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Report To The Inquiry Into Hyponatraemia-Related Deaths in Northern Ireland

RE: CONOR MITCHELL

Preliminary Paediatrician's Expert Report

INTRODUCTION

I have been requested by the Inquiry to give my expert opinion into certain aspects of the events leading up to the death of Conor Mitchell in May 2003.

I have been supplied with and studied the following documents:

1. Detailed brief from the Inquiry containing important details of the clinical aspects of the case and the involvement of the various professionals.

2. Copies of all the case notes relating to Conor Mitchell's admission to Craigavon Area Hospital in May 2003: (File 88).

3. A copy of the coroner's verdict from the inquest in June 2004: (087-057-221)

4. A copy of the Post-Mortem examination report: (092-028-141).

5. Copy of a poster distributed to all trusts in Northern Ireland by the Chief Medical Officer for Northern Ireland in March 2002 (007-003-004), accompanied by a letter explaining the basis for this advice to all clinicians (007-001-001).

6. I have not been supplied with the GP records, nor with records from the Royal Belfast Hospital for Sick Children.

7. Depositions made to the inquest by members of the family and a number of the medical and nursing staff involved. I have made reference to these, and

to the coroner's verdict, where they add to what is in the case records, using square brackets [ ].

This is a preliminary report based primarily upon my assessment and interpretation of the case notes relating to Conor Mitchell's admission to Craigavon. I will provide further relevant commentary when I have been furnished with the witness statements in this matter.

MY CREDENTIALS

I am a Consultant Paediatrician in a small district general hospital in England. I qualified in 1990 and took up my consultant post in 1992. My consultant post involves care of children presenting acutely with a wide variety of conditions and I have some experience with the conditions relating to this case. As I took up my consultant post in 1992 I am familiar with the standards of practice current in 2003.

SCOPE OF MY REPORT

As instructed by the Inquiry, I have limited my comments mainly to the appropriateness of Conor's intravenous fluid management at Craigavon, particularly in the light of the recently issued guidance. I have not considered the appropriateness of his care in the community before he was admitted to hospital, nor his management following transfer to Belfast.

BRIEF SUMMARY OF THE CASE

The following summary of events is extracted from the brief provided to me by the Inquiry and the case records.

Conor Mitchell was 15 years old at the time of his death. In infancy he had been diagnosed as having cerebral palsy. [According to his mother's statements, in spite of him having a severe physical disability, and an inability to walk and talk, he was nonetheless an intelligent boy and could communicate through non-verbal means.] His general health was good, apart from epilepsy, said to be largely absence epilepsy, for which he had been treated with Sodium Valproate for several years.

Conor became unwell on 27th April 2003, and was seen and treated a number of times in primary care before he was admitted to Craigavon Hospital on 8th May 2003. On admission he was thought by the adult medical team who saw him to have a viral illness and possibly a urinary tract infection (UTI), and was treated with intravenous fluids and IV antibiotics. He was admitted to the medical admissions unit (MAU).

About 9 hours after his admission, he had a seizure that resulted in a respiratory arrest, and he was resuscitated and transferred to the general intensive care unit at Craigavon. He was thought to have had a subarachnoid haemorrhage as part of his problem. He remained critically unwell, and the following day he was transferred to the paediatric intensive care unit in Belfast where he subsequently died after care was withdrawn.

TIMELINE OF EVENTS

Pre-admission

27th April 2003
Developed vomiting and sore throat and was seen by GP who diagnosed a viral upper respiratory infection. Paracetamol only advised.

30th April 2003
Seen by out of hours GP, thought to have throat or ear infection and given penicillin by mouth.

1st May 2003
Again seen by out of hours doctor, and because of vomiting treatment changed from penicillin to amoxicillin. Advised to take 30 ml per hour of water to maintain hydration.

2nd May 2003
Seen by GP. Vomiting continuing and amoxicillin stopped. Later, out-of-hours GP advised analgesia and less water by mouth.

3rd May 2003
Some improvement, drinking 30 ml an hour and continuing to have yellowish vomit thought to be due to previous day's antibiotic.
Comment: This yellow vomit is likely to have been bile rather than the antibiotic which would not still be appearing in the vomit so long afterwards.

4th May 2003
Vomiting continuing with hiccups. Eating better.

5th May 2003
Improved, more lively and drinking 60 ml per hour.

6th May 2003
More tired. Incontinence with urine which was uncharacteristic. Mother noticed 'residue' in urine. Reassured by out of hours GP.

7th May 2003
Improved, eating better but apparently in some discomfort and arching back. Mother reassured by out-of-hours doctor.

8th May 2003
Own GP visited at home and was sufficiently concerned to arrange admission to hospital. Was actually referred to the Royal Belfast Hospital for Sick Children, but

ended up going to the Emergency Department at Craigavon Area Hospital because it was closer to home. GP states in referral letter that he was well perfused, with a normal heart rate, and they were uncertain of the cause of his deterioration. He appeared to be in pain and his abdomen was tender. He was more drowsy and his fluid intake was poor: (088-002-022).

Comment about management in primary care: It is always very difficult for GPs to correctly assess children with severe disabilities like Conor. If they are unable to communicate it is difficult for GPs to assess how ill they are, and it is sometimes difficult to tell whether abnormal movements are a result of pain and illness, or just part of the cerebral palsy generally. The parents' instincts in this situation are often the most reliable indicator.

