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- **Annex A : DETAILED COMMENTARY ON CLINICAL REPORTS**
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ANNEX A : DETAILED COMMENTARY ON

- **Dr QUINN'S REPORT JUNE 2000**
- **DR STEWART'S 2001 REPORT AND REPORT FROM DR JOHN JENKINS IN 2002**

Para	*Dr Quinn Report*	*My comment*
1	*I have reviewed the notes of this child as requested and will make a short summary and some comments on the possible sequence of events*	Does not set out the terms of his brief
2	*Lucy had been admitted on 12.4.00 at around 19.30 hours. Her G.P's letter stated that she had been pyrexic, not responding to Calpol, that she was drowsy and lethargic, that she was floppy and not drinking. He noted her temperature to be 38 C and wondered if she could possibly have a urinary tract infection. On admission the history revealed that the fever had been going for 36 hours and indeed that she had been vomiting for a similar period of time. She had been off her feeds to an extent of 5 days and that she was drowsy for about 12 hours. Her stools were reported to be normal. She had a temperature of 38 C on admission and was noted to be 9.14kgs. This would be around the 2nd centile for her age. Her capillary refill time was said to be > 2 seconds. Her abdomen was soft and bowel sounds were present. A diagnosis of viral illness was made.*	Does not mention the pulse rate, respiratory rate, colour, moist tongue,& that she was passing urine. All important for assessment of the degree of dehydration. Moist tongue was recorded in nursing record (Ref 027-017-056). In WS 279/1 Q 22(a) P23 Dr Quinn lists features he would use in assessing degree of dehydration including pulse and respiratory rates and mucous membrane dryness and states "…*There is no mention of mucosal state in the hospital notes…."*
3	*Her urines were checked. A blood count revealed a somewhat raised WCC at 15 with 13000 of these being neutrophils. Urea & electrolytes were essentially normal apart from a raised urea at 9.9.It is reported that the taking of oral fluids by the child should be encouraged. An intravenous line was inserted at 23.00 hours by a Consultant Paediatrician and solution 18 was started. It would appear that this continued at a rate of 100 ml /hour over the next 4 hours. The child also drank about 150 mls prior to this. At around 02.30 hours the child passed a very large runny bowel motion and was transferred into a*	The nursing record notes 2230 hours was the start time of the infusion.(Ref 027-017-058) In WS Q18 (b) P21 Dr Quinn states he used Dr O' Donohoe hand written entry on [027-010-022] which records approx. symbol 2300 (but in my opinion this could be a retrospective

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Para	*Dr Quinn Report*	*My comment*
	side room. At around 02.55hours on 13.4.00 the mother buzzed a nurse to say that the child was rigid. When the nurse saw the child she confirmed that it was rigid in the mother's arms and called a second nurse at around 0300hours. Lucy's colour was recorded as being satisfactory and her respirations were satisfactory. A junior doctor was bleeped at that stage and the child was turned on her side and given some oxygen. 2.5mgs of Diazepam was administered rectally. However it is recorded that within one minute of this a large bowel motion occurred and I suspect most of the Diazepam was expelled. On reviewing the child's electrolytes in and around that time it was decided that because the sodium was low that normal saline should be given. [note 1] At 03.20hours it was noted the respiratory effort was decreased. An airway was inserted and the child was bagged with bag and mask. She was ultimately intubated by an Anaesthetist and Flumazenil 100mcg was given. Her pupils were noted to be fixed and dilated. She was transferred to the intensive care in the Erne Hospital and ventilated in a high percent of oxygen. Mannitol 20% was given and intravenous Claforan. [Note2]	entry) Records a IV rate but does not comment in this written report in contrast to the view noted by Mr Fee in the meeting of 21 June where Dr Quinn does draw attention to the high volumes used .: when it was noted that he stated fluid replacement 4 hours at 100 mL provided was greater than normal *but not grossly excessive* and … he considered did not cause the brain problem. Dr Quinn in WS Q23(c)P24 states *"I calculated the fluid volumes which could have been used depending on the degree of dehydration of Lucy (please see my summary at appendix X). None of the figures which I calculated indicated that a rate of 100 mL per hour was appropriate."* Dr Quinn comments on accuracy of Mr Fee's record in Q9(d)P13. [Note 1] Dr Quinn concludes that normal saline was to be given on the basis of the returned second electrolyte result showing a low sodium. He does not remark here on the high

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Para	*Dr Quinn Report*	*My comment*
		volume of normal saline used although Mr Fee noted Dr Quinn raised this as a query with in the telephone discussion held with him 2 May 2000 and Dr Quinn reports later in Para 13 that he was uncertain how much normal saline had been given. WS Q16 P19 Dr Quinn has no recollection of the telephone discussion or its content. [Note2] no reference is made to the case record entry by Dr Malik-was this present in the records or inserted retrospectively - my impression is the latter but exactly when not clear and could have been on the same evening -this point should have been clarified because it is likely to have been present in the notes reviewed by Dr Quinn this gives the timing of fixed non-responsive pupils at 03: 20? (027-010-024). I comment on this further in my review of the fluids. But it is a weakness of this report that cross-referencing is not specific to the record.
4	*At 06.3Ohours she was transferred to the Royal Belfast Hospital for Sick Children's ICU and I understand that she subsequently died.*	

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Para	*Dr Quinn Report*	*My comment*
5	*I have subsequently been made aware that the Pathologist reported that the child had a significant pneumonia and cerebral oedema.*	It is not clear whether Dr Quinn had sight of the final autopsy report of 13 June 2000 which was not received by Dr O'Donohoe until 26/6/2000 (036a-051-114). Although the preliminary anatomical report was available from April 2000 it did not mention bronchopneumonia. Dr Kelly and Mr Fee had obtained a copy of the final report ####### get my note from Gov and Dr Quinn WSQ14 P18 considers it may have been given verbally at the 21/6/2000 meeting
6 & 7	*I will attempt to answer a few questions which obviously came up from reviewing the notes.* *Why was the child noted to be floppy in the first place?* *I suspect she may well have been quite ill on admission. The raised WCC with a predominance of neutrophils may go along with a bacterial infection and could have been due to the pneumonia which was found on P.M. However as stated before this is speculation.*	Does not take account of the high normal pulse rate nor the fact that Lucy was drinking. Lucy had no cough, only a slight increase in respiratory rate and no sign of respiratory distress. WSQ9 P12. He had reported [Ref 115-041-002] that "*nowhere in the notes is it stated that the child gave the appearance of being shocked which would have required another fluid regime*" And WS Q23(b)p24 *"My perception was that the doctors admitting the child assessed her as requiring*

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Para	*Dr Quinn Report*	*My comment*
		maintenance fluids and at that time (2000), maintenance fluid used extensively was solution 18 so it was appropriate that they used that type of fluid at the time. If a child appeared shocked, a common practice would have been use 0.9% NaCl, however, it did not appear to me, from the notes, that they assessed that Lucy was shocked from hypovolaemia (reduced blood volume due to fluid loss)." If Dr Quinn considered that Solution 18 was being used for maintenance only then the rate and volumes used were too high.
8	*Was the child dehydrated on admission?* *I think the urea measurement of 9.9 on admission does indicate a degree of dehydration. This level of urea would certainly not go with renal failure.*	The statement about the level of urea is a quibble but a raised urea is an indication of pre-renal failure even if this is mild. The blood urea is not the only measure used in assessing dehydration and Dr Quinn does not address his overall assessment of dehydration in this report although it is noted that by Mr Fee that he had graded this as moderate in his meeting on 21 June. He should have done so because he was asked to assess the fluid

