Northern Ireland Inquiry into Hyponatraemia Deaths

Paediatrician’s Expert Report submitted 11 April 2011
Amended 12 June 2012

Introduction:
I have been requested by the Inquiry Chairman, Mr John O’Hara, to give my expert opinion into the circumstances leading up to and following the death of Claire Roberts on 23 October 1996. I have had access to and have studied the following documents:

1. Brief for paediatric expert compiled by the inquiry
2. Copies of the hospital notes from the Royal Belfast Hospital for Sick Children
3. Copies of the Coroner’s papers
4. Copies of all the papers held by the Police Service of Northern Ireland relating to this case
5. Detailed witness statements from all the relevant clinicians. These did not become available until May 2012, and I have added a note designated with a * where these statements have altered my original conclusions.

My credentials:
I am a Consultant General Paediatrician in a small District General Hospital in England. I qualified in 1980 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 of conditions relating to this case. As I took up my Consultant post in 1992 I am familiar with the standards of practice current in 1996.

My report will consist of responses to the specific questions asked by the Inquiry Chairman. I will not reiterate in detail the facts of the case that have already been spelt out numerous times in various documents available to the inquiry.

Background
Briefly, Claire Roberts was a nine year old girl who was admitted to the RBHSC on 21 October 1996, deteriorated over the next two days and was declared brain dead on 23 October 1996 when life-support was withdrawn. Many years later questions were asked about the cause of her death which resulted in the Inquiry deciding to include this case in its Terms of Reference.

Responses to questions set out in paragraph 94 of the brief:

i) Was the decision to admit Claire under the care of a Consultant Paediatrician rather than a Consultant Paediatric Neurologist appropriate?

Yes. The normal procedure in most children’s units then, as now, is for new acute admissions to be admitted under the care of the acute General Consultant on take whatever the nature of their problem, unless they are already known to one of the specialists. It is then the responsibility of the admitting Consultant to ask for their care to be taken over by a specialist later if necessary. It was entirely appropriate that Claire should have been cared for by a General Paediatrician.

ii) a) Was the differential diagnosis made by the admitting Paediatric Registrar Dr O’Hare on 21.10.96 that expected of a competent registrar?

Dr O’Hare’s admission notes are clear and competently set out. The important points in the history are clear, and a competent clinical examination recorded. Differential diagnosis was “viral illness”. “Encephalitis” was written and then crossed out. This should be regarded very much as a preliminary assessment. Viral illness and encephalitis are not mutually exclusive. With hindsight, a diagnosis such as Encephalopathy and/or Status Epilepticus could have been included but the fact these were not written down does not mean they were not considered.

b) Were the investigations proposed at the time adequate?

Although these days we would be inclined to do more investigations, I think even by the standards at the time initial investigation was somewhat limited. In a child who was clearly unwell, with altered conscious level, I would have expected to see more extensive biochemical tests including liver function tests, calcium, glucose and ammonia. If there was any question of accidental intoxication, toxicology could have been added. As far as I can tell, the only blood tests requested were a full blood count, urea and electrolytes. The question of whether a CT scan should have been done is discussed later. It was appropriate not to do a lumbar puncture: it would have been potentially hazardous in this situation because of the risk of coning.

c) Was the type, volume and rate of IV fluid prescribed at the time appropriate?

The IV fluid given was 0.18% Sodium Chloride in 4% Dextrose. This was absolutely the standard IV fluid given to most children needing fluids for any reason in 1996. This policy has changed over the last few years. Although now policy is universally to give either 0.45% or 0.9% Sodium Chloride, there would have been no reason in these circumstances to have deviated from the normal policy. Even when the
results of the electrolytes were available and the low sodium of 132 was noted, I believe at the time most practitioners would have continued with 0.18% Saline. The quantity prescribed was 65mls per kg per 24 hours. I believe that this was appropriate as at the time there was no particular reason to impose fluid restriction as a means of preventing Cerebral Oedema. There were various methods of calculating fluid requirements in use at the time, but they would all have resulted in a broadly similar quantity to this. Then as now, this is an acceptable quantity of IV fluid to give in this situation. From the history, Claire had been vomiting and if she was developing a gastroenteritic illness she may also have potentially developed diarrhoea and therefore been at risk of dehydration. Severe fluid restriction at that stage could potentially have been harmful. She was clearly not able to drink. Only later should fluid restriction have been considered.

iii) a) Were the blood results obtained on 21.10.96 abnormal and should they have prompted reassessment?