There do not appear to have been any comments about his urine output or concentration, which could have given an indication of his degree of hydration, and nor whether he had diarrhoea or not. If his vomiting was accompanied by diarrhoea he would have been at greater risk of dehydration.
[The mother's deposition, with its full description of Conor's symptoms, makes no mention of diarrhoea (087-002-015)]

Following admission to Craigavon on 8th May 2003

1050
Assessed in Emergency Department: (088-002-020).Thought to have dehydration due to vomiting, although afebrile and observations normal. An intravenous (IV) cannula was inserted and blood tests were taken which were essentially normal, apart from a slightly raised urea that may or may not indicate a degree of dehydration: (088-004-036). IV fluids were prescribed in the form of Hartmann's solution, initially 220 ml over about 25 minutes. According to the charts (088-004-063), 110 mls of Hartmann's was administered at 11.20, a further 110 mls at 11.45, and again at 12.00 midday. However the 'Volume in' column on the chart shows only two infusions of 110 mls. Conor was also prescribed intravenous antibiotics in the form of ciprofloxacin, which has to be given as an infusion contained in 200 ml: (088-004-061.

Comment: The standard fluid resuscitation bolus of a child thought to be severely dehydrated is between 10 and 20 ml per kilo , which in Conor's case would have been between 220 ml and 440 ml. (See APLS guideline: ref 1) This is usually given quite rapidly, over between 15 and 30 minutes. The fluid used should be isotonic i.e. normal (0.9%) saline or Hartmann's. Assuming that Conor was genuinely severely dehydrated (i.e. at least 10% of body weight lost), then this initial fluid management was appropriate. However the assessment of his degree of dehydration appears to have been somewhat subjective: there is no mention in either the A&E admission or the MAU admission records (088-002-021 and 088-004-045) of the specific physical signs of dehydration (see below under 'Compliance with Guidelines', part d 'Monitor'). In his assessment on MAU, Dr Murdock simply states 'dry' (088-004-045). Furthermore there is no clear indication from the case records that Conor was

showing signs of hypovolaemic shock (i.e. inappropriately high pulse or low blood pressure: his pulse was recorded as 72 and his BP as 118/69 which are normal). Dr Budd, the A&E doctor who first saw him, described him as 'drowsy, pale', which may have been interpreted as due to dehydration, but may have had other causes (088-004-038).

[In her deposition at the inquest, Dr Budd stated that Conor had 'signs of dehydration', although 'haemodynamically stable' (i.e. normal pulse and BP) (087-029-133). She assessed him as having 'mild' dehydration, i.e. 5% (087-028-135).]

Standard practice in paediatrics is for a bolus of fluid of 10-20 mls/kg to be given rapidly only if there are signs of actual or impending hypovolaemic shock (APLS guidelines, ref 1). In Conor's case there were none, apart from his decreased conscious level, which may have been wrongly interpreted. In my view, therefore, a rapid bolus was unnecessary, and a slower rehydration regime was more appropriate. What appears to have been given was somewhere in between the two: a quantity appropriate for a resuscitation bolus, but given over a longer period than would normally be used, which is 15-30 minutes. The exact period over which it was given is unclear from the fluid balance chart (088-004-063): it may have been 220 mls between 11.20 and 12.00, or 330mls between 11.20 and about 12.20, (assuming the third 110 bolus went in at about the same rate). Thereafter it appears from the chart that only the 200 mls of Ciprofloxacin in normal saline was given before the IV extravasated, unless, as mentioned above, the normal saline maintenance fluid was started before this.

It is noted on the IV prescription chart (088-004-064) that after the initial bolus of Hartmann's 220 ml, he was originally prescribed 3 litres of fluid, normal saline alternating with 5% dextrose, over a 24 hour period. This is a fairly standard fluid prescription for any full-sized adult, however it appears that the prescribing doctor then became aware of just how much smaller Conor was compared to a normal adult, and the fluid prescription was correctly revised to reflect this. If the fluid chart is to be believed, he actually received 110 ml more of Hartmann's than he was prescribed as the initial prescription was limited to 220 ml. However it is uncertain whether this was actually given. The nursing note (088-004-091) states that only 220 ml were given.

Regarding his antibiotics, in the Emergency Department it appears that the decision was made to give him IV ciprofloxacin. This was prescribed as a dose of 200 mg 12 hourly on a drug chart (088-004-061). IV ciprofloxacin comes as a dilute solution, dissolved in normal saline, that has to be given as an infusion, and this amounted to 200 ml of extra fluid.

Comment: The choice of antibiotic is surprising. Ciprofloxacin in paediatric practise is not normally given as a first line antibiotic, either orally or IV, for what was then thought to be a straightforward UTI. It is normally kept in reserve for more serious infections that are resistant to more standard antibiotics. It may be that a non-penicillin antibiotic was chosen because of the possible history of vomiting in

response to penicillin, although in my view it should have been clear that this was not a genuine penicillin allergy. More normal practise would have been to have given a third generation cephalosporin such as cefotaxime or ceftriaxone, which is unlikely to cause problems even in a penicillin allergic patient.

1300
Conor was admitted to the adult MAU at Craigavon, following an apparent discussion as to whether he should be admitted under the paediatricians. It appears that the decision to send him to the MAU was simply on account of his age as the policy at the time was that all young people over the age of 14 should go there.

Comment: It appears that from the outset the staff failed to take account of Conor's small size, and physical immaturity. In my view it would have been far more appropriate for him to have been initially assessed by a paediatrician, not an adult physician, and to have been admitted to the children's ward. I imagine that was also the opinion of the GP, which is why they originally referred him to the RBHSC.

[Dr Budd, in her evidence at the inquest, states that she referred Conor to the paediatric team, but was advised that because he was aged 15, he was not suitable for the paediatric ward' (087-029-134). She also states that 'his mother did not want him to go to the paediatric ward..' (087-029-134).]

There is some uncertainty about how much fluid he received between 13.00 and 14.00, around about the time of transfer to MAU.