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Para	*Dr Quinn Report*	*My comment*
		management and it is not possible to do this without a providing his estimate of the degree of dehydration in Lucy.
9	*Fluids.* *She was treated with Solution 18 which would be appropriate. On looking at the volume of fluids over the 7 hour period between admission and 3.00 a.m. when she had the possible seizure she got a total of 550mIs. This would include 150 mls oral and 400 mls i.v. as the intravenous drip was running at 100 mls per hour over a 4 hour period. Calculating the amounts over that period of time this would be about 80mIs/hr. I have calculated the rates of fluid requirements. If she was not dehydrated she would have required 45 mls /hour. If she was 5% dehydrated it would have worked out at 60 ml /hour and 10% dehydration works out at 80 mls/hour. I would therefore be surprised if those volumes of fluid could have produced gross cerebral oedema causing coning. I have however noted that there was no prescription written for the fluids indicating the volume per hour that should be given.*	Dr Quinn does not take into account the vomit which was recorded at 24:15 hours (00:15 h on 13/4/2000). This would represent a loss of the oral fluid taken. From later additional information given it appears that there was a further vomit at 1045 but this would not be known to Dr Quinn. (Nurse Swift's report 033-102-289,279,290 date stamped 8/5/2000). Dr Quinn does not here take account of the hyponatraemia (which he had noted in Para 3) as a potential contributor to cerebral oedema given the other biochemical markers of haemodilution in the change in urea, creatinine and total protein between the 2 samples. Dr Quinn does not here emphasise the high volume of No18 solution used when stating its use was *appropriate* but in WS : WS Q23(a)P24: *"The conclusion I came to*

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Para	*Dr Quinn Report*	*My comment*
		was that more IV fluids were administered had been intended" *WS Q23(d)P24:* *"Lucy's condition did not warrant IV administration of solution 18 at a rate of 100 mL per hour over a period of 4 hours, as this was in excess of the amount of fluid that I had estimated that Lucy would have required if she had been 10% dehydrated"* *WS Q23(e)P24:* *I was of the view that the administration of 100 mL per hour for 4 hours was excessive. I had discussed this at my meeting with both Dr Kelly and Mr fee and stated the volumes which might have been used, depending on the degree of dehydration. Nowhere did I state that 100 mL per hour over 4 hours was appropriate."*
10	*Was there evidence of renal compromise?* *I have noted that there was a urinary output and that there was no oedema of the face or peripheries noted. Ward testing of the urine showed some protein and ketones. However lab testing did not confirm proteinuria. The ketones would certainly be present in any child who is not eating well or indeed is vomiting.*	I agree that there was no evidence of renal damage & also the point about ketones.
11	*Did the child have a seizure or did she "cone" at*	I agree with this statement-it is not possible to know

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Para	*Dr Quinn Report*	*My comment*
	3.00am? *I feel it is very difficult to say what happened in and around this time. It is certainly possible that she had a seizure and may even have had a period of time when she was hypoxic before medical attention was drawn to the fact she was unwell. However I cannot say that this is the case. It may be that mother informed the ward staff immediately she noted the problem but again this is not clear to me from the notes provided.*	whether the observed rigid attack was a seizure or whether it originated from brainstem compression. Immediately following the seizure however she was able to breathe spontaneously until she started to struggle with breathing at around 03:15 to 03:20 when resuscitated with bagging. Her pulse remained present throughout.
12	*Apnoea.* *This could have occurred as the result of a seizure. It could have occurred as a. result of coning. I have looked at the possibility that it could have been due to medication with rectal Diazepam. I note the child was given 2.5mgs but it was stated that within one minute of administration of this she had a large bowel motion and I presume most of the Diazepam actually came out. . Certainly the recommended dose of Diazepam that can be given to a child who is seizing is 500mcgfkg. Therefore she could have been given up to 4.5mgs and certainly 2.5mgs given rectally to this age of child for a seizure would be appropriate. I am aware that some children have idiosyncratic reactions to Diazepam but normally this would be if they are given by the intravenous route and these events are very rare.*	I agree with Dr Quinn on these points.
13	*Was the resuscitation adequate?* *The notes state that the child had a good heart rate and colour throughout this event and that initially the child's respirations were adequate. Obviously when she became apnoeic in and around 03.2Ohours she required an airway insertion and bagging and she was ultimately then intubated by an Anaesthetist. During resuscitation it obviously became apparent that the*	Here Dr Quinn is probably referring to the respiratory resuscitation at 0315 but does not address the failure of detailed note keeping about the resuscitation itself. The anaesthetist should have made a contemporary

Para	Dr Quinn Report	My comment
	child's sodium had dropped to 127 and potassium down to 2.5 and a decision to use normal saline was made. I am not certain how much normal saline was run in at that time but if it was suspected that she was shocked then perhaps up to 20 mls/kg could have been given.	entry but in the event provided a note fairly shortly after the event dated 20/4/2000.(not seen by Dr Quinn) By 0320 it is possible that Lucy had received 75 ml of normal saline Dr Quinn concluded that a decision was made to use normal saline based on return of the blood electrolyte results but these were taken at least ½ hour after the saline started WS Q 25(d)&(f)P27. "*I was unable to identify any record of the amount of normal saline administered to Lucy in the Erne hospital notes."* Records show Lucy was given 250 ml of normal saline by 0430 in addition to the preceding fluid infusion of 100 ml per hour for 4 to 4 ½ hours. (Ref 027-025-076) A bolus of 20 ml per kg can be given stat in shock or repeated up to x2 but if x 2 there is a risk of pulmonary and/or cerebral oedema. In shock would be reasonable for a bolus to be given either 10 mls per kilo or 20 mls per kilo of fluid.

Para	*Dr Quinn Report*	*My comment*
		For Lucy 20 mls per kilo =183 ml and 10 ml /kg =91 ml . Lucy was given 250 ml. If 10 ml /kg used usually only for 2 hours followed by the maintenance and replacement regime over 24 hours.
14	*I hope these comments are helpful. I find it difficult to be totally certain as to what occurred to Lucy in and around 3.00a.m. or indeed what the ultimate cause of her cerebral oedema was. It is always difficult when simply working from medical and nursing records and also from not seeing the child to get an absolutely clear picture of what was happening. However I hope I have attempted to be as objective as possible with the information available to me.*	Dr Quinn here points out that he has not been able to conclude why Lucy developed cerebral oedema. I comment on this aspect in a separate commentary below.

