and severance of leads to poor compliance and maternal truculence. Two children had had cardiac pacemakers implanted.

Of the children who survived suffocation, five had undergone major surgery as part of the investigation or management of their recurrent bouts of apnea. Four had undergone fundoplication despite the fact that investigations had shown only a minor degree of esophageal reflux. One child underwent four major operations, and one child had severe brain damage as a result of suffocation by her mother, with the result that she now has severe spastic quadriplegia, choreoathetosis, and mental handicap.

Table I. Status of previous siblings of the 27 (index) suffocated children

Index cases	Previous siblings	
	Dead	Alive
18 Live	11	9*
9 Dead	7	6

*Includes two half-siblings.

It was possible to obtain details of the clinical features of 16 of the 18 dead siblings. Most of them had a similar pattern of presentation and features similar to those of the 27 suffocated children. The two groups are compared in Table II. The dead siblings who had had recurrent episodes before death had been investigated in a similarly detailed way. In many cases a working diagnosis of "recurrent apnea," "seizures," or other episodic illness had been made, but the confirmatory evidence for these disorders was weak and the children had not responded in the expected way to appropriate therapy.

Of the 27 suffocated children, 12 had had other disorders that had not been explained satisfactorily despite extensive investigation. The disorders for which they were investigated included unexplained bleeding, vomiting, and failure to thrive. One child had asymptomatic congenital heart disease.

The perpetration. Although most of the children were reported to have recurrent episodes and although the mothers are known to have suffocated the children, it is unlikely that all the episodes reported for each child represented an act of suffocation. Both the clinical findings and the mothers' subsequent confessions indicated that many of the alleged bouts of apnea, cyanosis, and seizure were fabricated. For most of the children, a minority of the episodes were a result of certain suffocation. Typical are the records of a 1-year-old child reported to have had 25 episodes; four were associated with respiratory arrest, collapse, and acidosis, which were caused by suffocation, another three or four may have been caused by suffocation, but it is likely that the others were merely false reports from the mother. However, one mother admitted to more than 30 acts of suffocation on one child. For five of the children it is likely that at least one episode of apnea and seizure was genuine because it was witnessed by an independent observer.

In each case it was the child's natural mother who suffocated the child. It was usual for the mother to use the same method of suffocation each time. The commonest method was occlusion of the nose and mouth with a pillow, pad of material, or hand. The child would be placed on his or her back and firm pressure applied over the face. An alternative position was for the child to be clasped firmly on the mother's lap as she occluded the airways. In a few cases the child's face was pushed firmly into the mother's chest. In four cases, immersion under water, in a bath, was also used; in two cases it is probable that occlusive plastic was sometimes used. The usual site of the suffocation was at home when the mother was alone. In a few cases, suffocation took place at home while the husband was in another room.

Table II. Comparative features of 27 suffocated children and their 18 dead siblings

	Suffocated children (n = 27)	Dead siblings (n = 18)
Age at death or discovery		
Range (mo)	2-48	1-36
Median (mo)	9	3
Children with previous episodes of apnea/cyanosis/seizures	24	13
1-10 Episodes	11	7
>10 Episodes	13	6
Children with no or unknown episodes		
No episodes	3	3
Not known	—	2

Nine children certainly incurred suffocation, and six children probably incurred suffocation, at the hand of their mothers while resident in the hospital with them during the time that the child was undergoing investigation.

The mothers. The mothers were of white European background; their ages ranged from 22 to 35 years (median 25). In all except one case the mother was still living with the child's father, who was unaware (and disbelieving) of the possibility of suffocation. Of the 27 mothers, eight had a history of abnormal behavior, previously presenting themselves repeatedly to hospitals with a variety of disorders that could not be explained despite intensive investigation.

At the time of confrontation with irrefutable evidence of suffocation, most of the mothers made only a limited confession. However, when interviewed after the legal proceedings, and particularly when interviewed several years later, eight of them talked freely about their recollections of the suffocation. They were able to talk about the number of times they had suffocated their child and remembered in considerable detail the days on which the incidents happened and the surrounding circumstances, as well as their own feelings at the time. Five mothers remembered the hatred that they had felt for their child. They did not feel hatred all the time but had "mixed feelings" about their child. The hatred was occasioned by the child's seeming so happy and healthy, when they themselves were miserable or when they had themselves had had such an unhappy childhood. Three had disliked their child because he looked and

behaved like his father, who was causing problems for the mother at the time. Others resented their child because of the limitation imposed on their own social or working life. Seven acknowledged that they were using suffocation as a means of getting into contact with friendly and helpful medical and social services, but when challenged why it was necessary to go to the length of suffocating their child to achieve that goal (when all they needed to do was make up a story of illness for the child), they said that there must have been a stronger feeling of hostility toward the child than they had realized.

None of the mothers was known to have a significant previous criminal record. In the 11 cases in which the mothers were prosecuted and convicted of suffocation, the usual sentence was 2 to 3 years of probation with the order that the mother receive "treatment." Twenty-five of the mothers had been assessed by psychiatrists and psychologists, the usual conclusion being that the mother did not have a treatable mental illness but did have a personality disorder.

The more detailed long-term assessment of both the mothers and the children is not yet complete, but at least 70% of the mothers had had unhappy childhoods and could be considered to have suffered emotional abuse, lack of maternal love was the outstanding feature. At least 25% had suffered physical or sexual abuse as children.

Ten mothers were involved in bizarre incidents about the same time that they were suffocating their child. Such events included the parents' home being broken into and burglarized during the time that the child was being examined in the hospital (three families), two home fires, and anonymous telephone calls, which seven families claimed to have received from people accusing them of having killed or harmed a child. For five families, either the police or child care agencies had received anonymous telephone calls alleging that the child was being harmed, and the agencies had come to the conclusion that it was probably the mother who was making those anonymous telephone calls. Six families had had unusually ostentatious funerals or prolonged mourning in relation to a previous child's death.

Despite some bizarre details, and despite the fact that eight mothers themselves had somatization disorders, most of the mothers did not seem unusual or superficial acquaintance to either their physicians or their neighbors.

Detection. Generally the realization that the illness story might be false, or that the cause of the child's repeated bouts of apnea or cyanosis were caused by the mother, came gradually. Polygraphic recordings in the hospital, which suggested violent obstructive apnea, led to initial suspicion in some cases. Apart from those cases in which the mother was recorded or seen to suffocate the child, helpful circumstantial evidence had come from a study of the incidence of attacks in relation to the mother's presence. In four cases formal statistical tests had been used to show that it was highly improbable that random attacks would occur only in the presence of the mother and that they never, or hardly ever, occurred in the presence of other people. For one child the probability that the attacks had occurred by chance only in the presence of the mother had a p value of 2.8×10^{-10}. Information from other family members was particularly helpful in leading to the truth. In all cases, useful confirmation of the mother's role in the attacks came from the sudden onset of good health and freedom from attacks when the child was placed in an alternative home away from her. After discovery and in the period surrounding court procedures, at least eight mothers threatened to kill themselves; none did, but five mothers took small overdoses of drugs or injured themselves. In the months that followed, two husbands killed themselves.

Management and follow-up. After discovery of the suffocation, it was usual for the children to be taken away from the parents and placed in the care of foster parents. Subsequently child custody proceedings took place and court decisions were made to ensure the long-term safety of the children. Only two children were subsequently placed permanently with their mothers within 2 years of suffocation. However, at least eight other mothers were allowed continued custody of other children, including two subsequently born infants.

Seven children who were suffocated when they were older than 6 months of age subsequently had intense fear of circumstances associated with the suffocation—for instance, having a pad placed on their face, being put in a bath, or going into a toilet cubicle. There were examples of children who were still frightened by the circumstances 2 years later.

DISCUSSION

In the past decade there have been a number of reports of suffocation of young children by their parents, usually by smothering. My involvement with such cases stemmed from a study of factitious illness in childhood (Munchausen syndrome by proxy),[3] particularly its most common presentation—factitious epilepsy.[4] Within these families there was an unexpectedly high incidence of sudden death of other siblings. Further investigation led to the conclusion that some of these had been suffocated.

The cases presented in this paper demonstrate some of the features of children who have been suffocated. For 18 of the 27 children the suffocation was discovered early enough for them to survive (although one has severe brain damage). Comparison of the features of the dead and live children showed that they were similar in most respects; thus it is likely that some of the live ones would be dead if the suffocation episodes had not been detected. The fact that 18 of

their previous siblings had died suddenly and unexpectedly in early life is a cause of great concern. It could be that the tragedy of a natural sudden, unexplained infant death causes a mother to behave unnaturally toward her next child and to start suffocating him. Alternatively, some of the dead siblings may have been killed, probably by suffocation, by their mothers; indeed, some of the mothers have confessed to the deaths and have described the circumstances with a degree of detail that is unlikely to be false. However, confessions have to be viewed with circumspection, so it cannot be certain what proportion of those dead siblings died naturally and how many died because of filicide.

For most of the dead siblings, and for some of the dead index children, the initial diagnosis was SIDS. Therefore it is worth comparing the features of suffocation in this series of children with the usual features of sudden infant death syndrome.[5-7] Table III shows important differences: the classic features of SIDS are the young age, the previous good health of the child, the lack of previous apnea or other illness, and the rarity of a positive family history. The finding in Australia of a subgroup of families (who had had a previous child die of SIDS) who had a 10 times increased chance of another such death demonstrated the need to check multiple deaths with great thoroughness.[8,9] Recurrent "cot death" is more likely to be an environmental problem than a genetic one; there is no increased risk in first cousins or in monozygotic compared with dizygotic twins.[10]

The differences shown in Table III are of limited value in that they apply only to this particular group of suffocated children. In most cases the suspicion of suffocation arose because no satisfactory cause for the apnea was found—recurrent apnea in a previously healthy child in the absence of viral infection or of cardiac, respiratory, metabolic, or neurologic abnormality is rare. For some, the unsatisfactorily explained death of a previous sibling led to suspicion. It will always be more difficult to detect suffocation when it occurs for the first time within a family, and particularly if it is the result of a single act rather than repetitive acts.

Although suffocation may cause obvious signs such as petechial hemorrhages, it can occur to an extent causing brain damage and death without any incriminating findings on examination or at autopsy. Therefore it will always be difficult to assess the number of deaths currently classified as SIDS that have been caused by the parents. Careful clinical and pathologic investigation, combined with laborious psychosocial studies of families, has disclosed that a significant proportion of children previously labeled as having died of SIDS are likely to have been killed by their parents.[11-16] The most authoritative review of this difficult area was by Emery,[17] who estimated that the proportion in the United Kingdom is greater than 1 in 50 but less than 1 in 10. It is likely that the proportion varies in different countries, different ethnic groups, and different cultures, and also that it varies from time to time. This current report provides qualitative information but does not allow quantitative conclusions.