[Dr Quinn, Medical SHO on MAU, in her evidence to the coroner states that at 'about 1.30 p.m.....I changed the fluid to 250 ml over 4 hours – again normal saline' (087-015-082)]

However, the fluid prescription chart (088-004-064), and the fluid balance chart (088-004-063) both indicate that the 250 normal saline was not started until 4.10 p.m., after the cannula had been re-sited. It is possible that some of the prescribed normal saline was given before 14.00.

Thus it appears that between about 11.20 and 14.00, he was probably given either 530 ml or 420 ml of fluid in the form of Hartmann's and IV Ciprofloxacin, but it may have been more if the normal saline had been started.

Comment: It is also possible that the original, deleted prescription of 1 litre of normal saline over 8 hours was actually started in error (which would have been twice the later-prescribed infusion rate), but I doubt this, as there is no record of normal saline being given at this time, [and the staff do not recall this].

1400-1610
At around 1400, the IV cannula tissued (extravasated) and IV fluids could then no longer be given. The IV was re-sited around 1610 and it appears that he was given

250 ml of normal saline, as prescribed, either started afresh or continued from before. There was a delay in re-siting the IV cannula, in spite of the apparent attempts by the nurses to get a member of medical staff to do this.

Comment: In my view this delay was not particularly significant. As stated above, Conor had already received at least 400-500 ml of IV fluid, which given that he was clearly not in a state of hypovolaemic shock, was a sufficient volume for initial fluid treatment. Thus although the delay in re-siting the cannula was not best practice, and would have been distressing for his family, I do not believe that this of itself contributed to his deterioration and death.

Comment: The diagnosis of urinary tract infection (UTI) seems to have been accepted but the evidence for this is lacking. A urine dipstick was done on his admission to the MAU and this is recorded in handwriting in the notes (088-004-045) as containing protein, blood, and ketones. There is no note about whether any nitrites or leukocytes were present. In paediatric practice, these items are nearly always tested for when there is any suspicion of a UTI, and these are far better indicators of whether a UTI is present than blood and protein. It does not appear that any attempt was made to get urgent microscopy done on the urine specimen.

Subsequently, a lab report on a specimen taken on 8th May 2003 was negative, with no significant numbers of cells or organisms on microscopy, and no growth on culture (088-004-127) thus ruling out a UTI.

Comment: Nonetheless if there was even a small suspicion of a UTI in an unwell patient, it would be justified to treat it prospectively with antibiotics while awaiting culture results. It should also be noted that the medical registrar, Dr Murdock, who saw Conor commented that he might have a viral illness in addition (088-004-045).

1830

The family were concerned about the development of an apparent rash on his abdomen, but this apparently disappeared by the time he was assessed by the medical registrar and was probably of no significance: (088-004-046). Around about that time the family were requesting a transfer to the RBHSC as they were clearly concerned about his condition. His case was discussed with the consultant adult physician on call, who agreed with his antibiotic and fluid management and suggested a review by a paediatric registrar.

Comment: This was the first time Conor had been seen by a paediatrician since arriving in Craigavon, even though it appears that one would have been available at any time.
[In his evidence to the inquest, the medical registrar Dr Murdock states that he asked his consultant Dr McEneaney to come and to assess Conor, but Dr McEneaney did not consider it necessary (087-025-125)]

2030

The paediatric registrar Dr Williams was taking a history from Conor's family when he appeared to suffer a brief generalised seizure that lasted only seconds: (088-004-048) A few minutes later while she was examining him he suffered a more prolonged seizure and stopped breathing.

[The evidence from Conor's mother and other family members given at the inquest suggests strongly that he may have been having seizures, possibly 10 to 12 between 1.00 and 8.00 p.m. (mother 087-002-021; grandmother 087-004-047; mother's boyfriend 087-010-063)]

Dr Smith, Consultant Paediatrician, also happened to be present on the MAU seeing another patient at the time and became involved in the resuscitation.

Comment: It is possible that the seizure witnessed by Dr Williams was the last of many, which had not been appreciated as such before this time by MAU staff. If he had been nursed on a children's ward, it is likely that there would have been more awareness of the need to observe for less obvious seizures.

[The coroner concluded at the inquest that significant seizure activity had taken place that afternoon (087-057-222)]

2045

Conor's second seizure was followed by a respiratory arrest and the medical staff present made appropriate resuscitation attempts. The airway was secured and bag and mask ventilation was instituted until an anaesthetist was able to attend and to carry out endotracheal intubation. It appears that oxygenation and cardiac output was largely maintained because of prompt resuscitation. The blood pressure was maintained with a good cardiac output. However the pupils were dilated and unresponsive to light from the outset.

Intravenous phenytoin was given to control the seizures. Aciclovir was added to his treatment because of the unlikely possibility of viral encaphalitis being the cause of his illness.

Comment: It appears that Conor's respiratory arrest was very promptly and appropriately managed, and the appropriate people were present at the time. It is surprising that he deteriorated so rapidly, when it appears that the paediatric registrar only a few minutes beforehand, did not consider that his condition gave cause for concern. The fact that his pupils became fixed and dilated so quickly, suggests that there was a severe brain problem preceding the respiratory arrest.

[Dr Williams, the paediatric registrar, in her evidence to the inquest, states that she did not think that Conor was having seizures during the 15-20 minutes when she was taking a history and examining him, until the two seizures that preceded the respiratory arrest (087-035-164). She also states that the family did not give a history of seizures during the afternoon (087-035-165). However these observations do not exclude the likelihood that he had a series of significant seizures before she arrived]

The choice of phenytoin as a first line anticonvulsant is unusual: normal practice then, as now, would have been to use an IV benzodiazepine drug such as diazepam or lorazepam. This normally works more rapidly than phenytoin, although its effects are not as long-lasting.