Report (Ref 036a-025-058)	***Report content***	***My comments including reference to WS-298/1 of November 2012***
Dr Stewart 26/4/2001	*I have examined the case notes of LC, including the post mortem report and the report provided by Dr Murray Quinn.*	
Dr Stewart 26/4/2001	*Dr.Quinn's letter of 22-06-00 summaries the clinical course following Lucy's admission to the Erne Hospital on 12-04-00. I shall briefly outline the clinical events following her presentation to the Erne Hospital. Lucy was referred to the Erne Hospital by her general practitioner on 12.04.00. The history was of pyrexia, drowsiness, lethargy, floppiness and not drinking, and a diagnosis of a urinary tract infection was queried. The paediatric admission notes confirmed that Lucy had not been feeding well for the past 5 days with pyrexia and vomiting for the past 24 hours, and sleepiness for the past 12 hours. On examination, temperature was mildly elevated and she was noted to have prolonged capillary refill time. The plan was to encourage feeding, check urinalysis, take blood samples for full blood count, urea and electrolytes, glucose, C-reactive protein and blood culture and commence IV fluids after IV cannulation. She was admitted about 7.30 pm in the evening, and around 10.30 pm an IV line was inserted and she was commenced on intravenous fluids, 0.18% sodium chloride. From the nursing notes it appears that venous samples were taken at this stage (blood urea mildly elevated at 9.9 mmol/L and CO2 reduced at 16 mmol/L. Note [1]*	Note [1] The blood sample was not taken at the time she was put on intravenous fluids it was taken before at around 20:30 hours WS Q7(a)P6: Dr Stewart states *"the time on the laboratory form is 20:50 hours so it is likely that the samples were taken independently of cannula insertion."*
Dr Stewart	*At 15 minutes past midnight on 13.04.00 she had a large vomit, and at 2.30 am had a*	

Report (Ref 036a-025-058)	***Report content***	***My comments including reference to WS-298/1 of November 2012***
26/4/2001	*large soft bowel motion. At around 3 am the nurses were alerted by the mother, and she was reported to be rigid in her mother's arms. She was not cyanosed and pulse and respirations were recorded as satisfactory. A junior doctor was contacted, Lucy put on her side and oxygen therapy commenced. There was some smacking of her lips and twitching, and rectal diazepam 2.5 mg. was administered. This was followed by a large, watery, offensive stool. Blood pressure was elevated at this time at 144/113, but other observations were within normal limits. Around this time the intravenous fluids were changed to normal saline and run freely into the intravenous line. Around 3.20 am decreased respiratory effort was recorded, an airway inserted and bag mask ventilation commenced.*	
	Intubation was carried out around 4 am but the notes in the chart state that heart rate and oxygen saturation measurements were satisfactory from the time of the respiratory arrest until intubation was carried out. Around this time pupils were noted to be fixed and unresponsive. Note [2] She was transferred to the Intensive Care Unit in the Erne Hospital and subsequently to Paediatric Intensive Care Unit in the Royal Belfast Hospital for Sick Children. Brain stem tests were carried out in RBHSC, both were negative and she was extubated at 1300 on 13.04.00. A post-mortem examination was carried out which showed extensive bilateral bronchopneumonia, swollen brain with generalised oedema and early necrosis, with relatively little congestion with some distension of large.	Note [2] Dr Stewart clarifies below Dr Malik did not note the specific time in the clinical record. Dr Stewart did not have sight of Dr O'Donohoe's report to the July 2000 review (Ref 033-102-293) which suggested that they were fixed when he took over bagging from Dr Malik which was probably after Dr O'Donoghue obtained venous samples for the laboratory. .

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	and small intestine with gas and clear fluid. Rotavirus was detected in stool samples sent from the Erne Hospital on 13.4.00.	
	The following comments have been made following careful examination of the nursing and medical records from the Erne Hospital, including the post mortem report, and the medical report from Dr Murray Quinn. They are necessarily limited to the information contained in the notes. It is apparent that Lucy's clinical deterioration was unpredicted, rapid and extremely distressing for all concerned. I appreciate that I may have missed some facts, and that my comments are made some time after the events had occurred.	
Dr Stewart 26/4/2001	*<u>Points</u>* *Vomiting and fever are very common symptoms in young children. In most children, these symptoms are self-limiting and require only supportive measures such as attention to fluid balance, and antipyretic medication.*	
Dr Stewart 26/4/2001	*Lucy was probably quite ill on admission. She had been off her food for 5 days, with fever and vomiting for 36 hours and drowsiness for 12 hours.*	In response to the WS question 9 (b) P6 Dr Stewart explains her assessment of "quite ill" when she states *"history stated she was more lethargic than usual, poor appetite for 5 days, vomiting everything, pyrexic. Respiratory rate was increased, pulse rate at upper limit of normal and she had prolonged capillary refill time."*
Dr Stewart	*Clinical examination as documented, was*	Clinical opinions provided on

Report (Ref 036a-025-058)	***Report content***	***My comments including reference to WS-298/1 of November 2012***
26/4/2001	*essentially normal, but she did have prolonged capillary refill time indicating a degree of shock. Investigation showed increased white cell count, (15,OOOx10^9/L) mainly leucocytes, suggesting bacterial infection, urinalysis had protein ++ and ketones ++ and venous CO2 was reduced (16 mmol/l suggesting hyperventilation. Urea was elevated (9.9 mmol/L). These results indicate moderate-severe dehydration with a degree of pre-renal failure. The low CO2 suggests compensated metabolic acidosis (we do not have arterial or venous astrup results). The plan was to encourage feeding, and commence intravenous fluids after cannulation. Given the symptoms and signs, and the prolonged capillary refill time (>2 secs), it would be appropriate to give an immediate fluid bolus of up to 2Oml/kg (N Saline, or less commonly, colloid) and then reassess. It was several hours after admission before intravenous fluids were commenced. The difficulty in obtaining intravenous access in young children, and toddlers in particular, is well recognised. The notes do not make clear the possible. reasons for the delay in addressing the problem of restoration of circulatory blood volume.*	Lucy differ in respect of presence of "a degree of shock". Dr Sumner suggested that 10 mL per kilogram bolus was reasonable but not on the basis of shock. Dr Quinn concludes shock was not present. Dr O'Donoghue makes an important point which argues against the presence of shock which was relatively contemporary (probably 3rd of May 2000) in his report to the review when he stated: *.."the 100ml was approximately 10 mL/kilogram and to cover the possibility that the cannula might not last very long and the succeeding rate was relatively slow since I had seen her taking oral fluid well and presumed the rate of fluid needed was relatively small. I looked into the treatment room a few minutes later and Lucy was standing on the couch in front of her mother and looking better."* *(Ref 033-102-293)* The clinical medical and nursing notes do not suggest that Lucy had appearances of circulatory collapse or even imminent shock. But when Dr Stewart was asked to identify factors which indicated to her

Report (Ref 036a-025-058)	***Report content***	***My comments including reference to WS-298/1 of November 2012***
		that Lucy was in shock she provides the following answer in her witness statement question 11 (9a): *" the factors suggesting shock were increased respiratory rate (40/minute), heart rate (140/minute)-upper limit normal, prolonged capillary refill time (>2 seconds) reduced CO2, lethargy. "*
Dr Stewart 26/4/2001	*It is difficult to determine the nature of the episode at around 2.55 am although nursing records indicate some form of seizure activity. At the time respirations ceased, around 3.15am, pupils were fixed and dilated, and thereafter no spontaneous activity was recorded. (Repeat BM was elevated (13.4 mmol/L), BP elevated (144/113), but she was not bradycardic. Repeat U&E showed hyponatraemia (Na = 127 mmol/L), hypokalaemia (K 2.5 mmol/L), and urea had decreased to 4.9 mmol/L.*	Dr Stewart explains her uncertainty about the time of the pupil fixation in her witness statement answer 8(a) Page6 *" medical notes-3:30 AM-pupils dilated and unresponsive but time recorded in medical notes is after 5 AM-I cannot make out exact time".*
Dr Stewart 26/4/2001	*There are several possible explanations:* *(i) Lucy had a febrile seizure (she was pyrexic and at an age when febrile seizures are common) which continued from 2.55am, leading to hypoxia and cerebral oedema. However most children who have febrile seizures suffer no long term sequelae and do not develop cerebral oedema, especially as there was a relatively short-time gap between the first episode (2.55am) and the respiratory arrest (around 3.l5am).*	
Dr Stewart	*(ii) She had a seizure like episode due to underlying biochemical abnormality. Initial*	Note [3]