It is important here to distinguish between what is “abnormal” and “unacceptable”. Frequently in paediatrics results are received that are outside the laboratory’s normal range. For very good reasons, clinicians often choose not to act on these technical abnormalities because the deviation from the normal rate is clinically acceptable under the circumstances. In acute paediatrics, for a number of reasons, it is very common to see a laboratory sodium well below the normal level of the range, i.e.: below about 135mmol/L. This can happen in a range of acute illnesses, sometimes due to a mild degree of inappropriate ADH secretion. It generally resolves itself as the underlying condition is treated and improves, and does not require any immediate response. In these circumstances it would have been appropriate not to have acted on a sodium level of 132. The textbook definition of ‘hyponatraemia’ is less than 130 mmol/L.(1) These days we might have considered giving a more concentrated solution of Sodium Chloride but that would not have been standard practice in 1996.

b) At what times should the electrolytes have been checked?

Although the result of 132 mmol/L is acceptable, it is my view that in the context of her lack of clinical improvement and the absence of a specific diagnosis to explain the low sodium level, it should have been repeated before 24 hours. It would have been reasonable to repeat it about twelve hours later. If, as is likely, a substantial fall had been noted then it may have been possible to intervene before the Cerebral Oedema became apparent. Having said that, standard practice at that time was to check serum electrolytes on children receiving IV fluid as a routine only once every 24 hours. Even now, following the National Patient Safety Authority (NPSA) alert about the use of hypotonic IV fluids in children which was issued in 2007, the advice is to check electrolytes four to six hourly only if the serum sodium level is below 130 (1). Thus even today a sodium level of 132 would not have warranted a repeat within six hours. In Claire Roberts’ case, although the low sodium in itself should not necessarily have mandated a repeat within twelve hours, in the context of a child who is not improving I believe that repeat blood tests should have been done earlier on 22.10.96 and, as well as checking the electrolytes, a number of other tests to try and explain her symptoms should have been considered at the same time.
iv) a) What was the reasonableness of Dr Sands' diagnosis of Status Epilepticus on 22.10.96?

Non-convulsive Status Epilepticus is always a difficult clinical diagnosis. Perhaps surprisingly, it is possible for a child to be constantly fitting and look relatively well without any obviously visible abnormal movements or vital signs outside the normal range. However, there is always a change in conscious level and alertness, and these latter signs were apparent in Claire Roberts at that time. Clearly Dr Sands' diagnosis was influenced by the fact that Claire had a history of epilepsy, had not been on regular anti-convulsants when she presented, and there was evidence from the history that she had a recent viral infection. The history of vomiting and exposure to a relative with a similar gastric illness would have suggested she could have had an entero-viral infection which is recognised to trigger seizures in susceptible children. This diagnosis was not unreasonable, but other differentials probably should have been considered at the time. This would include encephalitis or encephalopathy, or drug intoxication. Surprisingly, from the notes, there is no record of a discussion about attempting to obtain an immediate electroencephalogram (EEG). This is the only investigation that can make a firm and unequivocal diagnosis of non-convulsive status. Obtaining urgent EEG's can sometimes be difficult and it may well be that no technician or equipment was available at the time to do this, in which case it would have been acceptable to treat on the basis of presumptive diagnosis. However I would have expected this lack of availability to have been recorded. I would be surprised if no EEG service was available at all at the time in a large tertiary children's hospital with a paediatric neurology service.

* In his 2012 witness statement Dr Webb states that there was no urgent EEG service available at the RBHSC at the time. My criticism of the failure to obtain an EEG to confirm the clinical diagnosis of non-convulsive status may therefore be unjustified

b) Was using rectal Diazepam appropriate?