Table III. Features of the 27 suffocated children, compared with the incidence of those features in children considered to have died of SIDS

	Suffocation (%)	SIDS (%)
Previous apnea	90	<10
Previous unexplained disorder	44	<5
Age more than 6 mo	55	<15
Dead sibling	48	2

Data from: Froggatt P, Lynas M, Marshall TK (Am J Cardiol 1988;22:657-68); Brooks JG (Am J Dis Child 1982;136:1012-3); and Hoffman JH, Damus K, Hillman L, Krongrad E (Ann NY Acad Sci 1988;533:13-30).

Roser et al.[18] published the first account of the use of video recording in the hospital to prove that a mother was suffocating her child. Covert video recording has been used in several centers, and although it has both ethical and practical problems, it provides proof that a mother is suffocating her child.[19-21] It also reveals the violence that suffocation entails. Infants and young children struggle hard when their airways are blocked; the mothers have to lean on them with force. The struggling continues for 2 to 3 minutes, until the child becomes unconscious, unless there is intervention. Video recording probably has a low degree of sensitivity because it depends on the mother's suffocating her child during the period of close observation in the hospital; most child abuse, including suffocation, occurs at home. The cases reported here came to my attention largely because of a long-standing interest in factitious illness,[22] and therefore the character of these suffocations is more likely to be slanted toward a presentation as Munchausen syndrome by proxy. It does not necessarily mean that all suffocations take place in this way; case reports confirm that some deaths from suffocation are the result of a single act of suffocation.[2,23] More than half of the mothers in this study took their child repetitively to their physicians because of recurrent episodes of apnea or seizure. That picture is characteristic of Munchausen syndrome by proxy, which usually begins with an illness invented by the mother and progresses to false signs fabricated by her.[2] However, in most cases of Munchausen syndrome by proxy, the mothers do not directly harm the child, whereas suffocation, like poisoning, is an example of the mother's causing direct harm to the child. If these mothers merely wanted contact with physicians or wished for their child to be in the hospital, they did not have to occlude the child's airways; they could have persisted with a false story of seizures or respi-

ratory arrest, and most physicians would have believed them. In later interviews some mothers have admitted that they knew at the time that they did not have to suffocate the child to attract a physician's attention; yet they did so, and some of them remembered the hatred that they felt for their child at the time. Those who deal with mothers who have young children need to remember the normal mixed feelings that many mothers have for their children. They may love their child most of the time, but there are occasions when they hate their child, and for some unfortunate mothers the extent of that hatred may be deep and its duration long. We need to give parents the chance to discuss hostile feelings when they are having difficulties with their young children; the subject should not be taboo, because discussion can be therapeutic.

There are few reports of suffocation of children by men[24]; most case reports of suffocation incriminate the natural mother. There are two reports of women who suffocated unrelated children for whom they acted as temporary caretakers.[25, 26] The gender incidence in this series is similar to that found in a survey of recurrent obstructive apnea in which 22 children were probably suffocated by the mother and only one by the father.[27] As is often the case in Munchausen syndrome by proxy, the emotional absence and lack of involvement by the father at times amounted almost to passive collusion. A more involved, thoughtful, and supportive husband would have questioned the events, talked more with the mother's relatives and friends, and sought interviews with physicians.

General aspects concerning the management of factitious illness have been considered elsewhere.[28] One of the more difficult aspects of the management of these families has concerned the future care of suffocated children once the abuse has been discovered, and also the safest care for other children and any subsequent newborn child. The agencies concerned have taken varying courses of action. Some have thought that the abuse was so gross and so violent that it must signify a degree of breakdown in the parent-child relationship that makes it unsafe for the child to be with that mother again. Others, on hearing the mother's full confession and sometimes after a change in her life, such as a new partner, a new job, or new help, have believed that she should be given the chance to look after that child or a new baby again. Although one understands the view that such mothers may "continue this killing unless stopped or until she runs out of children,"[19] it is not necessarily so, and as with other forms of child abuse, the mother may be capable of satisfactory care for a subsequent infant despite having harmed another. Only two of the children reported in this article were reunited with their natural mothers in the short term.

The features described should enable some cases of suffocation to be detected earlier. Awareness not only should stop deaths but may also prevent brain damage and many terrifying ordeals for young children. The acceptance that death by suffocation sometimes masquerades as SIDS should lead to reevaluation of the use of the term and of the most appropriate investigations for infants who die suddenly and unexpectedly in early life. It would be unfortunate if the label "sudden infant death syndrome" became a barrier to the sensible and sensitive investigation of the death of young children. It ought to be possible to safeguard the welfare of infants as well as the feelings of vulnerable and innocent parents.

ADDENDUM

Since submitting this article, I have been involved with the case of a baby boy who had recurrent bouts of apnea from the age of 3 weeks until the age of 8 weeks, at which time the father was filmed while occluding the child's airways during a visit to the child in the hospital. The child has had no further bouts of apnea since being separated from his father. The father has a long history of false stories and self-injurious behavior. A previous child of the father died unexpectedly at the age of 5½ months; that male infant had also initially had unexplained recurrent apnea. A further example of a father's suffocating a baby was published recently (Pediatrics 1990;85:370-3). Therefore, although most cases of suffocation involve the mother, it is clear that sometimes the father is the perpetrator.

I am grateful to the many colleagues who referred these families to me, and to Drs. Christopher Bools and Brenda Neale for their help with recent cases.

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"A"

MEDICAL REPORT

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ON

CROWN -V- KATHLEEN MEGAN FOLBIGG

NEW SOUTH WALES SUPREME COURT

PREPARED AT THE REQUEST OF:
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SOLICITOR
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YOUR REF: 02C022602 PK:CM

By

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Regius Professor of Forensic Medicine
& Clinical Forensic Examiner.

Report dated: 06th November 2002.

RE:
CROWN -V- KATHLEEN FOLBIGG **NEW SOUTH WALES SUPREME COURT**

1. INTRODUCTION:

1.1 This report was compiled on the instructions of PETER KRISENTHAL ESQ., SOLICITOR, Criminal Indictable Section, Level 1, 323 CASTLEREAGH STREET, SYDNEY, N S W 2000, AUSTRALIA.

1.2 My qualifications and experience are as indicated hereunder and in the attached note.

1.3 I understand that I have a duty to the Court and I have complied with that duty. I believe that what I have stated in this report is true to the best of my knowledge and belief, and the opinions expressed in it are correct..

1.4 In the preparation of this report I had access to the following documents:

a. A letter of instruction from the solicitors indicated above.

b. A copy of the SUMMARY OF BRIEF.

c. Copy of an autopsy report on CALEB GIBSON FOLBIGG - 09/05/89.

d. A folder bearing the medical records of CALEB GIBSON FOLBIGG.

e. Copy of an autopsy report on PATRICK ALLAN FOLBIGG – 14/02/91.

f. A folder bearing the medical records of PATRICK ALLAN FOLBIGG.

g. Copy of a neuropathological report on the brain of PATRICK ALLAN FOLBIGG – 24/06/91.

h. Copy of an autopsy report on SARAH KATHLEEN FOLBIGG – 25/11/93.

i. A folder bearing the medical records of SARAH KATHLEEN FOLBIGG.

j. Copy of a neuropathological report on the brain of SARAH KATHLEEN FOLBIGG – undated.

k. Copy of an autopsy report on LAURA ELIZABETH FOLBIGG – 26/07/99.

l. A folder bearing the medical records of. LAURA ELIZABETH FOLBIGG.

m. A written medical opinion by Professor Roger Byard on this case.

2. Purpose of the report

To peruse all the documentary information listed above and to provide an expert pathological opinion on the manner and causes of death in the Folbigg siblings.

3. History

3.1 Accused

NAME: **KATHLEEN MEGAN FOLBIGG.**

3.2 DECEDENTS

- **CALEB GIBSON**

D.O.B.: 01.02.89

Died at the age of 19 days.

- **PATRICK ALAN DAVID**

D.O.B.: 03.06.90

Died at the age of 8 MONTHS

- **SARAH KATHLEEN**

D.O.B.: 07.08.92

Died at the age of 10 ½ MONTHS

- **LAURA ELIZABETH**

D.O.B.: 07.08.97

- Died at the age of 19 MONTHS

4. PRELIMINARY GENERAL COMMENTS

4.1. Reputable and experienced pathologists carried out the post-mortem examinations and the autopsy reports provided are of a good quality.

4.2. A number of appropriate ancillary tests were carried out and indeed some neuropathological examinations.

4.3. I am also aware that full histological examination was carried out on samples of the internal organs.

4.4. It must be stated from the very outset that it is extremely unusual and quite unprecedented to have four deaths of siblings in the same family occurring in succession over a period of eight years. I have never seen or heard of this occurrence before in over thirty years of practice in pathology.

4.5. The possibilities to be considered in this context are:

A. that all four deaths were the result of natural causes, albeit different causes in each individual death or the same cause repeated in all the deaths – in which case the possibility has to be

considered of a congenital intrinsic inherited anomaly of internal metabolism, which all these children exhibited at different neonatal ages and which went undiagnosed through the investigation of all these deaths.

B. That the deaths were associated with some form of induced or imposed airways obstruction leading to the death of these children in succession from a lack of oxygenation of the brain, i.e. cerebral hypoxemia, given that such a mode of death may not leave behind many tell-tale signs of its occurrence even in a very thorough post-mortem examination. Induced airways obstruction may show no features at autopsy, or indeed features, which are similar to those, found in S I D S – the sudden infant death syndrome or cot death.

4.6. Episodes of induced upper airways obstruction, i.e. attempts at suffocating the child by some form of air-impervious cover applied over the mouth and nose areas of the face - are likely to lead to internal bleeding within the substance of the lung, which results from internal damage to the fine structure of the lungs as the baby tries very hard breathe against resistance. If the child dies immediately, this is seen at autopsy; if the child survives for a period the presence of the blood-haemoglobin pigment later becomes broken down and remains for a lengthy period thereafter in the lungs as the derived breakdown pigment known as haemosiderin which can be subsequently

identified by specific tissue staining techniques in the lungs – the Prussian Blue reaction.

4.7. Such episodes would be associated with a struggle to breathe and very vigorous attempts by the baby referred to occasionally as the 'air hunger reflex'. This will be noted at the scene where this takes place in the shape of disturbances of clothing, bedclothes, etc.

4.8. In S I D S deaths, although no signs of disease are found, one would expect to find a small number of internal, pin-point sized areas of haemorrhage in soft unsupported tissues; these are known as petechiae and may be found over the lining of the thymus gland, the pleura lining of the lungs and the pericardium of the heart. These findings would assist the pathologist in reaching a positive diagnosis of S I D S, and in their absence one is more careful to directly attribute the death to this syndrome.