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26/4/2001	*sodium was 137 mmol/L, and potassium 4 mmol/L at 10.30 pm. At 3.00 am, and after administration of 0.18% NaCl, the repeat sodium was 127, and potassium 2.5. Note [3].* *Biochemical changes are often well tolerated and easily corrected with appropriate fluid replacement, although these results do show a change over a relatively short period of time. Note[4]*	Dr Stewart reports the blood sample showing the low-sodium was at 3 AM and that it was after administration of 0.18% rather than after administration of saline of unknown volume. It is possible to determine from the records that the normal saline started at around 03:00 hours from the charts and prescription. It is not clear however from the records or Dr Quinn's report at what time the blood sample was obtained nor that Dr O'Donoghue obtained it. Consequently Dr Stewart did not have enough information and made an assumption. Thus both 0.18% and normal saline at 250 ml/hour had been given before the blood sample. Note[4] A key conclusion for comment
Dr Stewart 26/4/2001	*(iii) The episode at 3.15 am was due to cerebral oedema and "coning". My impression from the notes is that Lucy never showed any signs of any recovery after 3.00 am and that this was a pre'-terminal event, followed by respiratory arrest around 3.20 am when pupils were noted to be fixed and dilated. BP was elevated at 144/ 113 but she was not bradycardic. Although intubation did not occur until 4.00 am,*	

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	nursing and medical records state that oxygenation was maintained and heart rate did not fall below 100.	
Dr Stewart 26/4/2001	*I agree with Dr Quinn that the administration of rectal diazepam is very unlikely to have been a contributing factor. The dose was correct, and in any case most of the drug was probably expelled shortly after being given by the per rectal route.*	
Dr Stewart 26/4/2001	*The fluid balance records between admission and the events at 3.00 am are incomplete. 0.18% saline was commenced at 10.30 pm, but the rate is not prescribed on the fluid balance sheet. My interpretation of the chart is that she received 100 mls/hr 0.18% saline until around 3.00 am when the adverse episode occurred. Note [5]*	*Note [5]* Note Dr Stewart is working on basis that 100 ml/hour started at 22:30
Dr Stewart 26/4/2001	*Once shock has been corrected with 20 mls/kg N saline (or colloid), APLS guidelines for a child with moderate/severe dehydration would be:* *Fluid deficit = 7.5% dehydration X weight (kg) X 10 i.e. - 750mls Maintenance fluids (24hrs) = 1000 mls i.e . a total of 1750 to be given over 24hrs - 70 - 80 mls/hr*	This is the calculation for a 10 kg child. Lucy was 9.14 kg on admission and thus slightly overestimates her fluid requirement which was 1599 ml not 1750 ml. (i.e. Lucy required on 7.5 % basis 65-67ml/hour. This is not a significant overestimate but Dr Stewart explains in answer to WS question 12 (a)P 9 "*actual weight was recorded at 9.14 kg. The reference to weight 10 kg was simply for ease of calculation. However, in assessing maintenance and allowing for dehydration, weight following rehydration is likely to be just under 10 kg."* This is a slightly unusual way

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		of calculating fluid but not a significant overestimate
Dr Stewart 26/4/2001	*The volume given, therefore, does not appear excessive. There is debate about the most appropriate fluid to use. Note [6]* *APLS guidelines; deficit should be replaced with normal saline and maintenance with 0.18% N saline.* *For convenience the 2 fluids are often combined and given initially as 0.45% NaCl in 5% dextrose, and the regimen altered on the basis of blood result.*	*Note [6]* This is a key conclusion for comment Lucy needed 67ml/hour. Dr Stewart calculated 70-80ml/hour. Lucy was given 100 ml per hour for 4 to 4 ½ hours.
Dr Stewart 26/4/2001	*After the respiratory arrest at 3.l5am, the fluids were changed to N saline. The clinical notes state that 5OO mls was given over the next hour. Note [7]*	*Note [7]* It is not clear on what basis Dr Stewart concluded that the timing of the switch to normal saline was 03:15 or that 500 mls was given over one hour. It is recorded as starting at 03:00 and that by 04:00 250 ml normal saline had been given. and this was corroborated by nursing report to Mr Fee noted in appendix to the July 2000 review. In responding to WS question 13 (a) and (b) P10, Dr Stewart states "*the medical notes written at 3:20 AM state that 500 mL normal saline was commenced at 3:20 AM. The nursing notes state that normal saline was started at 3:15 AM to run freely. The fluid balance chart states that*

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		500 mls normal saline was commenced at 3 AM but cannot work out rate. The medical notes state that 500 mls N saline was given over 60 minutes. The fluid balance chart is confusing as I cannot make out rate. (Reference 027-019-062).
Dr Stewart 26/4/2001	*A volume of 2Omg/kg would be indicated in a "shock" situation, although measurements recorded at this time do not suggest circulatory compromise, and her urea had fallen to normal levels.*	I agree that it was not evident that shock was present at 03:00 hours respiratory arrest to justify high volume IV bolus/ rate.
Dr Stewart 26/4/2001	*There was little warning of the rapid deterioration at around 3.OOam. There is nothing in the medical and nursing notes between admission and 3.00 am to indicate that medical and/or nursing staff were unduly concerned. Her temperature remained elevated (above 37.5oC until 22.30, but not markedly so. She was said to be floppy at 19.30 and asleep at 23.30. BM at 20.30 was 36 mmol/L. Although active resuscitation was commenced around 3.2Oam, there was never any response, and the fixed, dilated pupils almost certainly were an indication of severe brain pathology.*	Temperature had been 38.3 degrees at 2230 and 37.4 at 2330 hours (027-023-073)
Dr Stewart 26/4/2001	*<u>Summary</u>* *This little girl was admitted to the Erne Hospital in April 2000 and had a respiratory arrest 8 hours later, from which she never regained consciousness. Subsequent results indicate that she had gastroenteritis due to rotavirus (she may also have had bronchopneumonia). Initial investigations*	Dr Stewart is not clearly critical of the volume of fluid administered Dr Stewart acknowledges that a high volume of saline was given but does not report a view on whether this might have triggered the adverse