Rectal Diazepam was standard treatment at the time to treat any kind of acute epileptic episode including non-convulsive status. It is usually rapidly effective, so much so that the immediate improvement seen in non-convulsive status following Diazepam is almost diagnostic. Paradoxically, although Diazepam is a sedative, when given in these circumstances it actually causes the child to wake up and become more alert. These days we would probably use buccal Midazolam inserted into the mouth, or IV Lorazepam, but neither of these were in widespread use in 1996.

c) Was the referral by Dr Sands to Dr Webb, Consultant Paediatric Neurologist, reasonable?

It is entirely reasonable to involve a tertiary Paediatric Neurologist in circumstances where there is a difficult diagnosis of non-convulsive status to be made. However, there are two concerning problems with this referral: firstly,
there is no record that Dr Sands discussed the case with the Paediatrician under whose care Claire was admitted, Dr Steen. According to Dr Sands’ witness statement, Dr Steen was unavailable and, if true, as she was the Consultant responsible for that child, that is certainly unacceptable. These days, most hospitals have specific protocols for consultant referral to avoid these sorts of problems but there may have been no such protocol in place at the RBHSC at that time. As a minimum, a telephone call to Dr Steen should have been made under the circumstances, and given that it was during the working day, it would have been reasonable for Dr Steen to have seen this child who was clearly unwell and causing diagnostic difficulties. Secondly, it was not made clear at the time whether Dr Webb was being asked just to give an opinion or to take over care of the child. This is discussed further below. Whether it was appropriate for Dr Sands to consult Dr Webb on his own initiative depends to some extent on his seniority at the time. This is not clear from the documents. He is described as a Registrar as opposed to a Senior Registrar, and in 1996 the Senior Registrar Grade was still in existence. At that time, the Senior Registrar Grade often functioned virtually as Consultant and took on consultant-level responsibilities. If Dr Sands was indeed at this “pre-Consultant” level it may have been appropriate for him to take this on but if not he should certainly have discussed it with Dr Steen.

* From the 2012 witness statements, it is clear that Dr Sands was a relatively junior registrar and should not have been making consultant-level decisions.

* Dr Sands in his 2012 witness statement recalls that he did speak to Dr Steen some time on the day of 22/10/96, although he cannot recall when or what was said. This is not recorded anywhere and Dr Steen has no recollection of this discussion. It remains a concern that there was no documentation of the decision at consultant level to involve Dr Webb.

**d) Was the continuing IV fluid therapy appropriate?**

Given that Claire was clearly not alert enough to drink for herself, it was appropriate to continue IV fluids. As mentioned above, I believe the electrolytes should have been checked again in which case the 0.18% Saline may have been changed to a more concentrated solution. By today’s standards she would have been started on 0.45% or 0.9% Saline anyway and therefore there would have been no need for a change at that point even with the knowledge of the low sodium level. At that stage there was no particular reason for the clinicians to suspect Cerebral Oedema which would have mandated fluid restriction.

**v) a) Was Dr Webb’s note that the Sodium result of 132 was “normal” appropriate?**

As mentioned above, this level is technically abnormal but acceptable which is presumably what Dr Webb meant. I agree that a level of 132 would not in itself have resulted in any seizure activity or decrease in conscious level.

**b) Should any action have been taken regarding this biochemical status?**

As mentioned above, it should have been repeated but by the standard procedures at the time the IV fluids would not necessarily have been changed.
c) Was the apparent error in phenytoin prescription significant?

No. I think this error is irrelevant. Phenytoin is a drug that is handled very differently by different individuals in an unpredictable way and therefore the starting dose suggested in the literature is somewhat arbitrary. The important thing is to check blood levels at intervals after starting medication to ensure that they are within an acceptable range. In this case this was done as appropriate and the level was acceptable, and one can confidently say that this error was of no relevance.

d) Was the anti-convulsant therapy ordered by Dr Webb appropriate?

This question may be best answered by a Paediatric Neurologist who was in practice at the time. To me, it appears to be an appropriate use of anti-convulsant drugs that were in common use at the time and which are known to be effective. However, as mentioned previously in the context of the initial rectal Diazepam, lack of any improvement in Claire’s conscious level, in spite of what should have been adequate treatment of non-convulsive Status Epilepticus, should in my view have prompted a reassessment of the underlying cause.

e) Should Dr Webb have been aware that sodium metabolism may have been affected by an illness of this type?