2100
An urgent CT scan was done and immediately reported by a consultant radiologist. The appearance was very abnormal, with a large porencephalic cyst on the left side of the brain and a smaller similar cyst on the right. There were appearances suggesting blood around the basal cisterns which could have been due to a subarachnoid haemorrhage

Comment: Although I am not qualified to comment on neuro-radiological appearances, it seems likely to me that most of the abnormalities seen on the scan were longstanding, and the presence of bilateral porencephalic cysts is what is sometimes seen in severe cerebral palsy. The occurrence of the subarachnoid haemorrhage, with blood appearing within the subarachnoid space possibly causing acute raised intracranial pressure, could reasonably have been interpreted as a cause for his acute neurological deterioration at the time.

[Evidence to the inquest from Dr Hicks, paediatric neurologist from Belfast, subsequently indicated that he had had a CT scan many years previously, and that on comparison, many of these abnormalities must have been long-standing (087-033-148)]

2200
Conor was admitted to the general intensive care unit at Craigavon. On admission he was deeply unconscious with a Glasgow Coma Scale score of only 3 out of 15 with no sedation. Observations stable but clearly needing ventilation. It appears that his condition on ICU did not change much overnight.

Comment: There does not appear to have been any discussion at that time of transferring Conor directly to the paediatric intensive care unit in Belfast, which happened later but with hindsight possibly should have happened earlier. It may have been that arranging the transport was impractical.

9th May 2003 – 1005
Conor was assessed on ICU on the morning ward round by the consultant anaesthetist. His neurological condition was considered to be very concerning and the decision was made to do brain stem tests, with a view to possibly withdrawing intensive care if he was thought to be brain-dead.

1100
Some suggestion of reflex motor response to stimulation, therefore could not be considered brain-dead.

1500

Serum sodium following admission to PICU noted to be high at 149 , having risen from 139 at the time of admission to ICU in Craigavon (088-004-114). Serum sodium had been normal on several occasions the previous day. I note from my brief (paragraph 96) that the serum sodium increased further following his transfer to the RBHSC.

Comment: It is uncertain why Conor's sodium rose significantly after his deterioration and collapse, but since it was normal prior to his collapse one can be certain that neither hyper- nor hyponatremia was any part of the reason for it. In my view it is possible that the concentration of sodium in his blood rose because he started to develop diabetes insipidus, the opposite of inappropriate ADH secretion, where the posterior pituitary gland starts to fail and does not produce sufficient ADH. This allows the kidneys to excrete pure water without accompanying sodium, and thus the total sodium content in the circulation rises. However it is impossible to make this diagnosis without more detailed biochemical information, and other experts may be in a better position to comment on this if appropriate, having regard to the Inquiry's terms of reference. In any event the hypernatremia does not appear to have been as a consequence of inappropriate management either before or immediately after his admission to intensive care.

1520
Discussion about transfer to PICU in Belfast. It appears that before PICU would accept Conor, they asked for a paediatrician to assess him and establish that he was sufficiently immature to justify admission to a paediatric unit. Dr McDonald, a paediatric staff grade, duly obliged and confirmed that he had the body habitus of an 8-9 year old child (088-004-057).

Comment: In my view it is very surprising that the staff needed a paediatrician to tell them that Conor was 'childlike'. A 15 year old boy who weighed only 22 kg, could not talk and was immobile, would quite obviously have been childlike to any observer.

1615
Problems with measuring urine output so urinary catheter inserted prior to transfer. 80 ml of urine measured as output (088-004-058).

Comment: Prior to this it had been impossible to measure his urine output. No urinary output is recorded at all on the ward fluid chart (088-004-063), and on the intensive care fluid chart (088-003-026 and 088-003-030) his urine output is recorded only in terms of 'incontinent +' or '+ +' until he was catheterised at 1700.

A urine collecting bag ('Uribag') was applied when he arrived in A&E, according to the nursing record (088-002-021). This is a plastic bag designed to fit over the penis, which adheres to the skin around the genitalia. Its main purpose is to collect a specimen for analysis, but can also be used to measure urine output. However, it is unreliable for this purpose as it usually leaks or becomes displaced. It is more suited to younger children. It is not a substitute for a urinary catheter. It appears that although the bag was applied, no-one measured its contents.

[According to the mother's deposition to the inquest, when Conor was admitted to the MAU, the 'urine bag... was now overflowing and despite numerous requests it was not removed' (087-002-019.)]

I have no records from the RBHSC PICU. I understand that I am not expected to comment on management there.

POST MORTEM FINDINGS

The post mortem confirmed the long-standing brain abnormality seen on CT scan (092-028-141). However, the most important finding was severe cerebral oedema, which was undoubtedly the cause of death. The subarachnoid haemorrhage found on CT scan may have been the result of the brain swelling, rather than its cause. The pathologist found nothing to show that fluid balance or electrolyte problems had contributed to his death.

The coroner's verdict was that brain hypoxia, ischaemia and infarction and seizures led to the fatal cerebral oedema (087-057-223).

COMPLIANCE WITH 'GUIDANCE ON THE PREVENTION OF HYPONATREMIA IN CHILDREN' ISSUED MARCH 2002

The guidance was issued by the Chief Medical Officer for Northern Ireland to the Medical Directors of all acute trusts, and a wide range of consultants within Northern Ireland including paediatricians, surgeons, anaesthetists, and emergency medicine specialists, but not adult physicians.

It is clear that the guidance should have been issued to the accident and emergency department of Craigavon Hospital, and the A2 poster displayed. As currently instructed, it is unclear whether this was done.