5. Consideration of the death of CALEB GIBSON

5.1 He was found dead in his bassinette by his mother aged 19 days at 02:50 hours. She had fed him at 01:00 hours.

5.2 No investigation of the scene of death was made.

5.3 He was said to have died of the Sudden Infant Death Syndrome [SIDS]; no other morbid anatomical features were found. This diagnosis appears to have been based on the absence of any other pathological abnormalities that could be found at autopsy

5.4 No evidence of any trauma or of neglect were found.

5.5 Toxicology was negative.

5.6 His brain was not submitted to neuropathology.

5.7 The autopsy failed to reveal any infection.

5.4 Clinically soon after he was born he was said by Dr. Barry John Springethorpe to have a floppy or 'lazy' larynx; this matter was not investigated further at autopsy in particular by specific microscopic examination of the voice box [larynx], in particular to exclude the condition known as *laryngomalacia* in which the gristle [cartilage] that makes up the voice box is less substantial and softer than normal due to a congenital anomaly.

5.5 S I D S is rather unusual at this young age and indeed in the presence of a condition indeed known to, and actually, thought to be giving rise to respiratory obstruction on and off in life, this death should have been better classified as 'undetermined' – sudden infant death.

5.6 There was no suggestion in life or at autopsy of any internal metabolic problems in this child and genetic studies after death did not show – though completely exclude - such an anomaly.

5.7 Special stains showed the presence of haemosiderin within the lungs of this child, which raises the question of imposed upper Airways obstruction; haemosiderin may appear within days of such imposed upper airway obstructions.

5.8 It is debatable whether the laryngomalacia would result in episodes of upper airways obstruction given that the paediatrician noted the presence of 'stridor' i.e. airway obstruction.

5. 9 IN SUMMARY

- **This death should not have been attributed to S I D S.**
- **There was a congenital clinically-diagnosed but not pathologically confirmed condition which could have led to upper airways obstruction.**
- **The presence of some HAEMOSIDERIN in the lungs of this child raises the possibility of imposed airways obstruction.**
- **Imposed airways obstruction cannot be completely excluded.**
- **No other metabolic congenital anomaly was found in this child.**

6. Consideration of the death of PATRICK ALAN DAVID

6.1 He was found death in his cot at the age of 8 months.

6.2 No detailed and expert examination of the scene, where the death occurred, was conducted.

6.3 There was an episode of previous collapse, which became associated with severe brain damage due to prolonged a cardio respiratory arrest, resulting in the occurrence of seizures [fits, convulsions] as an after-effect.

6.4 He was treated for these seizures with drugs [anticonvulsants] and these were being taken as prescribed as confirmed on toxicological analysis of his blood taken at autopsy.

6.5 The brain was submitted for neuropathological examination and showed foci of scarring, depletion in size [atrophy] and focal inflammation. These changes could have been the result of a healed viral inflammation of the brain – at one time thought to be due to *Herpes simplex* virus or could have been due to oxygenation depletion due to some unspecified cause, or due to a combination of both.

6.6 These changes could also possibly have been the result of diffuse trauma of the 'diffuse axonal injury' type which would result from a shearing type injury to the brain as may occur occasionally with vigorous shaking. In such instances, subdural haematomas as well as retinal and choroidal haemorrhages are quite frequently found in the eyes, and none were present in this instance. There is nothing specific in the clinical history of this child to indicate that a shaking injury had occurred in him.

6.7 Induced or imposed airways obstruction may lead to brain damage of a diffuse type but the distribution in such instances would be quite specific and typical of cerebral hypoxia or hypoxaemia. It does not appear to be the case from the information available that the distribution of the brain changes in this case was of this variety.

6.8 Given his medical history, this child could therefore have died acutely as a result of an epileptic fit, this resulting in sudden damage to the

vital brain centres in the brain stem with cessation of breathing and of heart function or a secondary effect on the heart with problems with the heart beat [tachyarrhythmias]. This would not have produced changes, which could be specifically identified at autopsy, except perhaps of damage to his lips or tongue in the course of the convulsion. Given that this child would have been in nappies; it would not be possible to discover if he has been incontinent as would have been expected in a generalised fit.

6.9 The blood levels of anticonvulsants at the time of the autopsy were within therapeutic ranges but this does not mean that the convulsions were fully controlled.

6.10 No features of mechanical asphyxia were recorded e.g. petechial haemorrhages in the face, eyes, etc.

6.11 **IN SUMMARY**

- **This death should not have been attributed to S I D S.**
- **It should not have been attributed to asphyxia in the absence of typical asphyxial signs at autopsy.**
- **There was a brain condition, which could have given rise to serious life-threatening convulsions, and death could have occurred in the course of these convulsions.**
- **The diffuse generalised focal brain damage present could have been the result of a viral infection of the brain, which has healed and it would be almost impossible to specifically identify this cause weeks later - an encephalitis. This disseminated brain damage could also have resulted**

from depletion of the oxygen supply to the brain, and therefore imposed upper airways obstruction lasting for a period of minutes.

- **It is unlikely that this brain damage resulted from a shaking injury.**
- **No congenital metabolic problem was conclusively shows to be present in this child.**

7. Consideration of the death of Sarah Kathleen

7.1. She was found dead by her mother at 01:30 hours in her cot.

7.2. Her death was registered as being due to S I D S.

7.3. She recently was treated with antibiotics for a 'bad cold'.

7.4. No features of asphyxia were found.

7.5. There was no evidence of trauma.

7.6. Some internal petechiae were found.

7.7. No morbid anatomical cause of death such as pneumonia was found.

7.8. Although her lungs were water-logged [oedema] and congested – engorged with blood – posteriorly, this does not have much specific significance.

7.9. She was supposed to have been monitored with a breathing monitor as a sibling of a SIDS death, but this had been stopped weeks earlier.

7.10. Some bacteria – especially important being *Staphylococcus aureus* – were isolated from her airways at autopsy. This bacterium is not infrequently found in S I D S.

7.11 There was a suggestion that an usually congested uvula, i.e. the central pendulous structure situated at the back of the palate. Because it was engorged with blood and swollen, this may have given rise to some upper airway obstruction.

7.12 The internal features associated with S I D S were not found.

7.13 IN SUMMARY

- **No anatomical or other cause of death was found.**
- **This death approximates most of the four death being reviewed, a typical death from S I D S.**
- **The presence of the congested uvula may have produced some upper airway obstruction.**

8. Consideration of the death of Laura

8.1. There was a history of a recent upper respiratory tract infection.

8.2. In addition the heart showed inflammatory change which indicate that there was an abnormality of the heart muscle most likely caused by a virus which may have set off an abnormal fast rhythm of the heart and led to sudden death. No viral studies had been conducted at the time of the autopsy and thus

one cannot sure whether or not this heart damage was due to a virus and if so which virus its was.

8.3. It has to be conceded totally that this may be an entirely incidental finding, other babies will have had such a heart muscle infection in association with other viral conditions but somehow grow out of spontaneously and occasionally do not come to grief because of its presence.

8.4. No evidence of trauma or neglect was seen.

8.5. There were no other anatomical cases of death.

8.6. **IN SUMMARY**

- **This death should not have been classified as S I D S.**
- **There is a myocarditis which although may be completely incidental could also have caused serious heart problems and even death acutely and unexpectedly.**
- **This condition could not have been induced by imposed airways obstructions of this child either recently prior to death or previously.**

9.0 FINAL CONCLUSIONS

9.1 It is frankly surprising and disconcerting that these deaths occurring as they did in succession within the same family were not individually investigated more thoroughly and meticulously at the time.

9.2 Histological and other investigations were done but these were somewhat limited and initially restricted in their scope. In particular there was no detailed 'scene of crime' examination.

9.3 As far as one can ascertain, there was **no** congenital metabolic abnormality demonstrated in any or all of these children that could have caused them to die suddenly and unexpectedly.

9.4 These deaths are **not** all due to S I D S, and with exception of the third death other conditions, which could life-threatening, were present and should have been taken into consideration by the pathologist and by the Coroner in coming to an eventual cause of death.

9.5 It certainly **cannot** be said, indeed beyond reasonable doubt, that these deaths were irrefutably due to imposed or induced airways obstruction, as by suffocation.

9.6 In three of these deaths such a possibility should have carefully considered on pathological grounds in the differential diagnosis as one possibility among many; it certainly is **not** the only possible explanation for these deaths because of the presence of other physical disease which could have caused sudden unexpected death.

Professor Anthony Busuttil
OBE, MOM, MD, FRCPath, DMJ(Path), FRCPE, FRCPG, FRCSE.
Regius Professor of Forensic Medicine & Clinical Forensic Examiner.

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SHORT
CURRICULUM VITAE

OF

ANTHONY BUSUTTIL

O.B.E., M.O.M., K.H.S., M.D., F.R.C.Path., D.M.J.(Path.),
F.R.C.P. (Edinburgh), F.R.C.P. (Glasgow), F.R.C.S.E., F.R.S.S.A.

Regius Professor of Forensic Medicine and Head, Forensic Medicine Section, Department of Pathology, University of Edinburgh.

Hon. Consultant Pathologist – the Lothian University Hospitals N H S Trust at Royal Infirmary, Edinburgh.

Hon. Consultant Pathologist - Borders N H S Trust at Borders General Hospital, Melrose.

Clinical Forensic Examiner to the Lothian and Borders Police Force in Edinburgh.

QUALIFICATIONS

M.D. 1967 - Royal University of Malta
(fully registered with the General Medical Council [U.K.] - 1969)

M.R.C.Path. 1973 - Royal College of Pathologists

F.R.C.Path. 1985 - Royal College of Pathologists

D.M.J.(Path.)	1981 - Worshipful Society of Apothecaries - London
F.R.C.P.(Edin.)	1990 - Royal College of Physicians of Edinburgh
M.R.C.P.(U.K.) (qua physician)	1991 - Royal College of Physicians and Surgeons of Glasgow
F.R.C.P. (Glasg.)	1993 - Royal College of Physicians and Surgeons of Glasgow
F. R. S. S. A.	1995 Royal Scottish Society of Arts and Science
M.O.M	1998 – Membership of Order of Merit Awarded by President of Malta.
O.B.E.	2000 – Officer of the Order of the British Empire Awarded by Her Majesty the Queen.
F. R. C S.(EDIN).	2000 – Royal College of Surgeons of Edinburgh

PRESENT APPOINTMENTS

1988 – present Regius Chair of Forensic Medicine - University of Edinburgh.

1981 Police Surgeon [Clinical Forensic Examiner]in Edinburgh with the Lothian and Borders Police Force – to date.

PROFESSOR ANTHONY BUSUTTIL

In addition to carrying out investigations and autopsies in sudden, unexpected and suspicious deaths on the instructions of the Procurators Fiscal in the Lothian and Borders area of Scotland for several years, Professor Anthony Busuttil, Regius Professor of Forensic Medicine at University is also a Clinical Forensic Examiner to Lothian and Borders Police. In this capacity he has direct responsibility for the medical care and welfare of prisons while in police custody and still regularly performs these duties as part of a roster.

In 1986 conducted the pathological investigation that occurred in the aftermath of the Lockerbie disaster

In October 1992, at the request of the Government of the Netherlands, he took part in the investigation following the air disaster in Amsterdam.

In 1996 he directed the pathological aspects of the investigation related to the major shooting incident at Dunblane Primary School.