Yes, any General Paediatrician or Paediatric Neurologist should have been aware that acute cerebral illness can result in the syndrome of inappropriate antidiuretic hormone secretion leading to hyponatraemia, and also that hyponatraemia in itself can cause Cerebral Oedema with its resulting neurological symptoms. However, in the context of a child admitted under an acute General Paediatric Team who was acutely unwell, I do not believe that, either then or now, it would have fallen to the Consultant Paediatric Neurologist to take the lead in IV fluid management. This is very much within the remit of the General Paediatrician. The Neurologist should have been aware of any abnormalities, but responsibility for checking the electrolytes and actually prescribing the fluids should have fallen with the General Paediatric Registrar or Consultant. Obviously if Dr Webb had been aware of a falling sodium level before it was eventually checked then this would have prompted a change in Clare’s management.

f) Was Dr Webb’s request for use of intravenous Sodium Valproate appropriate?

Sodium Valproate had been available for many years as an oral anti-convulsant and was not originally intended for use in the treatment of acute status. In 1996, an intravenous preparation had been introduced relatively recently and there was a vogue for using it as a second, third or forth line anti-convulsant when other drugs appeared to have failed. Since then it has rather fallen out of use because of other drugs becoming available. At the time therefore, I think this was an appropriate intervention.

g) Should Dr Webb have taken further action after recording Claire’s neurological state at 1715h on 22.10.96?

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Yes. It appears that at this point there was some uncertainty as to who was taking ongoing responsibility for her. I believe that Dr Webb should have made it clear at the time whether he had taken over her care completely and was prepared to be consulted about all aspects of the treatment or whether he expected her ongoing acute management to be in the hands of the General Paediatric team, with him being available for specialist advice only. He should have spoken directly to the Paediatric Registrar or Consultant on the general team that was on call that evening, or possibly have discussed her directly with the Consultant on-call for Paediatric Intensive Care at the time.

vi)  
a) How should the transfer of care have taken place and should it have been recorded?

As above, it should have been either by direct Consultant conversation or experienced Registrar to Consultant. It should have been recorded in the notes by the team requesting the transfer of care.

b) How would this normally be communicated to the medical and nursing team?

This process would vary from hospital to hospital and it is not clear what procedure was standard practice at the RBHSC at that time. By today’s standards, it should have been formally documented and the Consultant’s name attached to the patient should have been changed. Even then, all medical and nursing teams should have been made aware of this at their respective handover meetings.

c) Who should have had responsibility for Claire’s care after 5pm on 22.10.96?

This should have been agreed by the teams but I would have expected the on-call Paediatric Team and either Dr Steen or her General Paediatric colleague who was on-call that evening. The General Paediatric Registrar on-call would then have had prime responsibility.

d) To whom should that individual be accountable?

The on-call Registrar would have been directly accountable to the on-call Consultant or to the General Consultant responsible for that patient.

e) How do teams know who has continuing responsibility?

When a child is referred from the admitting team to a Neurologist, ideally, as discussed, it should be documented in the notes and discussed at handovers. It is not at all clear what happened in this case.

f) Is the omission of any record in the notes of communication between Dr Webb and Dr Steen of any concern?

Dr Webb should have communicated his concerns to a senior on-call general colleague, either a Consultant or experienced General Registrar, or alternatively made it quite clear that the neurology team (i.e.: Dr Webb and his own juniors and Consultant colleagues) were taking over her care fully. He should then have
ensured that all of the neurology team on-call that evening were aware of the
details of the case.

vii) a) Was the assessment and reaction of the on-call paediatric team Dr Bartholome and
Dr Stewart at 2100h on 22.10.96 appropriate?

No. A further seizure in spite of having received a considerable amount of anti-
convulsant medication should have prompted reassessment. Her blood tests were
repeated but there is no record of a repeat neurological examination. There is no
record in the notes that the Registrar on-call actually saw and re-examined the
child which, given her deterioration, I believe would have been appropriate. Even
in 1996 it would not have been appropriate for a relatively inexperienced SHO to
manage this child without a more senior doctor seeing her.

b) Was the action following receipt of the result of serum sodium 121 appropriate?

