INQUIRY SCHEDULE: EXPERTS' VIEWS ON CAUSE OF DEATH

CLAIRE ROBERTS

CAUSE OF DEATH	REASONING
PROF BRIAN HARDING (PSNI expert in Neuropathology)	Brain swelling was immediate cause of death and hyponatraemia is the only causative factor that has been positively identified. (096-027-361)
Cerebral oedema caused by hyponatraemia **No evidence of meningitis / encephalitis / cerebral malformation**	Given the marked degree of brain swelling it is extremely unlikely that microscopic evidence of encephalitis would not be evident by 3 days. He has seen it occur within 36 hrs. (235-002-001) No evidence of meningitis or encephalitis. (096-027-360) No convincing evidence of [cerebral] malformation. (096-027-036)
DR RAJAT GUPTA (PSNI expert in Paediatric Neurology)	*"Hyponatraemia may result from recurrent vomiting and Claire did have a history of vomiting prior to and during has admission."* (097-011-026)
Cerebral oedema caused by hyponatraemia **No evidence of status epilepticus**	*"If Claire worsening hyponatraemia had been detected earlier on 22nd October 1996 and appropriate changes to her fluid management made earlier then it is possible, although not certain, that the hyponatraemia may not have been as severe and cerebral oedema may have been prevented."* (097-011-027) No clear evidence of non-convulsive status epilepticus - he believes that this was

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	unlikely.(097-011-026)
DR DEWI EVANS (PSNI expert in Paediatrics)	*"I am certain that the direct cause of death was cerebral oedema, and that the hyponatraemia was due initially to the syndrome of inappropriate ADH. The progression of the hyponatraemia was due to the failure to prescribe the appropriate fluid and the failure to take adequate measures to monitor sodium balance and the consequent failure to change both the type of fluid given and its volume. The failure was exacerbated by the delay in organising CT scanning which led to the delay in identifying cerebral oedema."* (096-022-139)
Cerebral oedema caused by hyponatraemia caused by SIADH	
DR WANEY SQUIER (Inquiry expert in Neuropathology)	The recent neuropathology is of brain swelling. (236-003-007) Agrees with the diagnosis on the Death Certificate (1(a) Cerebral oedema (b) Status epilepticus) but not that on the Verdict on Inquest (1(a) Cerebral oedema Due to (b) Meningo-encephalitis, Hyponatraemia due to excess ADH production and status epilepticus) (236-004-002) It is not possible to determine the cause of the brain swelling from examination of the brain sections, and it may be due to hyponatraemia. (236-003-006) There is a mild old hippocampal scarring (sclerosis) which would explain the history of epilepsy. (236-003-007)The cause for swelling is not apparent in the brain; there is no evidence of meningitis or encephalitis. (236-003-007)
Cerebral oedema caused by status epilepticus **Hyponatraemia is also a possible cause, but it cannot be determined from microscopic examination of the brain** **No evidence of meningitis / encephalitis / malformation**	

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	There is no malformation or migration disturbance. (236-003-007) *"The most likely cause is an epilepsy syndrome. [...] Her terminal illness appears to have been epileptic activity precipitated by a concurrent infection and complicated by hyponatraemia."* (236-004-017)
DR ROBERT SCOTT-JUPP (Inquiry expert in Paediatrics)	Increased number of white blood cells in the CSF would be evidence to support a diagnosis of Meningitis or Encephalitis contributing to CR's death with hyponatraemia as secondary to this. (234-002-011)
Cerebral Oedema caused by encephalitis / meningitis / encephalopathy **Possible hyponatraemia also cause of cerebral oedema** **Possible encephalitis made brain more susceptible to damaging effects of hyponatraemia**	Still plausible though that acute deterioration and the cerebral oedema with coning was caused by hyponatraemia. (234-002-012) *"However, it remains also plausible that the initial presenting illness was caused by a viral Encephalitis or an Encephalopathy, and that the Hyponatraemia was a secondary phenomenon."* (234-002-012) Plausible that a pre-existing encephalitic illness may have made the brain cells more susceptible to the damaging effects of hyponatraemia and thus more likely to swell up and become oedematous, than had this pre-existing condition not been present. (234-002-012)