Report (Ref 036a-025-058)	***Report content***	***My comments including reference to WS-298/1 of November 2012***
	indicate that she was quite ill on admission, with a degree of circulatory failure. There was a delay in implementing fluid resuscitation and there are deficiencies in the prescription and recording of volumes of fluids administered. The subsequent events which occurred about 8 hours after admission were likely to have been ' preterminal and on the basis of cerebral oedema and coning.	event. When asked if she had given any consideration to whether the type of fluid (solution 18) administered at a rate of 100 mL/hour to Lucy could be considered excessive, WS Q 12(c) P9 Dr Stewart answers *"Summary clearly states "deficiencies in prescription and administration of fluids".*
Dr John Jenkins report of 7/3/2002	*013-011-038 & -039* *Dr Jenkins report. Asked to prepare a report on the death of Lucy by the directorate of legal services, Central services agency. The report was based on hospital notes. Rehearses the chronology of the illness (see note below) . He then writes:* *There is then a gap in the observation sheet with no apparent entry until an episode of sudden* *collapse which occurred around 3 . 00 am. It appears that mother called nursing staff as Lucy had passed diarrhoea and then become rigid Dr Malik was called and felt that this could be a febrile convulsion so administered Diazepam. He discussed the case with Dr 0'Donohoe who then came directly to the hospital arriving at 3.20 am. At around this time Lucy's condition further deteriorated as she stopped breathing and*	Concludes mildly dehydrated. The detail in the admission note does provide some further evidence of degree of dehydration because it provides a pulse rate/ respiratory rate. It is possible to make some assessment of the fluid given from the case records. Also it would be useful to determine whether Dr Jenkins had sight of the review carried out in 2000. Also whether he had seen the review carried out by

Report (Ref 036a-025-058)	***Report content***	***My comments including reference to WS-298/1 of November 2012***
	required respiratory support.. The on-call Anaesthetist was called at 3.40 am and Dr Auterson arrived shortly after 3.50 am and assisted with the resuscitation including intubation and transfer to the Intensive Care Unit prior to stabilisation and transfer to the Paediatric Intensive Care Unit in the Royal Belfast Hospital for Sick Children later on the same morning. The doctors involved seem clear that there was no episode of cardiac arrest or circulatory instability during this period but it was noted that the pupils became fixed and dilated and did not respond to ventilation or the administration of Mannitol. Subsequently tests in Belfast revealed evidence of brain stem death and post mortem examination was performed This showed bronchopneumonia and cerebral oedema with evidence of herniation of the brain. The Pathologist is unable to comment as to whether the bronchopneumonia had been present from admission to Erne Hospital or had occurred in association with the collapse and resuscitation. Further specimens have shown rotavirus infection suggesting that the initial admission was likely to be due to rotavirus gastroenteritis. Urine cultures showed no significant growth .	Dr Moira Stewart .
Dr John Jenkins report of 7/3/2002	*Comment* *This child's admission to Erne Hospital was very typical of gastroenteritis in this age group. This is often associated with high temperature and vomiting with or without diarrhoea and young children can become very unwell. The standard treatment is to administer fluids either orally or (if there is significant dehydration or vomiting) by the*	Dr Jenkins points out the inappropriate fluid choice . His view that the 0.18% solution was used for standard therapy for intravenous use when

Report (Ref 036a-025-058)	***Report content***	***My comments including reference to WS-298/1 of November 2012***
	intravenous route. The solution used is one which is commonly used in Paediatric practice to provide maintenance fluids in these circumstances as it replaces small amounts of electrolytes but also gives Dextrose which is required by young children who are unable to take calories orally during the acute phase of the illness. Initial physical findings were suggestive of poor peripheral circulation with delayed capillary refill time >2 seconds. The GP noted that the mucosae were moist but there is little specific detail in the admission note regarding evidence of dehydration. However, the urea was 9.9 which is slightly elevated suggesting a mild degree of dehydration but with normal electrolytes at that time. This would again be very typical of the condition and would not normally indicate anything other than appropriate fluid replacement with careful monitoring and nursing observation. However, in this situation the intravenous fluids for replacement should contain a higher content of sodium (eg "normal saline" - 0.9% NaCl - sodium chloride). *In these circumstances it is always very difficult to understand an episode of sudden collapse. Sudden onset of convulsions is most commonly due to high temperature in young children and this was considered However, the features were not typical and the temperature had in fact improved since admission It is unclear as to what alternative diagnoses were considered at this time but the blood test for electrolytes was appropriately repeated immediately. This showed a significant fall in sodium from 137 to 127 and in potassium from 4.1 to 2.5,*	treating rehydration is correct for the time when the blood sodium has been found to be normal (as it was before the IV infusion started) but he highlights growing concerns about this practice : "Over recent years concerns have begun to be expressed regarding the use of 0.18% saline in Dextrose as a standard solution for intravenous use in young children and a number of cases of symptomatic hyponatraemia have been identified, some resulting in death or cerebral damage. It has been suggested that a more appropriate solution would contain a higher level of sodium" He alerts the Trust to a potential linkage between the fluid regime and Lucy's death.

Report (Ref 036a-025-058)	*Report content*	*My comments including reference to WS-298/1 of November 2012*
	together with an increase in glucose from 4.5 to 10.9. These changes do raise the question as to the fluid management in the period from insertion of the IV line at 2300 to the collapse at around 3.00 am. Unfortunately there appears to have been confusion between the staff involved as to the fluid regime ordered by the Consultant. In addition it is difficult to interpret the records made by nurses on the fluid balance chart and no totals have been calculated for this period It will be most important to determine from the staff involved exactly how much of each type of fluid was given at each stage throughout this time period, and following the change of fluids to normal saline through until the child arrived in the Paediatric Intensive Care Unit in Belfast. *Other aspects of this tragic case demonstrate a rapid and effective response by the medical staff concerned In particular both the Consultant Paediatrician and Consultant Anaesthetist appear to have been available within a very short time period of being called and to have done their best in the difficult circumstances involved in caring for a child of this age in an adult intensive care setting for stabilisation and transfer in the absence of a Paediatric transfer service in Northern Ireland.* *Over recent years concerns have begun to be expressed regarding the use of 0.18% saline in Dextrose as a standard solution for intravenous use in young children and a number of cases of symptomatic hyponatraemia have been identified, some resulting in death or cerebral damage. It has been suggested that a more appropriate*	

Report (Ref 036a-025-058)	*Report content*	*My comments including reference to WS-298/1 of November 2012*
	solution would contain a higher level of sodium and this has recently been the subject of discussions involving the Department of Health, Social Services and Public Safety and production of guidelines. However, it must be emphasised that this is a very recent development and that many Paediatric Units are continuing to use the solution which was initially given in this case. Although the sodium level of 127 is not in itself usually associated with severe problems, it is likely to be the rate at which the sodium falls rather than the absolute level which can cause problems in this setting. *While no definite conclusions can be drawn regarding the cause of this child's deterioration and subsequent death there is certainly a suggestion that this was associated with a rapid fall in sodium associated with intravenous fluid administration and causing hyponatraemia and cerebral oedema. It would have been advantageous if there had been available documentation regarding the fluid type and rate prescribed, together with clear records as to the exact volumes of each fluid which were in fact received by the child throughout the time period concerned. Unfortunately there appears to have been confusion between the staff involved with inadequate documentation and record-keeping. In this respect, unless this can be clarified in a satisfactory manner, it is my opinion and management fell below the standard which would be accepted by responsible body of medical opinion is reasonable practice at the relevant time.*	

ANNEX B GASTROENTERITIS EPIDEMIOLOGY

Diarrhoea and Vomiting

As a presentation on admission, diarrhoea and vomiting was the 4th commonest presentation in 5 Yorks hospitals studied in late 1990s:

In patients

- Stewart,Werneke, MacFaul,Taylor,Smith ADC 1998;79:219

Hospital (842)[1]	
Breathing difficulty	24.5%
Fit	16.1%
Fever illness	15.2
D&V	9%

Gastro enteritis was the commonest discharge diagnosis in paediatric admissions in England in 2002. ***(From MacFaul R, unpublished review 2004)***

Hospital Episode Statistics (HES) were examined for the years 1991/2 to 2001/2 including hospital admissions for all causes for children. Admissions coded as infectious diarrhoea and non infectious diarrhoea were reviewed and the total admissions resulting from both categories were used to determine the proportion of all acute admissions of children which were caused by gastroenteritis.