It is also unclear whether the guidance was issued to staff on the MAU, and whether the A2 poster was displayed. It seems clear that if young people under 18 were being cared for in the MAU, then the guidance would have applied to them and should have been adhered to on that unit. It is also unclear whether the Chief Medical Officer was aware that children in Northern Ireland were being looked after in adult units, and that their staff would therefore need to be aware of this advice.

I have been asked in the brief to comment whether, in Conor's case, the guidance was adhered to under a number of headings.

a. Baseline Assessment

The guidance states the following:
'Before starting IV fluids, the following must be measured and recorded:

- Weight: accurately in kg or a best estimate. Plot on centile chart or refer to normal range.
- U&E: take serum sodium into consideration.'

Comment: In Conor's case, his weight is recorded on the Emergency Department admission note (088-004-040) as approximately 22 kg. It is not clear whether this was from an attempt to weigh him, from his mother's estimate of his weight, or whether it was just a visual estimate. Certainly in Conor there would have been no point using normal centile charts as he was far from a normal child. An experienced paediatrician would have been able to make a reasonably accurate estimate of his weight just from looking at him. The weight at post mortem was 24 kg, so 22 kg appears to have been a close enough estimate.

His urea and electrolytes were measured on the blood tests taken shortly after arrival and these were close to normal – serum sodium level 138 mmol/l (088-004-118). These results are written in the MAU admission note at 088-004-038. It is unclear whether they were known about at the time the fluid resuscitation was started, but it is likely that they became known during the infusion. An arterial blood gas sample was also done, timed at 10.59, only a few minutes after his arrival in A&E (088-004-036.) The main purpose of this investigation is to assess the level of blood acidity (pH), oxygen and carbon dioxide, all of which give an indication of how critically ill the patient is. This test also gives a sodium level, which in this case was 135.7mmol/l. However this can be unreliable, and may differ significantly from the true sodium level from the sample sent to the lab, as seen here. The staff would have seen the blood gas sodium level, even before the lab result came back, and they may have considered this adequate to plan fluid resuscitation. This would be justifiable.

Conclusion: In respect of the baseline assessment, this aspect was complied with.

b. Fluid Requirements

The guidance states the following:
'Fluid needs should be assessed by a doctor competent in determining a child's fluid requirement. Accurate calculation is essential and includes:

- Maintenance fluid: 100 ml per kg for first kg of body weight plus 50 ml per kg for the next 10 kg plus 20 ml per kg for each kg thereafter up to a maximum of 70 kg.

- Replacement fluid must always be considered and prescribed separately, and must reflect fluid loss in both volume and composition (lab analysis of the sodium content of fluid loss may be helpful).'

Comment: regarding the stipulation that a doctor competent in assessing children's requirements should make the assessment, this was not complied with. Conor was seen initially by a non-specialist A&E doctor, briefly by an A&E consultant, and subsequently by an adult medical SHO and registrar. In my view, none of these

doctors are likely to have had the necessary skills, particularly in assessing a disabled child. My views on why he should have been assessed by a paediatrician are given below.

Here it is important to distinguish the meaning of resuscitation, replacement and maintenance IV fluids. Resuscitation fluids are given rapidly to restore the volume of the circulating blood when there are signs of impending or actual hypovolaemic shock of any cause. Replacement fluids are given to replace an estimated deficit in the total body fluid, i.e. dehydration, usually due to vomiting and/or diarrhoea, and are given over several hours. Maintenance fluids are given to replace anticipated ongoing losses from whatever source, and are usually continued until the patient can drink adequately. They are administered after, or at the same time as, replacement fluids.

It is not entirely clear how Conor's maintenance fluids were calculated. Resuscitation fluids (as opposed to replacement or maintenance fluids) are not mentioned in the guidance, but the quantity of resuscitation fluid prescribed, i.e. 10-20 ml per kg, was appropriate for his assumed body weight of 22 kg. However as this appears to have been given over a longer period, it was really being given as a replacement not a resuscitation fluid.

The initial maintenance fluid prescription of 1 litre 8 hourly, which is standard for a full size adult, was clearly inappropriate, but this was deleted and never acted upon. The subsequent corrected prescription was for normal saline, 250 ml over 4 hours followed by 250 ml over 6 hours, followed by 250 ml over 8 hours (088-004-064). This prescription equates to 750 ml over 18 hours, which if continued would have been 1000 ml over 24 hours. This is an average of 42 ml per hour, or in Conor's case 45 ml per kg per day. Taken over a 24 hour period this is actually quite a restricted quantity of fluid. However, it seems likely that it was written with the anticipation of him starting to improve sufficiently to take some oral fluid later.

If his maintenance fluid quantity had been calculated according to the formula given in the guideline, then the calculation (assuming weight 22 kg) for 24 hours' requirement would have been as follows:

100 ml per kg for the first 10 kg of body weight = 1000 ml

50 ml per kg for the next 10 kg = 500 ml

20 ml per kg for each kg thereafter (i.e. 2 kg over the first 20 kg) = 40 ml

Total 1540 ml

Total in 24 hours would therefore be 1540 ml (equating to about 63 ml per hour), which is significantly more than the 1000 ml 24 hour equivalent that was prescribed. Therefore it appears that the formula given in the guideline was not used to calculate his maintenance fluids. There is no documentation in the case records of how these quantities and rate were arrived at. In paediatric practice it would be usual to record how a rehydration regime was calculated. At 088-004-045, Dr Murdock writes 'd/w

paeds re rate' (i.e. to discuss with paediatric team about infusion rate) but there is no record of the outcome of any discussion.

However in the event, because of his acute deterioration, the full 24 hours of maintenance fluid was never given.