In his capacity of Police Surgeon (Clinical Forensic Examiner), he has set up with the Community Paediatricians and the local Social Work and Education Departments, the Joint Investigative Procedures and drafted the multi-disciplinary Guidelines in current use and in their regular update. He has set up the four local Video-Colposcopy examination suites run jointly with the Lothian and Police. He regularly examines children of all age groups in which allegation of physical or sexual abuse have been made, produces reports for the Courts and regularly almost weekly basis, gives evidence in court in such cases, both in Scotland and elsewhere in Britain. He is often asked to give 'second opinions' on cases.

He is responsible for many publications on an extensive range of pathology and forensic medicine topics (about 400), has written several chapters in forensic pathology textbooks and published the book entitled *'Suspicious Death Scene Investigation'*. He has actively participated in Home Office and ACPO(S) sub-committees and also formed part of a working party advising the Home Office on major disasters. He is finalising the production of two major multi-author textbooks entitled *'Paediatric Forensic Medicine'* and *'Forensic Medicine and Pathology'*.

He forms part of the editorial boards of a number of Clinical and Pathological international medico-legal journals

He lectures regularly at The Scottish National Police College at Tulliallan in Fife, at Bramshill National Police College, and to police forces throughout Great Britain.

He is the past Chairman of the European Council for Legal Medicine, Editor of the journal ' Proceedings ' of the Royal College of Physicians of Edinburgh and on a variety of other editorial boards. He is asked to testify very regularly in the Scottish and other Courts both for the prosecution and the defence.

His research interests include the ' Sudden Infant Death Syndrome ', fatal house fires, suicidology and the effects of H I V infection on the nervous system.

RECENT PUBLICATIONS

1. Bacterial toxins and sudden unexpected death in a young child. AJ Bentley, AA Zorgani, CC Blackwell, DM Weir and **A Busuttil**. Forensic Science International 1997 (88): 141-146.

2. Medico-legal considerations of deaths from watersports among Caribbean tourists. JO Obafunwa, S Bulgin, and **A Busuttil**. Journal of Clinical Forensic Medicine 1997 (4): 65-71.

3. The use of air weapons in attempted suicide. G. Campbell-Hewson, CV Egleston and **A Busuttil**. Injury 1997 (28): 153-158.

4. Homicidal tandem bullet wound of the chest. AJ Bentley, **A Busuttil**, B. Clifton and P Sibbald. The American Journal of Forensic Medicine and Pathology 1997 (18): 56-59.

5. The skeletonised body: Suicidal inhalation of motorbike exhaust. **A Busuttil**, GH Moody, JO Obafunwa, C Dewar and M McIntosh. The American Journal of Forensic Medicine and Pathology 1997 (18): 50-55.

6. Timing of paediatric deaths after trauma. JP Wyatt, L McLeod, D Beard, **A Busuttil,** TF Beattie and CE Robertson. British Medical Journal 1997 (314): 868.

7. The Joint Paediatric-Forensic Examination in Child Abuse. Mok JYQ, **Busuttil A**, Hammond HF. Child Abuse Review 1998; 7: 194-203.

8. Non-accidental Injury Inflicted on a Child with an Air Weapon. Campbell-Hewson GL, D'Amore A, **Busuttil A**. Medicine Science Law 1998; 38 No.2.

9. Motorcycle accident deaths in southeast Scotland. O'Donnell J, Wyatt JP, Beard D, **Busuttil A**. Journal of A/E Medicine 1998; 15: No.2.

10. The use of the absolute refractory period in the estimation of early post-mortem interval. McDowall KL, Lenihan DV. **Busuttil A**, Glasby MA. Forensic Science International 1998; 91: 163-170.

11. Lockerbie and Dunblane: Disasters and Dilemmas. **Busuttil A**. Medico-Legal Journal 1998; 66 (part 4): 126-140.

12. Fatal falls down stairs. Wyatt JP, Beard D, **Busuttil A.** Injury, International Journal of the Care of the Injured 1999; 30: 31-34.

13. A comparison of fatal with non-fatal knife injuries in Edinburgh. Webb E, Wyatt JP, Henry J, **Busuttil A.** Forensic Science International 1999; 99: 179-187.

14. Pyrogenic Toxins of *Staphlococcus aureus* in S U N D S in adults and older children Al Madani O, Gordon A., Raza N, Weir R. **Busuttil A,** Blackwell CC.. FEMS Immunol Med Microbiol. 1999, 25, 207.19.

15. Detection of IgA antibodies in human milk that bind to bacterial toxins in SIDS. Gordon A., Al Madani O, Raza N W R. **Busuttil A,** Blackwell CC. FEMS Immunol Med Microbiol. 1999, 25, 175-182.

16. The protective effect of human milk in SIDS Gordon A., Al Madani O, Raza, Weir R. **Busuttil A,** Blackwell CC. FEMS Immunol Med Microbiol. 1999, 25, 189-193.

17. Cortisol levels and control of the inflammatory responses to TSST-1. Essay SD. Raza MW,, Gordon A., Al Madani O, Weir R. **Busuttil A,** Blackwell CC. FEMS Immunol Med Microbiol. 1999, 25, 183- 189.

18. The effect of erect posture on nasal temperature in children in relation to induction of staphylococcal toxins implicated in SIDS. Molony N, Blackwell CC, **Busuttil A.** FEMS Immunol. Med, Micorbiol, 1999, 25, 109-114.

19. Use of the Abbreviated Injury Scale to identify preventable deaths. Wyatt JP, Beard D, **Busuttil A** 1999, Amer J For Med Pathol **20,** 102.

20. Missed resuscitation opportunities in drowning victims. Wyatt JP, Tomlinson GS, **Busuttil A** Resuscitation, 2000, 41, 101-4.

21. Suicidal high falls. Wyatt JP, Beale JP, Graham C A, Beard D, **Busuttil A.** J Clin For Med. 2000, 7, 1-5.

22. Epidemiology and prevention of pedestrian deaths in S E Scotland. Wyatt JP, Martin A, Beard D, **Busuttil A.** J Accid Emerg Med 2000, 17,65-71.

23. Deaths from blunt cardiac injury. Wyatt JP, Mills S E, Gardner D S, **Busuttil A.** J Clin For Med. 2000,**7**, 1-5.

24. Autokabalesis in S E Scotland. Graham C, Beale JP, Wyatt JP, Beard D, **Busuttil A.** J Accid Emerg Med. 2000; **17,** 62-7.

25. Deaths among car drivers and their passengers. Parris M, Wyatt JP, Beard D, **Busuttil A.** J Accid Emerg Med. 2000; **17,** 449-500.

26. Plastic bag asphyxia in S E Scotland. Jones LS, Wyatt JP, **Busuttil A.** Amer J For Med Sci 2000.

27. Pedestrian deaths following collisions with heavy goods vehicles. Wyatt JP, Martin A, Beard D, **Busuttil A.** Med Sci Law 2000.

This and the preceding 21 pages is the annexure marked "A" referred to in the attached affidavit of PETER Gerard KRISENTHAL sworn before me at Sydney this FOURTEENTH day of NOVEMBER 20 02.

~~A Justice of the Peace.~~
Solicitor.

the children's hospital at Westmead

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Ref: JC:Culverresids
18th February 2003

Ms J Culver
Crown Prosecutor
Crown Prosecutors' Chambers
Locked Bag A8
SYDNEY SOUTH NSW 1232
Fax # 9261 1485 or 9264 6975

Dear Ms Culver

Re: R-v-Kathleen FOLBIGG

Thank you for asking my opinion with regards to the clinical significance of several pieces of research that suggest that there may be genetic causes to at least some cases of sudden infant death syndrome (SIDS).

Firstly, as requested, I will provide you with an outline of my qualifications and current position.

1. MB BS (Honours class II) attained at the University of Sydney (Year of graduation – 1981)

2. Fellow of the Royal Australian College of Physicians - admitted to Fellowship - April 1988.

3. Clinical Geneticist - certified by the Human Genetics Society of Australasia – July 1989.

4. Doctor of Philosophy - awarded March 1991 by University of Melbourne Australia.

I am a clinical geneticist, having further specialised in the field of inborn errors of metabolism, and have worked virtually exclusively in this field for the last 12 years.

I am currently the Director of the Western Sydney Genetics Program, and Head of the Genetic Metabolic Diseases Service, Children's Hospital at Westmead.

With regards to the various pieces of research you faxed me about, I make the following comments:

1 Polymoprhisms of the 5-HTT gene and increased risk of SIDS

This work has identified that a particular sequence variation (the so-called "L-allele") in the serotonin transporter (*5-HTT*) gene is associated with an increased risk of having SIDS, and appears to be more common in the North American black population. As is pointed out by the authors of this research, individuals with the L-allele were more likely to be at risk of SIDS, but there are certainly individuals with the L-allele who never develop SIDS, and there are individuals who died of SIDS who did not have the L-allele. This is more a disease association rather than a true cause-and-effect situation. Thus, even if all four of the deceased children could be tested for the L-allele and all four were found to have this genetic variation, it would not be compelling

evidence that SIDS was the cause of death. On the other hand, if some of the children had the L-allele and others did not, then this would be compelling evidence against this genetic polymorphism being an influence in this family.

2 Mutations in the SCN5A gene as a cause of SIDS

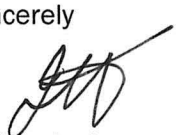
Mutations in the *SCN5A* gene have been found in a small number of children who have died of SIDS. This gene encodes a sodium channel, and is well established as a direct cause of the prolonged QT syndrome, where the heart tissue is at high risk of developing a potentially life-threatening arrhythmia (abnormality of heart rhythm). A paper by Ackerman and colleagues in the Journal of the American Medical Association (2001 286 (18); 2264 - 2269) reported definite disease-causing mutations in 2 out of 93 SIDS cases (approximately 2%). Here, we can be more certain that there is a direct cause-and-effect. In a family where there is more than one child who has died of SIDS, there would definitely be merit in exploring the possibility of genetic testing for a mutation in the *SCN5A* gene. Should this be something with which you would like to proceed, I can seek out a laboratory in Australia or overseas that could undertake such testing. However, there would very likely be charges for this testing, and even for medically indicated grounds these charges would not be rebatable through Medicare or private health funds.

3 Medium-chain Acyl-CoA Dehydrogenase (MCAD) Deficiency or other Fatty Acid Oxidation Disorders May Cause SIDS

Soon after it was found that most individuals with MCAD deficiency had a particular mutation in the *MCAD* gene, several retrospective studies suggested that up to 10% of SIDS could be due to MCAD deficiency. These studies were however flawed, and subsequent unbiased studies have shown that MCAD deficiency or other fatty acid oxidation defects probably cause less than 1% of SIDS. A fatty acid oxidation defect is particularly likely if there is pathological evidence in tissues of fat accumulation on microscopic analysis. This is not absolute however, so again in a situation where a family had more than one child die of SIDS it would worth considering testing to formally rule out a fatty acid oxidation defect. Reasonable screening tests would be urinary organic acid screening and/or plasma acylcarnitine analysis, but agonal or post mortem samples often yield uninterpretable results. A better way to test for one of a number of fatty acid oxidation defects (including MCAD deficiency) would be to undertake cultured skin fibroblast fatty acid oxidation studies, which is very sensitive and accurate. All of these biochemical studies are available through the NSW Biochemical Genetics Service at the Children's Hospital at Westmead, which is the national reference laboratory for such testing. Again, there would be likely to be non-rebatable charges for such testing. If such testing yielded a diagnosis, it might be possible to retrieve newborn screening cards from the siblings and perform limited biochemical testing in an attempt to confirm the diagnosis in them.