It appears the SHO received telephone advice from the Registrar and was advised
to restrict the fluids further. As above, I believe that the Registrar should have re-
examined the child with such a rapid fall in serum sodium without any other cause.
I believe that more severe fluid restriction should have been imposed at that point.
Even if there had been no ongoing losses through copious vomiting or diarrhoea,
she had already received a reasonable volume of fluid in a day and it may have
been appropriate to stop IV fluids completely. The usual advice is to allow the
serum sodium to rise by no more than 2 mmol/L per hour, the maximum safe rate.
Also, once hyponatraemia had been diagnosed, it would have been advisable to
check simultaneous urine and blood osmolality, as this would have helped give
support to or refute a diagnosis of inappropriate ADH secretion. Inappropriately
concentrated urine compared to ‘dilute’ blood would have suggested this.
However by this stage all this may have made very little difference to the eventual
outcome.

c) Should the resident team have involved a Consultant?

Undoubtedly yes. This was a serious situation. Even though thresholds for calling a
Consultant these days are considerably lower than they were in 1996, it is my
view that this child was sufficiently ill, with a number of problems that were not
improving, particularly as it was still relatively early in the night, informing a
Consultant would have been appropriate. It is not clear whether this did not
happen because the on-call team were uncertain whether to contact Dr Steen or
Dr Webb, but in any event one or other should have been contacted.

d) Should any action have been taken between 2100h and 2330h to prevent the latest
sudden collapse and should the team have been able to predict the devastating outcome?

With hindsight, it is likely that Cerebral Oedema was already beginning to develop
by the time the blood test for serum sodium was taken at 2130h. It is very difficult
to be certain whether any action taken at that stage would have made any
difference to the outcome. As mentioned, more severe fluid restriction would
have been appropriate but this may not have been enough to prevent the collapse
which happened only three or four hours later. I do not believe the on-call team at
5pm would have had any idea as to the seriousness of the underlying problem, and I do not believe they would have been aware of the potential for collapse. It must be remembered Cerebral Oedema from hyponatraemia was and remains a rare condition and it is likely that none of the doctors involved had ever seen it before. However, once the severe hyponatraemia was known about, as well as informing the Consultant on-call it would have been appropriate to inform the Paediatric Intensive Care Unit that there was a child on the Ward who could acutely deteriorate. By today’s standards, this would be normal practice but in 1996 the threshold for informing PICU about ill children on the Ward was considerably higher so this may not have been inappropriate at the time.

viii) Was the neurological observation chart satisfactory and should its observations have prompted earlier medical review?

A standard neurological observation chart was kept and appears to have been appropriately recorded. Later during the night, no record of limb movements are recorded but this is presumably because Claire was sleeping and not moving spontaneously. The fall in GCS score from 9 to 7 had in fact happened earlier in the afternoon and the fall from 8 to 6 occurred around 9pm which is when she deteriorated anyway so the chart in itself would not necessarily provoked earlier medical review.

ix) a) Should an earlier CT scan have been sought, what effect might this have had?

By today’s standards, undoubtedly a CT should have been requested on admission because of the finding of a change in conscious level and focal neurological signs. However, in 1996 the situation was very different and the threshold for requesting a CT scan was considerably higher for three reasons. Firstly, scanners then were less widely available and in this case it appears it would have involved an ambulance transfer from the RBHSC to the neighbouring Royal Victoria Hospital where the nearest scanner was. This in itself presents a risk and is potentially hazardous. Secondly, in those days it took much longer to produce a scan and because of the need for the child to lie still during a procedure it often required either sedation or a general anaesthetic. This in itself added to the risk and difficulty of getting a scan. Thirdly, the scan quality images were not as good then as they are now and important features could be missed. Even now, in early cerebral oedema, the CT scan can appear normal. A decision to request a CT scan was a difficult one for the staff involved. Clearly by the time Claire had collapsed with respiratory arrest it was mandatory. Even if she had had a scan done earlier on 22.10.96 it is by no means certain that it would have showed the Cerebral Oedema that caused the collapse. However, given the uncertainty of her diagnosis, before the hyponatraemia was recognised, CT scan would certainly have helped rule out a number of other possible diagnosis, e.g.: cerebral haemorrhage, infarction or acute hydrocephalus.

x) a) At what stage should Claire have been transferred to PICU?