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PROF BRIAN NEVILLE (Inquiry Expert in Paediatric Neurology)	Unexplained acute encephalopathy with terminal cerebral oedema with hyponatraemia related to inappropriate ADH secretion (232-002-014)
Cerebral oedema caused by encephalopathy / hyponatraemia (related to SIADH) **Possible pneumonia** **No evidence of status epilepticus**	The most likely antecedent cause of SIADH was a virus infection involving the brain and pneumonia could have been a part of the intercurrent viral illness given the abnormality on the chest x-ray. (232-002-015) Unlikely that the nature of encephalopathy was subclinical epilepsy. (232-002-014) *"I do not see evidence for status epilepticus."* (232-002-015)
PROF KEITH CARTWRIGHT (Inquiry Expert in Microbiology)	CR results consistent with an acute and fulminant encephalitis. (233-002-006) Viral encephalitis is most likely cause of death. (233-003-003)
Cerebral oedema caused by viral encephalitis **Possible that hyponatraemia caused or contributed to cerebral oedema. SIADH well-recognised complication of encephalitis.** **No evidence of meningitis**	Not within his expertise to assess whether hyponatraemia caused or contributed to cerebral oedema though he observes that inappropriate ADH secretion is a well-recognised complication of both meningitis and encephalitis. (233-002-006) CR's respiratory arrest at 2.30am on 23/10 was almost certainly caused by coning secondary to cerebral oedema and raised intercranial pressure. Both seizures and respiratory arrest could have been caused by an encephalopathy of infective or non-infective origin.(233-003-005) CR did not die from meningitis, either viral or bacterial. (233-002-006)

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	Total peripheral white blood cell in the blood sample collected on day of admission was highly suggestive of infection. (233-002-010) Substantial relative excess of white blood cells in the (post mortem) CSF sample which could not be explained by blood contamination. (233-002-015) Although CR had negative antibody test results these are meaningless as it takes a minimum of 6-8 days for antibody levels to rise in such infections. (233-002-016) [Dr Evans highlighted that] cytology studies of CSF white blood cells revealed them to be predominantly lymphocytes, in absence of mononuclear cell CSF infiltration caused by a malignancy, such a finding makes a viral CNS infection a real possibility. (233-002-015) CR showed no signs of meningitis such as fever, stiff neck, photophobia. (233-002-016) Meningitis would have been obvious at autopsy. (233-002-016) Alternative hypothesis of hyponatraemia does not explain: She was unwell prior to admission with what appeared to be an acute infection High peripheral white blood cell count on admission Relative leucocytosis and lymphocytosis in CR's CSF. (233-003-006)
DR CAREN LANDES (Inquiry Expert in	It is not possible to differentiate between infection, changes related to inhalation

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Radiology)	or aspiration and pulmonary oedema from other causes on the basis on imagining alone. (230-002-003) The changes on the CT scan [between the first and second chest x-rays] are suggestive of cerebral oedema. There is no evidence of intracranial haemorrhage or space occupying lesion. (230-002-004)
Cerebral oedema – cannot comment as to cause	
DR IAN MACONOCHIE (Coroner's Expert in Paediatric Accident & Emergency)	Dr Maconochie does not outline his reasoning as to the cause of death.
Cerebral oedema caused by encephalitis / encephalopathy and hyponatraemia; Status epilepticus	
DR R M BINGHAM (Coroner's Expert in Paediatric Anaesthesia)	Thinks it likely that Claire's neurological illness caused ADH secretion. (091-006-021) Thinks it most likely that hyponatraemia was the cause of the neurological deterioration on the evening of 22nd October culminating in the respiratory arrest at 03.00 on the 23rd. It is not, however possible to exclude the possibility that the serum sodium was an isolated artefact and the deterioration was due to acute encephalopathy. (091-006-027)
Cerebral oedema caused by encephalitis / encephalopathy and hyponatraemia; Status epilepticus **Neurological illness likely cause of SIADH**	

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PROF IAN YOUNG (RBHSC Expert in Clinical Biochemistry)	Hyponatraemia may have made a contribution to the development of cerebral oedema in Claire's case. (096-007-040)
I (a) cerebral oedema (b) meningoencephalitis (ii) hyponatraemia due to excess ADH production (iii) status epilepticus. (096-008-045)	