I hope this information answers your questions. Should you require any further information feel free to contact me.

With kind regards
Yours sincerely



John Christodoulou
Associate Professor & Director
Western Sydney Genetics Program

cc: JC file

NSW Police Service

EXPERT CERTIFICATE

Section 177, Evidence Act 1995 No. 25

In the matter of :	Kathleen Megan Folbigg
Place Statement Taken:	Children's Hospital at Westmead
Date:	6 March, 2003

Name:	Dr Richard Elkington HAWKER
Work Address:	Children's Hospital at Westmead
	Locked Bag 4001 Westmead
	NSW 2145 Australia
Work Telephone	98450000
Occupation:	Medical Practitioner

STATES:

1. This statement made by me accurately sets out the evidence which I would be prepared, if necessary, to give in court as a witness. The statement is true to the best of my knowledge and belief and I make it knowing that, if it is tendered in evidence, I shall be liable to prosecution if I have wilfully stated in it anything which I know to be false, or do not believe to be true.

2. I am 61 years of age.

3. I have a specialised knowledge based on the following training, study, and experience

Qualification :

MBBS FRACP

Other Study/Experience:

Witness: _____ Signature: _____

Richard Elkington HAWKER

I have practiced as a Consultant Paediatric Cardiologist for the past twenty-eight years at the Royal Alexandra Hospital for Children (now known as the Children's Hospital at Westmead). My duties include interpreting the majority of in-patient Electrocardiographs at the Children's Hospital Westmead in addition to those in my own practice.

In the past twenty-eight years I have diagnosed and treated a condition known "Long QT syndrome". There are a group of genetic medical conditions that are associated with sudden death that have an electrocardiograph feature of prolongation of the QT interval. This interval on the electrocardiograph corresponds to depolarization of specialized heart muscle cells responsible for electrical conduction. In my experience a patient suffering this condition often has a history of recurrent syncopal episodes and a family history of sudden, premature death and sometimes deafness.

4. At about 02.00 PM on the 6 March, 2003 at Children's Hospital at Westmead, I examined a twenty two page "Medi Trace" print out marked with the numeral 10 contained within medical records of Caleb Folbigg, an eleven page "Medi Trace" print out marked with the numeral 19 contained within medical records of Patrick Folbigg, a two-page photocopy from a nine lead single channel electrocardiograph recording on the baby Folbigg born 03/06/90, a one page photocopy of a recording of nine electrocardiograph leads mounted on an "E.C.G sheet" relating to the patient Patrick Folbigg, three single page print outs of a single channel electrocardiograph recording taken during a sleep study of the patient Laura Folbigg on 19/08/97, and one NSW Ambulance electrocardiograph report/print out in relation to the patient Laura Folbigg taken on 1/03/99 commencing 12.17.01 to 12.29.59 inclusive. I also attended the Sleep Laboratory of Dr Chris Seton at the Children's Hospital at Westmead and observed the records of a sleep study relating to the patient Laura Folbigg.

5. Based wholly or substantially on the above knowledge, I am of the opinion that the two "Medi Trace" print outs marked with the numerals 10 and 19 are of poor quality and I am not able to interpret them.

6. The two page photocopy of the nine lead single channel electrocardiograph recording of the baby Folbigg born 03/06/90, although not a complete standard electrocardiograph (twelve leads) nevertheless shows no abnormality. The corrected QT interval in this recording is 388 milliseconds (normal less than 440 milleseconds).

7. The one page photocopy of a recording of nine electrocardiograph leads relating to the patient Patrick Folbigg recorded probably on 18/10/90, although not a complete standard electrocardiograph nevertheless shows no abnormality. The corrected QT interval in this recording is 390 milliseconds.

8. The three single page print outs of a single channel electrocardiograph recording taken during a sleep study of the patient Laura Folbigg on 19/08/97 identifies a normal heart rhythm. In the absence of fine

Witness: _____ Signature: _____

Richard Elkington HAWKER

time lines the QT interval cannot be measured accurately but is of the order of 420 milliseconds and shows no abnormality.

9. The NSW Ambulance electrocardiograph report/print out is not of adequate quality to measure the QT level. It documents a heart rate of about 40 beats per minute at 12.17.32 and then recording artifact consistent with external cardiac massage. I have read the statement of Brian Wadsworth, ambulance officer which indicates that no pulse could be felt in this patient when he attended 8 Millard Close Singleton on 01/03/99. The presence of electrocardiograph complexes and no pulse is explained by electromechanical dissociation which is usually observed shortly before death. I cannot be more specific than this.

10. On the evidence presented to me, I am of the opinion that there is not an indication of prolonged QT interval in the electrocardiograph recordings that are available relating to the children Patrick and Laura Folbigg. The techniques of genetic identification of families with various forms of long QT syndrome is currently in the research phase and would only be pursued in the event of supporting medical evidence. The evidence presented to me does not give any indication that long QT syndrome was responsible for the deaths of Patrick and Laura Folbigg. I am unable to comment on the deaths of Caleb and Sarah due to the fact that I have not examined appropriate electrocardiograph records in regards to these children.

Witness: _____ Signature: _____

B. Ryan
D/Sgt.
6.3.03
5.30p.m.

Richard Elkington HAWKER

IL-10 GENE

Janice J. Ophoven, M.D.

6494 Crackleberry Trail
Woodbury, MN 55129
651.458.0541
fax 651.768.0994
ophoven@usinternet.com

March 27, 2003

Re:

Ms. Culver,

This correspondence is in response to your request for a summary of my opinions regarding the results being provided by Dr. David Drucker as to the significance of his discovery that Sarah Folbigg was homozygous for the IL-10 "cot death gene."

My clinical practice is pediatric forensic pathology. I have completed a residency in pediatrics, pediatric pathology, and a fellowship forensic pathology. During my career, I have participated in the care of children and young adults in such areas as:

Pediatric practice in rural and urban settings,
Management of a clinical laboratory for a children's hospital,
Diagnosis of solid tumors in children and adolescents,
Participation in and development of systems to evaluate quality of care [quality assurance]
Evaluation of medical care with unexpected or negative outcomes to identify areas for improvement [risk management]
I have conducted hundreds of autopsies in children and young adults for the purpose of making a diagnosis of cause and manner of death.

In addition, I have dedicated my clinical practice to research and education in forensic pediatric pathology and have written and taught workshops for a variety of professionals including physicians, coroners and medical examiners, law enforcement, pediatric caregivers, first responders, and members of the legal profession on such issues as:

Forensic analysis of injuries and death of children
Death investigation in childhood
Munchausen's syndrome by proxy
SIDS and homicidal asphyxia

In preparation of this report I have reviewed the following materials:

Materials

"Association of IL-10 Genotype with Sudden Infant Death Syndrome" Drucker, D, et. al. Human Immunology 2000 (61): 1270-1273
E-mail from Dr. Drucker
E-mail (12 Mar 2003) from Dr. Drucker to Bernie Ryan
Copy of the publication by Summers A., Summers CW., Drucker DB., et al., Assocaition of IL-10 Genotype with Sudden infant Death Syndrome, Human Immunology 61, 1270-1273 (2000)
12 Abstracts of articles with DB Drucker as co-author involving topics
Hypothesis suggesting a role of bacterial endotoxins and endotoxemia in SIDS
The role of bacterial endotoxin and products of cigarette smoke
The role of prone sleeping and upper airway bacterial flora

Prior review and report included review of those elements listed below.

Medical records of Kathleen Folbigg
Health Insurance Commision Records
Expert Certificate by Dr. Innis
Medical records supplied by Dr. Marley
Medical records supplied by Dr. Cash
Medical records of Caleb Folbigg
Statement from Dr. Bridget Wilcken
Newborn Screening Blood results
Newcastle Western Suburbs Hospital records
Coroners Brief
Ambulance Records
Medical records of Patrick Folbigg (Vol I & II)
Statement of Dr. Bridget Wilcken
Medical records of Sarah Folbigg
Statement by Dr. Wilcken
Newborn Screening Blood Results
John Hunter Hosptial Records
Statement by Dr. Marley
Pediatric discharge
Perinatal database
Reports: Dr. Hardacre to Dr. Marly
Buckner to Holland
Hardacre to Marley
Hardacre to Holland
Pickford to Marley
Edwards to Hardacre

Newborn screening blood results
Medical records from Newcastle Western Suburbs Hosp
Statement from Dr. Wilkinson
Medical Certificatee of cause of death
Cause of death certificate (hand written)
History, examination and progress notes
Report by Dr. Wilkinson to Marley
Report by Dr. Wilkinson to Dr. Morris
Adelaide Children's Hospital Pathology Report
Mater Hospital Pathology reports
Report by Dr. Challinor to Dr. Wilkinson
Biochemistry reports
Report by Dr. Wilkinson to Dr. Thomas
Physiotherapy report
Autopsy report
Report by Dr. Wilkinson to Dr. Bale
HAPS reports
Histopatholgy Dept Report
eport by Dr. Wilkinson to Folbiggs
eport by Dr. Colley to Dr. Wilkinson
Report by Dr. Marley to Dr. Holland
Dr. Colley to Dr. Wilcken
Dr. Wilckinson Dr. Colley
Dr. Edwards to Dr. Hardacre
Newcastle Mater Hospital Records June 14, 1990
Newcastle Mater Hospital Records October 18, 1990
Newcastle Mater Hospital Records November 4, 1990
Newcastle Mater Hospital Records November 14, 1990
Newcastle Mater Hospital Records December 22, 1990
Statement by Dr. Marley
Pediatric Summary
Ambulance Records
Beresfield Crematorium records
Ambulance records
Handwritten notes
Ambulance Records
Coroners Brief
Medical records of Laura Folbigg
Statement of Dr. Wilcken (1.14.00)
Newborn Screening Blood Results
Statement of Christopher Seton
Handwritten sleep notes by Kathleen Folbigg
Report by Dr. Seton to Det. Ryan
Referral by Dr. Seon to Dr. King
Letter by Mr. Folbigg to Dr. Seton
Report by Dr. Seton to Mr. Folbigg
Newborn discharge summary
Report by Dr. Seton to Dr. King
Corometrics monitor supply record
Urine medabolic profile
Sleep study report (10.7.97)
Royal Alexandria Hospital for Children Medical History
Sleep study report by Seton to Sanders
Letter by Craig Folbigg to Margaret Tanner
Report by Seton to Craig Folbigg
Report by Seton to Dr. Sanders
Patient alarm traces (Corometric monitor print outs)
Statement of Dr. Innis
Information sheet
Progress Notes
Singleton Hospital Records
Ambulance report
Fairholme Surgery Records
Statement of Dr. Cash
Newborn discharge summary
Report by Dr. Seton to Dr. King
Sleep study reports 10.7.97 and 2.3.98
Report by Dr. Seton to Craig Folbigg
Report by Dr. Seton to Dr. Sanders

Findings

In preparation for establishing my opinions I have accessed the medical literature specific to publications referencing genetics and SIDS as well as Dr. Drucker's publications. Specifically, I utilized a search engine that provided access to Medline through the ighWire Library of Sciences and Medicine. This library provides access to searchable content of 12,493,985 articles in over 4500 journals. My search included publications from January 1948- March 2003. I have included in the attachments a summary sheet which includes the journals listed by publisher.