Trends in hospitalisation for children and in the number and proportion for gastroenteritis show a rise in hospitalisation over 11 years up to 2002 and my analysis shows that gastroenteritis is the commonest single cause of hospital admission for children in England. In contrast over the same period asthma and wheeze admissions have fallen[1].

The data shows that gastroenteritis is the commonest single cause of hospital admission of children in England and hospitalisation and has increased over the 11 years up to 2002. Hospitalisation rates are high in UK.

Table 1 In-patient episodes for Gastro-enteritis as proportion of all child in-patient episodes using total child admissions under all specialties from 1998 to 2002. Newborn episodes excluded.

Episodes by code	***1998-1999***	***1999-2000***	***2000-2001***	***2001-2002***
All episodes age 0-14 years	1703763	1682094	1625649	1655961
Newborn infant codes				
All P code episodes	189095	186215	178526	175395
Z37 and Z38 codes	387267	372249	359930	378519
Episodes other than newborn	1127401	1123630	1087193	1102047
Gastroenteritis codes				
A codes	24716	24760	23422	26063
K52 codes	19692	19802	18071	18752
All gastroenteritis codes (A and K)	44408	44562	41493	44815
K Code episodes as proportion of gastroenteritis codes	44%	44%	44%	42%
Gastroenteritis as proportion all child episodes (including surgical)	4%	4%	4%	4%
(other than newborn)				

Table 2 Next most common child in-patient episodes (other than gastroenteritis)

Episodes by code	*Year 1999*	*1998-1999-2000*	*2000-2001*	*2001-2002*
J06.9 Acute upper respiratory infections multiple and unsp site	43,974	39967	39,028	39,411
R69.X Unknown and unspecified causes of morbidity Unknown and	34,146	40985	39,227	35,424
J35.0 Chronic diseases of tonsils and adenoids Chronic tonsil	29,295	26457	20,280	20,189
H65.3 Nonsuppurative otitis media Chronic mucoid otitis media	28,044	23632	23,099	21,991
B34.9 Viral infection of unspecified site Viral infection	26,872	25891	33,920	36,688
J45.9 Asthma unspecified	22,400	20118	17,940	18,636

Admission rate GE age < 5yr

Country	Year	Rate per thousand
E&W	1996	11.7
Eng	2001	10.7
Aust	1996	15
Netherlands	97/98	7.1
Spain	1994	10
US insured	1997	3.6
US not insured		8.3
Finland	1998	14.4
Ireland	97/98	23.7

SEVERITY OF ILLNESS AND DEHYDRATION VARIES BETWEEN HOSPITALS AS ALSO DOES RECOURSE TO AND USE OF IV THERAPY.

These data formed part of my own studies on gastroenteritis admissions and formed background to presentations and a submitted but unpublished paper MacFaul R and Thompson 2007.

UK studies-Leeds !

Conway et al 1990
Conway et al 1994

	1986/7	1991
N =	1148	300
LOS	7 days	5 days
IV rate	5%	9%
Moderate dehyd	1%	6.7%
Severe dehyd	0.1% (I child)	2.3 %

IV rates UK audit Yorks 2002

	PGI	***PGH***	***DGH-A***	***DGH-B***
N	31	87	38	42
Dehydration				
-Moderate	1	8	1	1
-Severe	2	2	0	1
IV	4	18	4	11
IV -mild/mode	0	6	0	9
Percent IV	*13%*	*21%*	*11%*	*26%*
Percent severe	*6%*	*2%*	*0%*	*2%*

DEATH FROM GASTROENTERITIS (MacFaul 2004)

Childhood death from Gastroenteritis is now very rare in a developed country.

In summary the number of deaths recorded in gastroenteritis in 2002 in England & Wales was 4: an improvement on previous years, for example in 1990-1993 the annual number was 17.

In 1999 the number was in the order of 46 and in year 2000 was 43 (and possibly including newborns) and only 4 over age 12 months (falling to a total of 12 in 2001). See Tables below from ONS data sets (MacFaul analysis). In contrast the number of children admitted to hospital for treatment of gastroenteritis in 2002 was 44,815 (MacFaul R 2004 review) -the commonest single diagnosis for which children are admitted acutely to hospital. Although recommendations have been made to use oral rather than intravenous fluid therapy when possible e.g. *Armon K, Stephenson T, MacFaul R, , Eccleston P, Werneke U, An evidence and consensus based guideline for acute diarrhoea management. Arch Dis Child 2001; 85:132-142.* , IV therapy was (and is) still a frequent therapy used and in 2002 the proportion of children admitted to hospital who were given intravenous therapy varied between the 4 hospitals included in an audit (Macfaul 2004) ranged from 11% to 26%. Thus intravenous therapy for gastroenteritis without complication was very frequent in practice.

Table 7. Numbers of annual deaths in children from gastroenteritis

Year	Annual deaths from gastroenteritis in England &Wales
1978 [1 and 2]	164
1980 [1 and 2]	59
1986 [1 and 2]	27
1990-1993 [3]	17
2002 [4]	4

[1] Conway SP, Phillips RR, Panday S. Admission to hospital with gastroenteritis. *Arch Dis Child* 1990;**65**:579–84.

[2] Conway SP Newport MJ. Are all hospital admissions for gastroenteritis necessary.J Infect 1994; 29: 5-8

[3] Crowley DS, Ryan MJ, Wall PG. Gastroenteritis in children under 5 years of age in England and Wales. Communicable Disease Report 1997; 7: R82-R86

[4] ONS data : ***possibly undercounted :see recent analysis below from***

MACFAUL ONS DATA TABLES ANALYSIS

Ill-defined intestinal infections ICD-9 Codes used

0090 Infectious colitis, enteritis and gastroenteritis

0091 Colitis, enteritis and gastroenteritis of presumed infectious origin

0092 Infectious diarrhoea

0093 Diarrhoea, of presumed infectious origin

Age	***Code***	***Year 1997 Deaths***	***Year 1998 Deaths***	***Year 1999 Deaths***	***2000 Deaths***
Age<1 year*	0090-9903	31	40	38	39
Age<1 year*	558			6	0
Child Age > 12m ,<14yr	0090-9903	1	1	0	0
1-4yrs	558	n/a	n/a	2	4

* may contain newborns

ICD -10 CODES

Of all cases a small proportion result from bacterial infection but the remainder result from a range of viral causations including adenovirues, calcinivirus, enteroviruses, norwalk virus etc. In practice most paediatric clinicians describe a child discharged from hospital with acute diarrhoea as gastroenteritis and by that they mean or imply an infectious cause. However, unless the discharging clinician qualifies the description of the illness resulting in admission in some way to indicate an infectious cause, the episode will be coded on the Hospital Episode Statistics as a non-infectious diarrhoea. This is despite the fact that the majority of these cases coded as ICD-9 code 558 (K52.9 in ICD-10) are of an infectious aetiology. The episode will be coded as an infectious cause if terms such as “viral”, “infectious”, “rotavirus” etc are used and then the admission will be coded using codes from the ICD-9 group of codes 001-9 which are equivalent to the ICD-10 A00.1 to A09.X codes.