With hindsight and by today’s standards she should certainly have been admitted to PICU with a GCS as low as 6. However, those standards did not apply in 1996.
It was not uncommon for children who these days we would consider to be far too ill to remain on a general children’s ward to nonetheless remain there. PICU beds were less readily available and at that time very few hospitals had a High Dependency Unit, which these days is used as a ‘half-way house’ between the general ward and the PICU. In 1996 the need for artificial ventilation would probably have been a pre-requisite for admission to PICU. However, it would have been appropriate to discuss her earlier with a PICU Consultant who could then have assessed her on the general ward and possibly given some advice about management while being pre-warned about a possible later admission.

b) Who should have responsibility for deciding on admission to PICU?

This would normally be the on-call General Paediatric Consultant or Senior Registrar, in discussion with PICU Specialist.

c) Would earlier admission to PICU have had an effect on her diagnosis and treatment?

Earlier admission may have helped and if the impending Cerebral Oedema had been suspected sooner as a result of this, treatment with severe fluid restriction, Mannitol, etc may have been possible.

xi) What was Dr Steen’s clinical role during Claire’s time on ICU?

Both from the case notes and from Dr Steen’s witness statement, it appears that her first involvement with and examination of Claire was at 4am on 23.10.96, after the collapse and diagnosis of Cerebral Oedema. It would appear from the discussion that her role in PICU was largely one of “damage limitation”. The actual management of intensive care would have fallen to Dr McKaigue. Dr Steen’s role would have been in making a diagnosis, discussing management decisions with the intensive care team and speaking to the parents.

xii) Was the information updated on the death certificate appropriate?

Clearly, with hindsight, Hyponatraemia should have been entered on the death certificate. However, given the information available this would have been a secondary not a primary diagnosis as there was good evidence that the acute illness was caused by the viral Encephalitis or Encephalopathy which can in itself cause Status Epilepticus and Cerebral Oedema without Hyponatraemia necessarily being implicated. Hyponatraemia can be a consequence rather than a cause of Cerebral Oedema, through inappropriate ADH secretion. This may have been the reason why it was not included in the death certificate. I do not believe that there was necessarily an intention to deceive when the death certificate was written.

xiii) a) Was the communication and counselling given to Claire’s parents after her death appropriate?

Given the information available, and the clinicians views at the time, I think it was appropriate. It would appear that the clinicians genuinely did not believe that hyponatraemia was the primary cause of her death, given that it could be explained by other causes.
b) Is Dr Steen correct in stating that Claire’s parents should have been spoken to around 2130h on 21.10.96 and who should have spoken to them?

Yes, undoubtedly. It appears that the parents were not appropriately informed of the severity of her condition at the time, even though medical staff were clearly concerned. It may be that medical staff assumed the nursing staff had done so and vice versa. They should have been spoken to by a Senior Doctor, i.e.: Registrar or Consultant, or possibly a senior member of the nursing staff, not necessarily just the nurse who was caring for her at the time. The parents should have been told that she was quite unwell and the diagnosis was still not entirely certain, and further investigations might have been necessary and that there was a possibility that if she did not improve, a transfer into Intensive Care might be necessary.

xiv) Would I have expected Claire’s death to have been reported to the Coroner?

Yes, undoubtedly. Although the threshold for reporting to the Coroner is lower now that it was in 1996, even then a sudden acute death in a child who had no life-threatening illness before, and where there was some diagnostic doubt, the Coroner should have been informed. This could have been explained to the parents as a routine procedure and should not have been unduly traumatic for them. The only circumstances in which a child’s death should not be reported to a Coroner is if there is already a firm diagnosis made that is known to be fatal. The General Paediatric Consultant, i.e.: Dr Steen should have made the report.

xv) Are the comments of Dr Dewi Evans regarding the CSF findings appropriate?