The quantity of replacement fluid required would have been difficult to assess because his fluid loss was not quantified in any way. There is no record on his ward fluid chart of any vomiting, any urine output, or whether or not he had his bowels open. In fact the output column is completely blank (088-004-063). Thus although an appropriate type of replacement fluid was chosen (see below), it was impossible to prescribe an appropriate quantity of replacement fluid without any quantification of his output.

In any situation where intravenous fluids are being given, it is necessary to have some estimate of output: this would include, vomit, stool, and most importantly, urine. If the urine output is inadequate, it may be because either: a) the patient's kidneys are failing, or b) because the fluid deficit has not yet been replaced, and so the kidneys are conserving fluid by reducing urine production. Further clinical and biochemical assessment should distinguish these. Further management will depend on this assessment. If urine output is low because of inadequate replacement, then the infusion rate needs to be increased.

The need for replacement fluids should have been assessed before the initial infusion was started, and then again at intervals during the day, by clinically assessing his state of hydration, and his urine output.

In the regime used in this case, the 'bolus' (i.e. 10 or15 mls/kg of Hartmann's) effectively became the replacement infusion. This should have been adequate to treat 'mild' dehydration. The continuation of maintenance fluids after that was probably necessary, given that Conor was not drinking. It is not clear that maintenance fluid was 'considered and prescribed separately' from resuscitation fluid, as suggested by the guideline.

As described above (p 5-6), there is uncertainty about how much fluid Conor actually received between 11.20 and 19.40. However, even if this had been correctly and accurately documented, the management would still have been non-compliant with the guidelines, as the correct method of calculation was not used

Conclusion: In respect of 'a doctor competent in determining a child's fluid requirement' should be involved, this was not complied with.

In respect of the prescription for the quantity of maintenance fluid, this part of the guidelines was not complied with, since the suggested method of calculation was not used, resulting in a lesser quantity being given.

In respect of the prescription for the quantity of replacement fluid, the guideline was also not complied with. There is no estimate of fluid output, no calculation of estimated replacement requirement, and there is confusion between resuscitation and replacement fluid.

c. Choice of Fluid

The guidelines state the following:

1. 'Maintenance fluids must in all instances be dictated by the anticipated sodium and potassium requirement. The glucose requirements, particularly of very young children, must also be met.

2. Replacement fluids must reflect fluid loss. In most situations this implies a minimum sodium content of 130 mmol per litre.

3. In resuscitating a child with clinical signs of shock, if a decision is made to administer a crystalloid, normal (0.9%) saline is an appropriate choice, while awaiting the serum sodium.

4. The composition of oral rehydration fluids should also be carefully considered in light of the U&E analysis.

Hyponatremia may occur in any child receiving any IV fluids or oral rehydration. Vigilance is needed for all children receiving fluids.'

Comment: In Conor's case, it appears that medical staff were aware of potential losses from continued vomiting, although he did not appear to have had diarrhoea. His blood glucose was checked by an immediate test on arrival, and subsequently with the U&E blood test, and was normal and therefore glucose requirements did not appear to be a problem.

Regarding the type of fluid used, it is unusual to use Hartmann's solution as initial resuscitation fluid in children. Hartmann's solution is commonly used in surgical cases, both in children and adults, and sometimes in trauma, but not normally in paediatric medical cases. Its composition is sodium 131 mmol per litre, plus a number of other electrolytes including potassium, as compared to 0.9% saline which contains 150 mmol per litre of sodium and no other electrolytes. Hartmann's is considered to be isotonic, and therefore is appropriate as an acute resuscitation fluid, and complies with the guidelines. I imagine that it was used in the Emergency Department simply because it was the most readily available fluid, and was continued thereafter as the fluid replacement continued. In most situations, in practice it does not much matter which of these two isotonic crystalloid fluids are given.

Later, for reasons that are not clear, they switched from Hartmann's to normal saline. This may simply reflect what was available on MAU as opposed to A&E.

The composition of oral hydration fluids does not appear to have been considered but at that time Conor was not taking significant amounts of fluid by mouth anyway.

Conclusion: In respect of the type of maintenance fluid used, although not normal practice, the guidelines were complied with. To take each bullet point from the guideline in turn:

1. Anticipated sodium and potassium requirements were accounted for in that a solution containing adequate amounts of these electrolytes, Hartmann's, was used. Glucose requirement was not a problem.
2. Similarly, Hartmann's, and subsequently normal saline, have a sufficiently high sodium concentration and were therefore appropriate choices by this guideline.
3. Regarding resuscitation fluid, they chose to use Hartmann's rather than normal saline and therefore in this respect they did not comply with the guidelines. However, as Conor was not in clinical shock, this stipulation in the guideline was not applicable. The 'resuscitation' fluid given was effectively replacement fluid.
4. As Conor never recovered sufficiently to take oral fluids, this is irrelevant.

d. Monitor

The guidelines state:

- 'Clinical state: Including hydrational status. Pain, vomiting and general well-being should be documented.

- Fluid balance: Must be assessed at least every 12 hours by an experienced member of clinical staff.

Intake: All oral fluids including medicines must be recorded and IV intake reduced by equivalent amount.

Output: Measure and record all losses (urine, vomiting, diarrhoea, etc) as accurately as possible.

If a child still needs prescribed fluids after 12 hours of starting, their requirement should be reassessed by a senior member of medical staff.

- Biochemistry: Blood sampling for U&E is essential at least once a day – more often if there are significant fluid losses or if clinical cause is not as expected.

The rate at which sodium falls is as important as the plasma level. A sodium that falls quickly may be accompanied by rapid fluid shifts with major clinical consequences.

Consider using an indwelling heparinized cannula to facilitate repeat U&Es.

Do not take samples from same limb as the IV infusion.

Capillary samples are adequate if venous sampling is not practical.