Age	***Code ICD-10***	***Year 2001 Deaths***	***Year 2002 Deaths***

Age<1 year*	A09	1	0
Age<1 year*	K529	4	2
Child Age > 12m -4yr	A09	0	0
Child Age > 12m -4yr	K529	7	2

ANNEX C: GUIDANCE ON IV FLUID TREATMENT IN GASTROENTERITIS /DEHYDRATION

Fluid loss in gastroenteritis results from vomiting, diarrhoea, or loss into the bowel lumen of fluid faecal matter prior to its being passed as diarrhoea.

The fluid loss leads to dehydration, reduced circulating blood volume and reduced urine output and, if it advances, with significant reduction in circulating blood volume leading to tachycardia which is increasing, reduced renal perfusion and pre-renal renal failure and in severe hypovolaemia to reduction in blood pressure and impaired perfusion of the brain-the latter leading to drowsiness or confusion.

There is loss of electrolyte-sodium and potassium particularly and, with poor perfusion a degree of metabolic acidosis.

The clinical assessment of dehydration is subjective. The clinical measures used are

(a) from the history to determine the frequency of vomiting, the reduction in fluid intake and the volume and frequency of stools as a measure of fluid loss and the amount of urine passed to determine in part the degree of reduced circulating blood volume.

(b) On examination the clinical assessment includes measurement of the pulse rate, assessment of the skin turgor -increased skin laxity/ reduced elasticity-dryness of the oral mucosa, sunken eyes or in infants with an open anterior fontanelle or soft spot -a sunken fontanelle. In advance dehydration with acidosis, the respiratory rate increases, there is pallor and following an increased heart rate , reduced peripheral perfusion may be evident with prolonged capillary return beyond two seconds. In further advanced dehydration with imminent or established shock there may be drowsiness and floppiness/poor body tone or posture and reduction in blood pressure.

"The severity of dehydration is most accurately assessed in terms of weight loss as a percentage of total body weight (prior to the dehydrating episode). This is the "gold standard" against which other "tests" are measured. …….. T*he sensitivity and specificity of all clinical signs were low in a number of studies. (Armon K, Stephenson T, MacFaul R, Eccleston P, Werneke U, An evidence and consensus based guideline for acute diarrhoea management. Arch Dis Child 2001; 85:132-142)."*

Mild to moderate dehydration represents around 3-5%. Where there is circulatory collapse-shock-the weight loss is around 9% to 10%.

Volume used for IV therapy

There is fairly more consistent advice available in Texts although as Forfar & Arneil acknowledge regimes used vary to a slight extent. It is conventional to assess fluid requirements per day as follows

Maintenance :

- First 10 kg-100 mls per kilogram body weight
- Second 10 kg 50 mls per kilogram body weight
- Subsequent kilogram 20 mls kilogram body weight

To maintenance is added the estimated deficit as % of body weight

Deficit

- Mild dehydration less than 5% (equivalent to 50 ml/kg/body weight per 24 hours)
- Moderate dehydration 7.5 % (equivalent to 75 ml/kg/body weight per 24 hours)
- Imminent or existing shock severe dehydration 9 to 10%. (equivalent to 100 ml/kg/body weight per 24 hours)

In practice :

In the moderately or severely dehydrated patient between 10-20 mls per kilogram of 0.9% saline is given as a bolus. Options include 10-20ml/kg if there is concern about evolving shock or circulatory failure either as bolus – when it might need to be repeated given as a push over 15-20 minutes or over the first one or two hours, or in less severe degrees of dehydration as 10-20ml/kg per hours over 4 hours after set up of infusion. In imminent or established shock this can be given as rapidly as possible for example in a bolus of between 10 or 20 mls per kilogram of body weight and this can be repeated once or more times (although more than x2 may lead to need for intubation and ventilatory support) in order to ensure that the circulation returns before starting a continuous infusion.

ADVANCED PAEDIATRIC LIFE SUPPORT MANUALS (BMJ Books)

APLS Edition	***Advised management of Fluid deficits***
Second edition. 1997-1998 revisions current in 2000	"If we were following the sums exactly we should put out two drips-one of 750 mls with a sodium hundred and 40 mmol per litre and another of 1000 mls with 30 mmol sodium. As fluid balance is not often an exact science (ongoing losses, clinical estimations etc) it is usually more convenient to pick one intravenous fluid with a sodium concentration somewhere between the two and give the total volume using this. The fluid which fits the specification best in this case is 0.45% saline which has 75 mmol per litre. This can be changed to fluid containing more or less sodium depending on subsequent serum sodium results. To make it isotonic 0.45% saline is usually made up of 2.5% dextrose. Beware of using IV fluids with no dextrose and small children as they may become hypoglycaemic. In patients with low or normal sodium lost fluid can be

	replaced over a 24-hour is. In hypernatraemic patient must be replaced over at least 48 hours and sometimes longer….” Note this was calculated on the basis of the 10 kg example with 7.5% dehydration.
Third edition 2001	The text is the same as for the second edition.

These APLS editions emphasise that where there has been fluid loss it is likely to be with the sodium content almost equivalent to normal saline and therefore there will be a sodium deficit to be replaced. The maintenance solution number 18 does not contain sufficient sodium to replace deficit.

THE FOLLOWING PAGES ARE FROM THE APLS MANUAL SECOND EDITION

Intravenous fluids are available in a variety of electrolyte compositions. In particular, there are a number of different strengths of dextrose and saline (often as a mixture in the same bag) – the concentration of sodium being expressed in mmol/l on the side of the infusion bag, as well as a percentage. Always check the sodium concentration in mmol/l is actually what you require and take great care to specify the concentration of both the dextrose and the saline (if a dextrose/saline solution is being used) when writing the prescription to avoid ambiguity. Tables B.4 and B.5 show the composition of commonly available fluids.

Table B.4. Commonly available crystalloid fluids

Fluid	Na^+ (mmol/l)	K^+ (mmol/l)	Cl^- (mmol/l)	Energy (kcal/l)	Other
Isotonic crystalloid fluids					
Saline 0·9%	150	0	150	0	0
Saline 0·45%, dextrose 2·5%	75	0	75	100	0
Saline 0·18%, dextrose 4%	30	0	30	160	0
Dextrose 5%	0	0	0	200	0
Saline 0·18%, dextrose 4%, 10 mmol KCl/500 ml	30	20	50	160	0
Hartmann's solution	131	5	111	0	Lactate
Hypertonic crystalloid solutions					
Saline 0·45%, dextrose 5%	75	0	75	200	0
Dextrose 10%	0	0	0	400	0
Saline 0·18%, dextrose 10%	30	0	30	400	0
Dextrose 20%	0	0	0	800	0

Table B.5. Commonly available colloid fluids

Colloid solutions	Na^+ (mmol/l)	K^+ (mmol/l)	Ca^{2+} (mmol/l)	Duration of actions (hours)	Comments
Albumin 4·5%	150	1	0	6	Protein buffers H^+
Gelofusine	154	<1	<1	3	Gelatine
Haemaccel	145	5	12·5	3	Gelatine
Pentastarch	154	0	0	7	Hydroxyethyl starch

DEHYDRATION

Dehydration is the result of abnormal fluid losses from the body which are greater than the amount for which the kidneys can compensate. The natural mechanisms for compensation have the primary aim of maintaining circulating volume and blood pressure at all cost. Thus the majority of patients with dehydration maintain their central circulation satisfactorily. Loss of central circulatory homeostasis constitutes *hypovolaemic shock* and is dealt with in Chapter 10.