The material submitted by Dr. Drucker to date includes one article suggesting that there is an allele of IL-10 associated with SIDS or, as he characterizes it, the SIDS gene. There is no SIDS gene. The IL-10 gene is associated with immunity, specifically a type of inflammatory molecule called a cytokine, in this case interleukin. Publications in the medical literature about the IL-10-592 allele with the exception of the single article referenced by Dr. Drucker relate to human liver graft rejection, primary sclerosing cholangitis, graft-versus-host disease, cystic fibrosis, diabetes and sepsis. These are all related to the reaction of the human body to immune stressors or inflammatory disease. There is no consensus or even discussion in print that this molecular finding is linked genetically to the SIDS event. There is no evidence that SIDS is an inflammatory process and there are no other articles suggesting that this gene can be linked to the SIDS event. To call this finding 'the SIDS' gene is misleading at a minimum and a mischaracterization of current thinking in this tragic condition.

The literature in SIDS is vast and even a subset of articles identified in a search under SIDS gene identified 197 articles. However, review of these articles does not identify a SIDS gene, nor does the review identify any new evidence of a hereditary pattern to this disorder. To date no specific metabolic findings are linked to SIDS and no genetic linkage of any kind has been verified.

The universe of theories pertaining to causation of SIDS submitted over the last 4 decades has, with understandable enthusiasm, hoped to identify the cause of SIDS. The list of possible mechanisms has been long and intensively studied over the years. Apnea, abnormal

cardiac conduction, abnormal metabolism, infectious agents and so on have been posited as "the cause" of this devastating condition. What we know now is that we don't know the cause but we recommend placing children to sleep on their backs instead of their tummies or sides. Babies still die from SIDS on their backs but the incidence has come down world-wide over the last 2 decades as more and more communities have adopted back-to-sleep campaigns and the diagnostic criteria has been more strictly adhered to.

What we have learned over the years is how to define and refine the criteria to make the diagnosis of SIDS and when **not to include** babies who die suddenly in the cohort of children identified with this condition. One thing we have discovered is that some children who were thought to have died of SIDS, did not. Some of these children were murdered.

Opinions

All opinions are stated to a reasonable degree of medical certainty.

What is the IL-10 gene theory in respect of SIDS?

Dr. Drucker supports the hypothesis that there is a genetic basis for SIDS that can be linked to variation in the interleukin cytokine production in the immune response. He cites literature that suggests that these variations or polymorphimsms are associated with certain inflammatory diseases such as autoimmune disorders and inflammatory bowel disease. He supports this hypothesis with a study of 23 children dying of SIDS. He found an increased frequency of a particular allele of IL-10 [IL-10-592*A] in the study group. He then compared this genome pattern with cytokine allele frequencies previously published in a transplant journal in 1998. Unfortunately, there is no evidence that this finding has anything to do with the SIDS event.

Is this theory accepted in the medical community?

This theory is not accepted by the medical community. Known risk factors for SIDS are not related to patterns of genetic inheritance and Dr. Drucker's is the only paper that suggests a connection between the IL-10 gene and SIDS. It is well recognized that the incidence of SIDS varies seasonally which does coincide with the 'respiratory" season. However, no infectious agent or etiology has ever been identified in the SIDS mechanism. As a matter of fact, if an infectious agent is identified the case is by definition excluded from the diagnosis of SIDS.

Risk factors in SIDS include periconceptional use of alcohol, maternal smoking and associated low birth weight, young mother, higher parity, single motherhood, low level of maternal education, ethnicity, age and sex of the infant, excess thermal insulation or overdressing, and prone or side sleeping position. Infection is not considered a risk factor in SIDS.

The theory has not been subjected to testing or review independently of the studies conducted by/through Dr. Drucker?

There is no proper or reliable scientific basis for drawing an association between the A allele of the IL-10 gene and SIDS. This hypothesis has not been verified nor have the studies been validated in the medical literature.

Were the tests which were performed by Dr. Drucker in this matter performed in a reliable, scientific and repeatable manner?

The tests may have been conducted properly, however the conclusions are pure speculation. I have not read the paper upon which the study based their control data. However, before the prevalence of this allele can even be considered important, the presence and profiling of the genome must undergo a much wider population analysis than cited in the transplant paper by Perrey.

Can those test results be regarded as accurate and reliable given that the DNA was extracted from histological samples in paraffin blocks and that, according to Dr. Drucker, multiple repeats in the analysis of that material had to be made due to "the very degraded nature of the DNA?"

Postmortem DNA testing is certainly in the preliminary phase, especially for the level of detail proposed in this case. These studies have not been verified or duplicated, nor are the conclusion drawn part of the mainstream of thinking in SIDS research at this time.

Is there anything known professionally about Dr. Drucker which may undermine or discredit his authority in reaching the results and conclusions which he purports to reach?

The only opinion I have is specific to Dr. Drucker's background. As I understand, he is part of the Dental School and is a reader in Microbiology. SIDS research brings many field of expertise to the table, but I would not be able to understand his qualifications to render opinions about factors relating to cause and manner or circumstance of death in this matter. These are Forensic issues and I would be surprised if he was qualified to render Forensic opinions or has done so in the past.

Please include (if possible) the figures regarding incidence of this particular genetic variation, in heterozygous and then in homozygous forms, in the general community.

The only citation reflecting the prevalence of this allele pattern (IL-10-592*A) is derived from a study of individuals in Manchester, England. I do not know how large the study was or the distribution of individuals by age and gender within the population. However, this is obviously insufficient to extrapolate to other populations without significant further investigations and analysis.

If you have any additional questions, or should you need additional information, please do not hesitate to contact me.

Sincerely,

Janice Ophoven, M.D.
Pediatric Forensic Pathologist

PROFESSOR ROGER W. BYARD
MBBS,BMEDSCI, MMEDSCI(PAEDIATR), MD,
CCFP, MACLM, FRCPC, FCAP, FRCPATH
SPECIALIST FORENSIC AND PAEDIATRIC PATHOLOGIST

9 Opey Ave.,
Hyde Park,
South Australia,
AUSTRALIA, 5061.

14/4/03

To: Mr Peter Krisenthal,
Solicitor,
Legal Aid NSW,
Central Square Building,
Cnr. Castlereagh & Hay Sts.
Sydney, NSW 2000,
PO Box K847,
Haymarket, NSW, 2000

Re: the deaths of Caleb, Patrick, Sarah and Laura Folbigg

I have been asked by Mr. Peter Krisenthal to comment on the possible significance of myocarditis in Laura Folbigg. In offering this opinion I have examined:

1) a video tape marked 'video footage of Laura Folbigg 28/2/99 copy'
2) 7 histological slides marked 99/9322 Folbigg Dr Cala: B, C, AA, AB, AC, BC, BD

Significance of the video footage:

The video footage of Laura shows an apparently normal little girl playing with her family in a swimming pool. She does not appear to be unwell. I do not, however, place much significance on this video for the following reasons:

i) Infants and young children may have quite significant and potentially lethal disease and yet show very little external manifestations. I have just completed the second edition of my text entitled *Sudden Death in Infancy, Childhood and Adolescence*' (Cambridge University Press)[1] which describes a multitude of such disorders. In fact one of the points of the text is to demonstrate to pathologists and clinicians that they should not be deceived by outward appearances in children – critically ill children may have surprisingly little symptoms and signs. I have found this point to be of great comfort to parents who I have counselled when an unexpected disease has turned up in their child at autopsy.

ii) A home video is not a clinical examination. Although I have a reasonable clinical background in paediatrics I could not tell from the video if Laura had a fever, was off her food, or was simply not her usual self

Significance of the myocarditis:

Examination of the 7 slides showed 8 pieces of heart muscle. In each of the pieces there was an inflammatory cell infiltrate with and without degeneration of heart muscle cells (myocyte necrosis). This indicates an established myocarditis.

Myocarditis refers to a potentially lethal condition of the heart, usually caused by a viral infection, in which the heart is infiltrated by inflammatory cells resulting in the death of these cells, as we see with Laura. The clinical signs and symptoms of myocarditis are very variable. In some case there may be obvious signs of a fever with heart failure. An affected child may breath with great difficulty, look very unwell and may be comatose. In other cases affected children who have died may have had no indication of any illness, or had only very mild symptoms that resembled a cold.

I have personally had several cases of infants and young children who have died from myocarditis with minimal or no symptoms. I also conducted a review of 16 children who died of myocarditis at the Adelaide Children's Hospital over an approximately 35 year period (1954-1989)[2]. Sudden death occurred in 5 of the 16, 3 of whom had no prodromal symptoms (19% of the total, 60% of the children dying suddenly and unexpectedly). This is not an isolated result, as it is a well-recognised problem with a number of similar reports in the literature. For example:

> De Sa *et al*[3] found that 17 out of 24 cases of isolated myocarditis presented as sudden death or with a clinical history of under 24 hours, 13 of whom had no preceding symptoms (54% of the total, 77% of the infants/children dying suddenly and unexpectedly or within 24 hours of symptoms). The authors comment that myocarditis, particularly in infants, 'often presented as a sudden death without prodromal symptoms or signs'.

Wentworth *et al*[4] describe a 12-year-old girl who died of myocarditis, who 'after playing basketball for 1 hour, swam two lengths (40m) in a pool, then suddenly stopped and flapped arms. Was pulled out within 15 seconds but had died'.

Grady and Costanzo-Nordin[5] have commented that 'patients with myocarditis may present with highly variable clinical pictures ranging from no clinical manifestations to overt clinical congestive heart failure or sudden death.' The highly variable clinical presentation was one reason why these authors called myocarditis an 'enigma'.

Neuspeil[6] addressed the issue of failure to recognise myocarditis as a cause of sudden death and commented, specifically with regard to sudden death, that 'clinicians may be unaware of this abrupt manifestation of myocarditis, because most of these patients have non-specific prodromes and are not referred to a cardiologist'.