Urine osmolarity/sodium: Very useful in hyponatremia. Compare to plasma osmolarity and consult a senior paediatrician or a chemical pathologist in interpreting result.'

Comment: Conor's clinical state, particularly his degree of dehydration was not well monitored. In the initial Emergency Department assessment, he was said to be dehydrated on examination but the physical signs indicating this are not described (088-002-020). Subsequently on his admission to the MAU, he was said to be 'dry', but again the signs are not listed (088-004-045). His pulse and blood pressure were normal so he was not in hypovolaemic shock as a result of dehydration. No attempt to quantify his urine output prior to arrival at hospital was made.

There is very little comment anywhere in the medical or nursing notes as to whether he actually continued to vomit or not. If he had continued to have frequent troublesome vomits, then consideration should have been given to placing a nasogastric tube, so that gastric losses could be accurately measured. This does not seem to be mentioned anywhere.

[In the mother's evidence to the inquest, there was no mention of continuing vomiting after admission to MAU (087-002-019).]

Similarly, in order to accurately measure his urine output ideally a urine catheter should have been placed and this happened only much later when he was on intensive care.

However, both these procedures are distressing for a child and it is entirely understandable that the staff chose not to go ahead with them.

To make a full assessment of a child's hydration status, I would expect the following to be examined and documented: urine output, urine concentration either observed or measured, vital signs, presence or absence of sunken eyes, dry tongue, loss of skin turgor. Also the patient's conscious level and responsiveness is important, although in Conor this would have been difficult for a doctor unfamiliar with him to assess. None of these were recorded apart from a mention of him being 'drowsy' on admission to MAU (088-004-038).

The fluid balance was poorly recorded in that although IV intake was recorded on the chart, there is no output recorded at all. There is no mention of oral fluids and it is likely that Conor was too unwell to drink. No urine output was measured.

Regarding biochemistry, a blood test for U&E was taken appropriately on admission, and he deteriorated and got admitted to ICU before 12 hours on IV fluids had elapsed, and therefore it cannot be assessed whether this aspect of the guidelines was complied with.

No urine specimen was taken for osmolarity or biochemical analysis. According to the nursing record (088-004-091), a urine specimen was taken at 1.30 p.m. and a dipstick test was done, which showed protein +, blood + and 'large ketones'. The specimen appears to have been sent to the lab for microbiological analysis to look for a UTI, but not for biochemical analysis An assessment of urine concentration (i.e. urine specific gravity), was not done. Even without plasma and urine osmolarity, this is a useful indication of degree of dehydration. The small amounts of blood and protein are probably insignificant. The presence of a large amount of ketones in the urine suggests significant dehydration, but may have other causes and this test is not what is suggested in the guidelines. More specific biochemical analysis would have helped quantify the degree of dehydration, and the ongoing requirement for fluid replacement.

The failure to make a more accurate assessment of his state of hydration could have led to either excessive or inadequate fluid replacement, or to replacement with fluid that contained an inappropriate electrolyte content.

Conclusion: The monitoring of Conor's clinical state was inadequate and did not comply with the guidelines. His output fluid record was very poor and did not comply with the guidelines.

Comment: In Conor's case, the only senior member of medical staff who was asked for advice was a consultant physician (in fact a cardiologist, Dr McEneaney) up until his seizure and acute deterioration, apart from a very brief review by the A&E consultant Dr Kerr. Even though it seems likely that there was a consultant paediatrician, and/or consultant anaesthetist on site and available to contact, this did not happen until the acute deterioration. In fact even a paediatric registrar's advice was not sought until he had been in hospital for about 9 hours.

It would have been important for him to be reviewed by a more senior member of the team for a number of reasons, including the question of whether there was seizure activity, and the family's apparent dissatisfaction. In the context of fluid management, a more senior doctor, particularly one with experience of young people with cerebral palsy, may have been able to make a better clinical assessment of his state of hydration, and may have asked for other action to be taken, including accurate documentation of fluid balance, urine specific gravity or osmolarity, further blood biochemistry, etc.

Conclusion: In respect of seeking advice from an appropriate senior member of medical staff, this aspect of the guidelines was not complied with.

GENERAL COMMENTS

1. **How unwell was Conor?** Although it is difficult to be certain with the limited information available, in my opinion it is quite likely that throughout this illness Conor was considerably more unwell than any of the medical staff realised. This tendency to underestimate his illness was probably a direct result of his cerebral palsy, with profound neurological abnormality, and his inability to communicate. A child with verbal skills would have been able to tell his parents how unwell he felt, or his inability to speak because of his level of illness would have caused concern. A child with normal motor function would have been able to indicate by their movements their level of distress. Both these would have been difficult with Conor. I believe this underestimate of his level of illness probably occurred both before his admission to hospital, and in his passage through the Emergency Department, MAU and possibly even subsequent later assessment by a paediatrician.

My reasons for stating this is the fact that his respiratory arrest with fixed dilated pupils was clearly not the result of any acute cardiac or respiratory problem, but rather due to a neurological deterioration which must have been going on for some time beforehand.

2. **Seizure Activity.** There seemed to be a lot of uncertainty whether, and to what extent, Conor was having seizures before his acute deterioration. According to the brief, one of the doctors who attended him in the Emergency Department apparently thought he was having brief seizures while his cannula was being adjusted, but this is not recorded in the medical notes. The GP who saw him on the day before admission apparently noted that he was arching his back. Abnormal movements like this in children with cerebral palsy can be very difficult to interpret in that they may sometimes be abnormal dystonic movements as a result of distress, or they may be seizures. It is often extremely difficult to distinguish these two types of movement.. A parent is often the best judge. It is possible that Conor was having sub-clinical seizures which did not result in any obvious abnormal movements for some time before his deterioration.