Although I am not an expert in this area, I agree that Dr Evans’ finding of an abnormal ratio of white cells in the CSF compared to the blood and is significant. My caveat in this is that the CSF was taken post-mortem, and I am not sure what changes in the CSF would be expected after death, but assuming that it is the same as in life then this is significant. From this finding there is good evidence there was some inflammatory activity in the meninges resulting in the increased number of white cells above expected in the CSF, and this would be evidence to support a diagnosis of Meningitis or Encephalitis contributing to Claire’s death. It is also of note the CSF protein was raised at 0.95 mg/L (upper limit of normal is 0.45). I am surprised that this finding was not mentioned by either the Pathologist who did the initial autopsy or by the expert Neuropathologist commenting on the case.

xvi) What is my opinion on the differing interpretations of the findings by the various experts?

I think there will always be uncertainty in this case. Regarding Claire’s history of presenting symptoms, these could be caused by a progressive viral Encephalitis or Encephalopathy, as suggested by the CSF findings. This was also suggested by the
preliminary post-mortem. However the expert neuropathology opinion suggested
there was no evidence at all of this illness in the brain histology. I am not
competent to arbitrate between two differing opinions between pathologists, but I
should point out that paediatric neuropathology is a particularly difficult area which
often results in differences of opinion, as for example in the case of “shaken baby
syndrome”. These differences of opinion can have major implications. The initial
brain post-mortem was done by a General Neuropathologist, whereas the expert
was a Specialist Paediatric Neuropathologist. On the facts of the case as presented,
it is entirely plausible that acute deterioration and the Cerebral Oedema with
coning were caused by hyponatraemia. 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.

It should be remembered that at the time, and for many years afterwards, many children
presenting with a broad range of acute illnesses, some similar to Claire’s, would have been
treated with exactly the same fluid regime as she received. The vast majority, even though the
serum sodium level may have been well below the normal range, would have made a
complete recovery. The very small number that develop serious or fatal consequences from
hyponatraemia are the exception rather than the rule. It seems to be entirely 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. In other words, if Claire had presented
to hospital with a different illness that did not involve any inflammation of the brain and had
received exactly the same fluids then she may not have become hyponatraemic and may not
have developed Cerebral Oedema.

**Historical note:**
Looking at these cases with hindsight, and looking at the literature starting with the paper by
Arieff published in the BMJ in 1992 (3), it seems remarkable that it took so long for the IV
fluid policy in children to change throughout the UK and elsewhere. It is not until 2007 that
the National Patient Safety Authority issued an alert advising all areas treating children to
stop using 0.18% saline. (2) As late as 2003 standard paediatric textbooks and pocket
handbooks in both the UK and the US were still recommending hypotonic saline (0.18% or
0.25%) as a possible choice of standard IV fluid management. (1,5,6) Regarding the choice of
IV fluid treatment in this case, it is important to remember that the clinicians concerned
would have treated very many children, as indeed I did myself, with this fluid regime with no
adverse effects whatsoever.

**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.

Signed: ..................................................
Dr Robert Scott-Jupp
20/5/11
References


Statement of Truth

I understand that my duty as an expert is to provide evidence for the benefit of the Inquiry and not for any individual party or parties, on the matters within my expertise. I believe that I have complied with that duty and confirm that I will continue to do so.

I confirm that I have made clear which facts and matters referred to in my report(s) are within my own knowledge and which are not. Those that are within my own knowledge I confirm to be true. The opinions I have expressed represent my true and complete professional opinions on the matters to which I refer, having studied all the relevant documents supplied to me.

I confirm that I have no conflict of interest of any kind, other than any disclosed in my report(s). I do not consider that any interest that I have disclosed affects my suitability as an expert witness on any issue on which I have given evidence. I undertake to advise the Inquiry if there is any change in circumstances that affects the above. I have no personal interest in supporting any particular point of view.

I understand that I may be called to give evidence.

Signed:  

Dr Robert Scott-Jupp

Date: 19/9/20