It is certainly true that myocarditis may be completely coincidental to the cause of death[7] and I have had several cases where this has happened: a 19-month-old boy who choked on a peanut, a 7-month-old boy who drowned and a 3-month-old boy who accidentally suffocated. In these cases clearly corroborated death scene findings and autopsy features enabled a precise alternative cause of death to be given. However, in the absence of such findings, the deaths would have been attributed to myocarditis.

Conclusion:

It is very well recognised in paediatrics and paediatric forensic pathology that children with myocarditis may die suddenly and unexpectedly with no symptoms or signs. There are numerous reports in the literature confirming this. Thus, completely normal behaviour on the day before Laura died with myocarditis does not in any way exclude myocarditis as a possible cause of death.

Clinical Professor Roger W. Byard
BMedSci, MB, BS, MMedSci(Paed), MD,
CCFP, MACLM, FCAP, FRCPC, FRCPath

REFERENCES

1) Byard RW. *Sudden Death in Infancy, Childhood and Adolescence.* 2ND Ed. Cambridge; Cambridge University Press, (In press).

2) Smith NM, Bourne AJ, Clapton WC, Byard RW. The spectrum of presentation at autopsy of myocarditis in infancy and childhood. Pathology 1992; 24: 129-131.

3) DeSa DJ. Isolated myocarditis as a cause of sudden death in the first year of life. Forensic Sci Int 1986; 30: 113-117.

4) Wentworth P, Jentz LA, Croal AE. Analysis of sudden unexpected death in southern Ontario with emphasis on myocarditis. Can Med Assoc J 1979; 120: 676-706.

5) Grady KL, Costanzo-Nordin MR. Myocarditis: review of a clinical enigma. Heart and Lung 1989; 18: 347-354.

6) Neuspiel DR, Sudden death from myocarditis in young athletes. Mayo Clin Proc 1986; 61: 226-227.

7) Byard RW. Significant coincidental findings at autopsy in accidental childhood death. Med Sci Law 1997; 37: 259-262.

I am Owen David Hugh Jones.
My age is 53 years.
My qualifications are BA MB BChir MRCP(UK) FRCPCH
I am a duly registered medical practitioner practising the specialty of paediatric cardiology. I have been consultant paediatric cardiologist for 20 years; at Guys Hospital, London, UK from 1983 to 1987 and at Sydney Children's Hospital (formerly Prince of Wales Children's Hospital) since 1987. I have broad expertise in clinical paediatric cardiology.

I have been asked to provide a report in this matter by Mr Peter Krisenthal, Solicitor, Legal Aid New South Wales.

I have been provided with 5 folders of information:
Medical File – Caleb
Medical File – Patrick
Medical File – Sarah
Medical File – Laura
File containing reports of Dr Janice Ophoven, Prof Peter Berry, Dr Allen Cala, Prof Peter Herdson, Dr Susan Beal, Dr Robert Ouvrier, Dr Roger Byard, Prof Anthony Busuttil, Dr Richard Hawker and statements from Ambulance Officers attending (duplicates of those in medical files).

I have carefully considered all the information available to me. My statements and opinions in this report are true to the best of my knowledge and belief.

The Folbigg Family:

Father Craig
Born 21 November 1961
He has 8 siblings and 23 (or more) nephews/nieces one of whom died in infancy. No details of any abnormalities of cardiac relevance are known.

Mother Kathleen
Born 14 June 1967
Married 5 September 1987
She had a faint/fit in the last trimester of her first pregnancy. Subsequent EEG performed.
She is adopted and details of family history are not known save for the information that she has siblings and that her mother died unnaturally from foul-play.

ODHJ

Caleb
Born 1 February 1989
Died 20 February 1989
Age 19 days

There is no evidence in his medical records for clinically apparent congenital or acquired heart abnormality. No ECG recordings taken.

Clinical diagnosis of congenital laryngeal stridor attributed to laryngomalacia made by Dr B Springthorpe.

According to the documentation, he was last seen alive and well when given feed by mother at 1:00am 20 February 1989. Found by his mother to be lifeless at 2:50 am. Call to 000.

Ambulance Service of NSW arrived at 2:59 am (2 officers) and 3:03 am (2 officers).
Observed to be in cardiorespiratory arrest. ECG showed asystole.
CPR performed with no response.
(Medical File Items 41, 42, 44, 43, and 45 being Statements of R BAINES, D HOPKINS, A REED, and PATIENT REPORTS Q005 & Q006 refer).

Reported to Police and Coroner; post-mortem ordered.

Dr R Cummings states in his **AUTOPSY REPORT** (item 37):
INTERNAL EXAMINATION
Cardio-vascular System
Pericardial sac was normal.
The heart (25g) was of normal size and was normally developed.
Valves and chambers were normal.
Myocardium showed no evidence of disease.
Great vessels were normal.
HISTOLOGY
Heart Normal
CAUSE OF DEATH:
SUDDEN INFANT DEATH SYNDROME"

Patrick
Born 3 June 1990
Died 13 February 1991
Age 8 months

There is no evidence in his medical records for clinically apparent congenital or acquired heart abnormality.
ECG recordings from the early neonatal period and 18 October showed normal sinus rhythm and normal intervals including QTc 380ms.

ODHJ

Echocardiography on 16 November was normal.

Apparent life-threatening episode 18 October:
According to the documentation he was not obviously unwell when put to bed at 8:30 pm the previous day, seen twice subsequently, found near-dead by his mother at 4:00-4:30 am. Father awakened. Mother called 000. May have had a period of CPR.

Ambulance Service of NSW arrived at 4:41 am (2 officers)
Observed to be in respiratory distress.
Improvement with high-flow oxygen via face-mask.
Transported at 4:45 am to Newcastle Mater Hospital, arriving at 4:52 am. (Medical file Items 188 and 189 being Statement of D HOPKINS and PATIENT REPORT Q007 refer).

Subsequently had evidence of brain damage and epilepsy. Several hospital admissions; seizure-related apnea noted.

According to the documentation, he was not obviously unwell when put to bed at 7:30 am on 13 February by his mother. Found by his mother to be lifeless about 2 hours later. Mother called father, who returned home immediately, the neurologist, and 000.
Ambulance Service of NSW arrived at 10:10 am (2 units, 3 officers).
Observed to be in cardiorespiratory arrest.
CPR commenced.
Transported at 10:15 am to Newcastle Mater Hospital, arriving at 10:18 am. (Medical file Items 190, 191, 192 and 193 being Statementsof K COYLE, A MULLINS, M HETHERINGTON and PATIENT REPORTS Q036 & Q037 refer).
ECG on arrival showed asystole, no response to further resuscitation measures, declared deceased at 10:40 am.

Hospital post-mortem performed with father's consent.

Dr J Bishop states in his **AUTOPSY REPORT** (item 175):
"MACROSCOPIC REPORT
CARDIOVASCULAR SYSTEM:
Pericardium: No abnormality detected.
Heart: Weighed 49 grams. The atria, ventricles and the valves were examined and showed no abnormality. The origin of blood vessels from the heart was normal. The atrio-ventricular ring from the heart was kept for further histological studies (if required). Heart tissue was collected for EM and metabolic studies.
Aorta and its branches: No abnormality detected.
Venous system: No abnormality detected.
Lymphatic system: No abnormality detected.

ODHJ

CROWN-V-KATHLEEN MEGAN FOLBIGG

Death Certificate issued: (Items 31 32)
Cause of death:
I(a)Asphyxia due to airway obstruction (b)Epileptic fits
(Encephalopathic disorder. The underlying cause of encephalopathy not determined on investigation.)

Sarah
Born 14 October 1992
Died 30 August 1993
Age 10 months

There is no evidence in her medical records for clinically apparent congenital or acquired heart abnormality. No ECG recordings taken.

According to the documentation, she was not obviously unwell when put to bed at 9:00 pm on 29 August 1993, and last known to be alive between 12:00 and 12:30 am on 30 August 1993. Found by her mother to be lifeless about 1 hour later. Father awakened and started CPR. Mother called 000.

Ambulance Service of NSW arrived at 1:30 am (1 officer) and 1:48 am (3 officers).
Observed to be in cardiorespiratory arrest. ECG showed asystole.
CPR continued according to protocols.
No response to resuscitation.
Declared deceased at 2:10 am.
(Medical File Items 28 30 and 29 being Statements of D MARTIN, L ALDERSON, and PATIENT REPORTS Q604 & Q001 refer).

Reported to Police and Coroner; post-mortem ordered.

Prof J M N Hilton states in his **AUTOPSY REPORT** (item 41):

"INTERNAL EXAMINATION:
...uvula...congested/heamorrhagic...
Thoracic cavity:
The heart was normal in size shape and location.
Layers of pericardium separate.
No pericardial effusions present.
There was a very occasional petechinum mesentery present on the epicardium.
Atria normal.
Intra atria septum was intact and the foramen ovale was firmly closed.
There was the usual cribriform multi focal pro patencies in the intraventricular septum.
The ventricular myocardium appeared normal.
Leaves and cusps of the various valves were healthy.
The great vessels were healthy and normally formed and distributed.

Microscopic examination:
Sections of uvula ...vascular congestion...
Section of heart were normal.
CAUSE OF DEATH:
S.I.D.S"

Laura
Born 7 August 1997
Died 1 March 1999
Age 18 months

There is no evidence in her medical records for clinically apparent congenital or acquired heart abnormality.
Review of printouts from Sleep Study performed on 18 August 1997 with regard to the ECG channel shows normal sinus rhythm and normal intervals including QTc 380ms.

Noted to have cold/flu-like symptoms from 21 February 1999.
According to her mother, she was asleep and not obviously unwell when put to bed sometime after 11:00 am on 1 March 1999. Half an hour or so later her mother heard her cough, went to check her a few minutes later and found her lifeless.
Her mother carried her to the breakfast bar where she commenced CPR and called 000.

Ambulance Service of NSW arrived at 12:14 pm.
Observed to be in cardiorespiratory arrest. ECG showed asystole.
CPR continued according to protocols.
Transported to Singleton hospital at 12:29, arriving at 12:32.
Resuscitation measures continued.
No response; declared deceased at 12:45 pm.
(Medical File Items 48,49,51and 50 being Statements of H PICTON, B WADSWORTH, T AU, and PATIENT REPORT V70724 refer).
Review of the hardcopy printouts downloaded from a device recording the ECG on 1 March 1999 from 12:17:01 to 12:40:37 shows the initial rhythm to be a broad complex bradycardia at a rate of 30 per minute. This is consistent with clinically evident cardiac arrest and may be referred to as electro-mechanical dissociation (EMD) or pulseless electrical activity (PEA). The remainder of the recording is consistent with cardiac massage artefact and/or other agonal rhythm abnormality.

Reported to Police and Coroner; post-mortem ordered.