3. **Type of fluid used.** It is worth commenting that because Conor was on an adult unit, they would probably not have considered using 0.18% saline for his fluid treatments in any case. This has never been used as a standard fluid in acute adult medical patients as far as I am aware. Therefore the fall-out from the death of Raychel Ferguson from hyponatremia, would probably not have affected the MAU at Craigavon and would not have prompted any change in their fluid management policy. Even if the staff were aware of this, they probably would have considered that it was something that did not apply to them.

4. **Record-keeping**. There are a number of inadequacies in the standard of record-keeping in the case records, although probably no worse than one would find in many hospital admissions. However it is worth focussing on the fluid balance (intake/output) chart at 088-004-063, which is particularly poor:

   a. It is unclear when the first normal saline infusion was started, as discussed above.

   b. It is unclear how many 110ml Hartmann's infusions were given, and what total volume had been administered by the time the cannula tissued around 14.00.

c. In the 'volume in' column, at 5 p.m., 250 ml is recorded, but this doesn't appear to relate to any entry in the 'volume erected' column.

d. There is no record of what volume had been infused by 7.40 p.m., when a new 250 ml bag was erected, no record of whether this was actually given, and subsequently no record at all of what his total fluid input had been by the time he was admitted to ICU at 10 p.m. A new chart was started at that time (088-003-026) but this contains no record of what had gone in previously. It would be important for ICU staff to know this in order to plan his subsequent fluid management.

5. **Appropriateness of ward**. Although not specified in my brief, I would like to comment on the appropriateness of children under 16 being admitted to adult units under any circumstances. This is something about which there has been considerable discussion in recent years, and the trend throughout the UK has been to increase rather than lower the age threshold of admission to a children's ward. In 2003, it was unusual for district general hospitals to have 14 as the age cut off for choosing between an adult and children's ward. For most it was 16, although for some it may have been as high as 18. The reasons for this policy at Craigavon are not clear but may have been related to bed availability. There are a number of reasons why it is now considered by most people to be preferable to admit teenagers between 14 and 18 to a children's rather than adult ward, when no inpatient adolescent unit is available. This applies to all young people, not just those with disabilities like Conor. These could be listed as follows:

1. **Medical management**. More appropriate medical management could be expected from paediatricians as opposed to adult physicians with regard to drugs, fluids etc. This is particularly relevant to fluid management. As a good example of this, recent NICE guidelines of the management of diabetic ketoacidosis (DKA), specify very different fluid management regimes for young people under 18 as compared to adults with DKA (Ref 2). This is because young people have a different physiological response to certain types of fluid and electrolyte regimes, even up to the age of 18. For this reason, many hospitals now admit all young people whatever their level of maturity with DKA to the children's ward, up until their 18th birthday.

2. **Child Friendly Environment** Adult wards can be frightening and disturbing places for young people to be admitted. They are likely to be surrounded by old people many of whom may be terminally ill. When given the choice between this, and being on a ward surrounded by younger children, most adolescents prefer the latter. There is a significant literature on the expressed preferences of adolescents in hospital.

3. **Practical Procedures** Even at 18, some young people get very anxious about practical procedures, and these are done in a different way on a children's ward to an adult ward, e.g. greater use of local

anaesthetic, sedation etc. Also the staff are likely to be more skilled in doing these procedures on young people.

4. **Resident parents** Most adult wards will not allow a parent to stay overnight with a child however unwell they are. On children's wards it is the norm for this to happen. Adolescents, even quite mature young people, often want a parent with them when they are unwell.

5. **Care of the Family** The practical arrangements and the philosophy on children's wards are more orientated towards supporting and caring for the parent than they are towards supporting relatives on an adult ward.

In conclusion, many of these factors apply in Conor's case. I would like to list speculatively some of the differences in treatment that I think may have occurred had Conor been admitted directly to a children's ward, either at Craigavon or at RBHSC.

1. **Urine Test:** As mentioned above, on a children's ward greater attention is given to the early diagnosis of a UTI and more appropriate urine testing sticks are used.

2. **Choice of Antibiotic:** As mentioned above, on a children's ward it is likely that he would have been given a different antibiotic which would have required less volume of fluid.

3. **Fluid Type:** As discussed above, on a children's ward, after the withdrawal of 0.18% saline, it is likely that he would have been treated throughout with 0.9% saline. This would have been given both for immediate resuscitation and for maintenance fluids.

4. **Cannula**: The issue with re-siting the tissued cannula would probably have been addressed more speedily, and possibly with less distress to Conor.

5. **Observation**: Conor's seizures may have been noted and acted on sooner by paediatric nursing and medical staff.

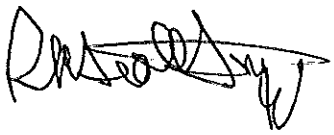
6. **Family**: There may have been better support for the family. It is likely that staff would have had experience of dealing with parents of severely disabled children.

OVERALL CONCLUSION

There were a number of inadequacies with Conor's management, and areas where there was poor compliance with the NI fluid management guidance.

However in my opinion overall, the fluids management was not the cause for his acute deterioration and subsequent death. In this respect, his case may differ from some of the others considered by the inquiry.

Declaration
I declare that the above is my own true opinion having studied all the relevant documents supplied to me, given to the best of my knowledge and ability. I have no personal interest in supporting any particular point of view, I do not personally know any of the clinicians involved in this case and I have never worked in Northern Ireland.



Dr Robert Scott-Jupp
19 September 2013

References:

1) Advanced Paediatric Life Support manual 5th edition (ALSG 2011). Page 95
2) National Institute for Health and Clinical Excellence (2012) Clinical Guideline 15: Type 1 diabetes in children, young people and adults.