ODHJ

CROWN-V-KATHLEEN MEGAN FOLBIGG

Dr A D Cala states in his **AUTOPSY REPORT** (item 56):

"INTERNAL EXAMINATION OF THE BODY:
Cardio-vascular system:
The heart weighed 62g.
The pericardium was normal.
The valves and atria of the heart were normal apart from an 8 mm diameter area of haemorrhage on the posterior surface of the left atrium.
The free wall thickness of the right ventricle was 2 mm and that of the left ventricle was 7 mm.
The myocardium was normal on section.
The coronary arteries had a normal distribution and were free of disease.
The venous system, portal veins and hepatic portal system showed no abnormality.
The aorta and its branches were normal.
There was no evidence of congenital heart disease.

MICROSCOPIC EXAMINATION OF TISSUES:
Heart:
Within the myocardium is a moderately dense infiltrate of lymphocytes which have aggregated in certain areas particularly subendocardially and along the superficial surface of the myocardium, although further sections show large aggregates in the central area of the left ventricle. In these areas, there are large clusters of lymphocytes surrounding degenerate myocytes. Myocytolysis is present. No viral inclusions are seen. The appearances are of myocarditis, which is probably viral in actiology.
Heart: (2nd/3rd cuts)
Further blocks taken confirm the presence of aggregates of lymphocytes in a similar distribution to those in the first histological examination of the heart.
REPORT SUMMARY AND OPINION:
Histological examination of tissues showed an inflammatory infiltrate in the heart, consistent with myocarditis, of probable viral origin. This accords with the history of a cold/flu-like illness for several days prior to the death of the child.
Although there was an inflammatory infiltrate in the heart consistent with myocarditis, this may represent an incidental finding.
CAUSE OF DEATH:
Undetermined"

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CROWN-V-KATHLEEN MEGAN FOLBIGG

Prof P J Berry in his **REPORT** comments as follows:
"Examination of microscope slides:
Myocardium. Heart muscle shows a patchy but widespread intersitial mononuclear infiltrate in the right and left ventricles. There is no definite myocyte necrosis.
Comment:
Nevertheless, taken in isolation I would have ascribed this death to myocarditis recognising that although the infiltrate was quite extensive, I could not see actual damage to heart muscle."

Dr J Ophoven in her **REPORT** comments as follows:
"The microscopic sections of heart reveal the presence of myocarditis, most probably viral in origin."

Prof P B Herdson in his **REPORT** comments as follows:
"...I agree that histopathology of the heart reveals a myocarditis which is most probably of viral origin..."

Prof R W Byard in his **REPORT** comments as follows:
"I would agree with Dr Cala and Prof Berry that the slides from the heart demonstrated myocarditis.
Given the finding of extensive myocardial inflammation with no other abnormalities present I would have attributed the death to myocarditis."

OPINION:

There is no credible evidence for an inherited disorder of cardiac rhythm, such as long QT syndrome (LQTS), in this family.

There is no evidence for an intrinsic congenital or acquired cardiac abnormality causing or contributing to the deaths of Caleb, Patrick and Sarah.

There is good evidence that Laura had myocarditis at the time of her death. The histopathological findings of all the experts who have examined the heart sections are conclusive. This fact does not appear to be disputed in any of the expert reports.

Myocarditis is an inflammatory condition of the heart muscle for which there are many causes. Viral infection is a frequent cause and there may be features in the affected individual of a recent viral illness. Myocarditis is a well recognised cause of unexpected sudden death in children of all ages. Alternatively the affected individual may present with features of cardiac failure ranging in severity from mild to severe; such individuals may die or make a partial or complete recovery. Finally there will be individuals who have myocarditis who neither die nor exhibit features of cardiac failure; in such individuals

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who die for other reasons the histopathological features may be considered *incidental.*

The broad spectrum of consequences of myocarditis outlined above is well recognised and indeed described in the reports from other experts.

The crux of the matter is whether myocarditis was causal, contributory, or incidental to the death of Laura.
To my knowledge, in myocarditis, there is no evidence that there is a threshold of severity of histopathological features below which appropriately attributed sudden unexpected death cannot occur.
The absence of clinical features of heart failure cannot be used to argue that myocarditis has not caused the sudden death of an affected individual.

Considering the death of Laura in isolation from the standpoint of a paediatric cardiologist, I would have no difficulty in attributing her death to myocarditis.

I am not qualified to speculate in the overall picture of Laura's death whether myocarditis was causal or incidental.

O. D. H. Cary

15/04/03

ODHJ

IL-10 GENE

R-v Kathleen FOLBIGG

Hand delivered in Court 25/4/03. to P Krissell.

~~Draft~~ Supplementary Report

PJ Berry (Paediatric Pathologist)

I have been asked to provide a supplementary report describing the significance of Dr David Drucker's work on IL-10 in the Sudden Infant Death Syndrome, and his statement that:

"the test on Sarah [Folbigg] found she had two copies of the 'cot death gene' which would obviously increase her risk of SIDS". (Email 12th March 2003)

I have also been shown a separate undated communication from Dr Drucker in which he states:

"The samples we received have proved exceptionally difficult... we had to repeat the analyses five times until we had optimised conditions for the sample. Sarah is homozygous for the so-called cot death gene. She is at higher risk than even a baby with one copy would have been."

I have not been shown a formal report from Dr Drucker.

Background

In a paper submitted to the journal Human Immunology in May 2000 and published later that year, Dr Drucker and colleagues described an association between a particular naturally occurring variant of a gene and the Sudden Infant Death Syndrome. The gene in question encodes for IL-10, a substance that is important in inflammation and the response to infection. The particular variant (IL-10 592A) implicated by Dr Drucker and colleagues is believed to result in lower production of IL-10. They hypothesised that this genetic variant might predispose to SIDS "by tardy initiation of protective antibody production [and hence susceptibility to infection] and a lower capacity to inhibit inflammatory cytokine production".

This, and other theories implicating novel mechanisms and infection have rightly attracted considerable interest among SIDS researchers.

This paper also received considerable attention in the media, which prematurely conferred the pejorative label "cot death gene". This label is misleading, not least because many cot death victims do not have this gene variant, and the vast majority of people with it lead healthy lives.

SIDS research is littered with abandoned theories so that most cot death researchers do not accept new findings until another independent group has confirmed them. For example, more than a dozen separate studies involving hundreds of SIDS victims have confirmed the risk of placing babies to sleep in the prone position, so that risk

factor is accepted. Many other theories have not been independently confirmed, and so are not generally accepted.

The most common problems with SIDS studies involving statistics are small numbers of cases, case selection and inappropriateness of controls. The IL-10 study, while it involves very sophisticated laboratory methods, is essentially a statistical study comparing the frequency of the IL-10-592A variant in a group of SIDS cases and controls.

The size of the study

The study examined just 23 SIDS cases, and so cannot be regarded as anything more than an interesting preliminary study at this stage.

Case selection

The SIDS cases were included on the basis that they had been "subject to detailed postmortem examination in the North West Regional Perinatal Pathology Department in St Mary's Hospital (Manchester, United Kingdom)." The only way of being certain that a group of cases is unselected is to study all consecutive cases from a defined population over a defined period. If this is not done, then there is a possibility of selection bias, as for example if coroners directed "medical" sounding cases to the perinatal pathology department at St Mary's, and "legal" sounding cases to forensic pathologists elsewhere. Another possible source of bias arises if the diagnosis of SIDS is simply taken from the post-mortem report without further critical review; in this study no exclusions were made, and the authors state "it is possible that babies who died of other causes may have been included." From the paper alone, it is not possible to be confident that the SIDS group is free from selection bias.

Controls

The selection of appropriate controls depends on the question being asked in the particular study. In this case the question is; do SIDS babies carry the IL-10-592*A gene variant more often than babies who do not die? The appropriate control group should therefore be made up of living babies carefully matched for possible confounding factors, and that matching should be tested as part of the analysis of the results of the study.

In this study the authors state that "The control group and their cytokine allele frequencies were those described by Perrey et al consisting of healthy, and sex- and ethnically-matched individuals from the North West Region." The study of Perrey et. al. uses as its control group "principally healthy volunteers or cadaveric renal transplant donors". Curiously, the figures for IL-10*-A and IL-10*-C among the 330 controls quoted in the Drucker paper (161 and 499 respectively) are not the same as those in the Perrey paper (151 and 509 respectively).

This control group is clearly prone to serious selection bias, and is inappropriate as a control group for a SIDS study. Indeed, Perrey et. al. warn that "Any study should have a set of matched controls, particularly in studies of other ethnic groups. We have reason to suggest that allele distribution may be quite different in other populations".

The significance of the study
Assuming that the results are unaffected by selection bias, then the presence of the IL-10-592*A gene confers about a 3-fold increased risk of SIDS. This is a relatively weak association, comparable to side sleeping, but considerably less than other established risk factors such as prone sleeping where the risk is increased by about 8-fold, head covering where the risk is increased about 30-fold, or sleeping with an adult on a sofa when the risk is increased about 50-fold.

Using the SIDS rate quoted in the paper of 0.62 per 1000 live births, a 3-fold increase in risk would mean that an affected baby would have a risk of dying of SIDS of less than 1in 500.

Dr Drucker says that Sarah Folbigg "is at higher risk than even a baby with one copy would have been". It may be that, if the association is substantiated, then babies with two copies will be at greater risk than babies with one copy of IL-10-592*A. In their paper, the authors pooled the results from babies with one and two copies of IL-10-592*A, so that the figure of 3 is an "average". It is therefore not possible to say from the data presented that the risk for babies with two copies is greater than that for babies with one copy of IL-10-592*A.

Is IL-10-592*A a cause of death in SIDS?
Assuming that the results are unaffected by selection bias, then the authors have demonstrated an association between IL-10-592*A and SIDS.

They have not presented data to support their theory that this gene variant predisposes to infection or an aberrant inflammatory response. For example, there are no data about antibody levels, nor are the presence of infection or inflammation in SIDS babies with and without this gene variant compared.

IL-10-592*A therefore remains a possible *association* only, and cannot be invoked as a *cause* of death in SIDS. No pathologist to my knowledge has ever invoked IL-10-592*A when certifying the cause of death of a baby who has died suddenly and unexpectedly.

Independent confirmation
Dr Drucker and his colleagues conclude, "If the present study can be confirmed in a larger analysis, then IL-10 genotyping may provide a means to identify children at increased risk of SIDS..."

I am not aware of any other study confirming the association between IL-10-592*A and SIDS. I have carried out a Medline search with negative results.

I am aware that Dr Drucker has received funding to carry out a further study, but to my knowledge this has not yet been published.

Conclusion

1. The work of Dr Drucker and his colleagues is of scientific interest

2. The published results of this group concerning IL-10-592*A and SIDS can only be regarded as a preliminary study
3. There are several possible sources of selection bias in cases and controls which may make the conclusions unreliable
4. The results have not been confirmed in an independent study
5. If these findings are confirmed, then IL-10-592*A confers a modest increase in the risk of SIDS
6. This is an association only, and not a cause of SIDS
7. The great majority of individuals with IL-10-592*A do not die as cot deaths
8. The use of the term "cot death gene" in respect of IL-10 is pejorative and frankly misleading

Unsigned
26/03/03

29/04/03