The major causes of dehydration in children are gastrointestinal disorders and diabetic ketoacidosis. Some renal disorders (polyuric tubulopathy with urinary tract infection, polyuric chronic renal failure and diabetes insipidus) might also present in this way. Depending on the source of fluid losses and the quantities of electrolytes lost (Table B.3), dehydration can be divided into three types:

1. Isotonic dehydration – sodium and water lost in proportion to each other.

2. Hyponatraemic dehydration – more sodium lost than water proportionately.
3. Hypernatraemic dehydration – more water lost than sodium proportionately.

In all three types there is usually a total body deficit of *salt and water*. Between the three types the relative amounts of salt and water loss vary. Table B.6 shows the symptoms and signs of dehydration and gives a guide towards the assessment of the degree of dehydration. On the whole, the more severe the dehydration the more likely that hypovolaemia will be a problem; most patients with more than 10% dehydration are hypovolaemic at presentation. However, speed of fluid loss is important. Slow, prolonged losses can give rise to massive dehydration without hypovolaemia, similarly acute, severe loss can present as hypovolaemia without apparent significant dehydration. The latter is not infrequently the case in acute gastroenteritis in infants where acute fluid loss into the bowel causes hypovolaemia and the patient can present even before any diarrhoea has occurred.

Table B.6. Symptoms and signs of dehydration

Sign/symptoms	Mild <5%	Moderate 5–10%	Severe >10%	Notes/caveats
Decreased urine output	+	+	+	Beware watery diarrhoea making nappies appear "wet"
Dry mouth	+/−	+	+	Mouth breathers are always dry
Decreased skin turgor	−	+/−	+	Beware the thin, use several sites
Sunken anterior fontanelle	−	+	+	Crying increases pressure
Sunken eyes	−	+	+	
Decreased eyeball turgor	−	+/−	+	Difficult to assess in young
Tachypnoea	−	+/−	+	Metabolic acidosis and pyrexia worsen this
Tachycardia	−	+/−	+	Hypovolaemia, pyrexia and irritability cause this
Drowsiness/irritability	−	+/−	+	

Management of dehydration

Mild dehydration (<5%) can usually be managed with oral rehydration if vomiting is not a major problem. Oral rehydration fluids are better absorbed if they contain a small amount of sodium and glucose in addition to water. Commercial preparations contain, for example, 35–50 mmol of sodium per litre when made up as instructed.

Moderate and severe dehydration will require more accurate replacement of fluid loss and although oral rehydration may sometimes be possible, intravenous therapy may be needed.

For fluid balance purposes, as the body is mostly water, a weight loss of 1 kg equals a fluid loss of 1 litre, as one millilitre of water weighs one gramme. Thus fluid loss or gain can be measured easily by weighing the patient. The child's fluid deficit can be worked out from the child's weight and a clinical assessment of the percentage dehydration. For example, a 10 kg child is 7·5% dehydrated. How much fluid will the child need for rehydration and what sodium concentration will be required?

The child will need maintenance + replacement of deficit. Calculate them separately and add them up.

Step 1

What is the fluid deficit?

7·5% of 10 kg = 0·75 kg = 750 g
750 g is the weight of 750 ml fluid

A convenient formula to remember is:

$$\text{Percentage dehydration} \times \text{Weight in kg} \times 10 = \text{Fluid deficit (ml)}$$

Thus the fluid deficit is 750 ml. The fluid deficit is essentially made up from (roughly) 0·9% saline (which has 150 mmol/l) since it is mainly extracellular fluid that has been lost which has a sodium concentration of approximately 140 mmol/l.

Step 2

The child also needs maintenance fluids. These can be worked out in the normal way. A 10 kg child will need 10×100 ml/day for normal maintenance (Table B.1) = 1000 ml. The sodium required for maintenance (Table B.3) will be approximately 3 mmol/kg $\times$ 10 kg = 30 mmol/day.

In total, then, the child needs 1000 ml maintenance plus 750 ml replacement of losses, totalling 1750 ml, for adequate rehydration.

If we were following the sums exactly we should put up two drips – one of 750 ml with sodium of 140 mmol/l and another of 1000 ml with 30 mmol of sodium. As fluid balance is not often an exact science (ongoing losses, clinical estimations etc.), it is usually more convenient to pick one intravenous fluid with a sodium concentration somewhere between the two and give the total volume using this. The fluid which fits this specification best in this case is 0·45% saline, which has 75 mmol/l. This can be changed to fluid containing more or less sodium depending on subsequent serum sodium results. To make it isotonic 0·45% saline is usually made up with 2·5% dextrose. Beware of using IV fluids with no dextrose in small children as they may become hypoglycaemic.

In patients with a low or normal sodium lost fluid can be replaced over 24 hours. In hypernatraemic patients it must be replaced over at least 48 hours and sometimes longer depending on the severity – the higher the sodium the slower the rehydration must be. If the sodium and water are corrected too rapidly in the extracellular space, water will pour into cells, and if this happens in the brain, cerebral oedema and even death may occur. Aim to bring down the serum Na in a hypernatraemic patient by no more than 5 mmol per day, for example, in an infant who presents with a Na of 170 mmol/l, the Na should be no less than 165 mmol/l by the next day. In these patients, the electrolytes should be checked 4-hourly, at least initially.

The very sick child

In the very sick child it may be uncertain whether normal homeostatic mechanisms will work. The patient may be progressing into renal failure and be oliguric or inappropriately polyuric. In such cases the best management is to:

- Catheterise the patient.
- Calculate and replace deficit, over 24 hours, with normal saline.
- Calculate insensible losses and replace with 0·18% saline, 4% dextrose.
- Measure urine output and replace ml for ml on an hourly basis with 0·18% or 0·45% saline with dextrose according to the plasma electrolytes.

This technique is applicable to all patients with all conditions in all states of hydration. Moreover, subsequent measurement of urinary electrolytes can allow exact tailoring of IV fluids to maintain normal serum electrolytes.

DIABETIC KETOACIDOSIS (DKA)

DKA is a special case in which a relative or absolute lack of insulin leads to an inability to metabolise glucose. This leads to hyperglycaemia and an osmotic diuresis.

THE PAEDIATRIC VADE MECUM (BIRMINGHAM CHILDREN'S HOSPITAL) 14TH EDITION

In guidance available around 1993 or earlier more caution was exercised in the rate of replacement giving **advice to correct deficit with two thirds of the calculated deficit volume within the first 24 hours but later guidance advises correction within 24 hours unless there is hypernatraemia. Solutions advised were half normal saline in infancy and normal saline in older children.**

Advises use of normal saline for treatment of dehydration.

THE FOLLOWING ARE RELEVANT PAGES FROM FORFAR & ARNEIL FIFTH EDITION 1998 (REPLACED BY SIXTH IN 2003

Table 10.12 Sodium depletion

Sodium deficiency (mmol)		Signs and symptoms
Adult (70 kg)	Infant (6 kg)	
300	30	Just detectable. Cold, gray skin
300–600	30–60	Tachycardia, blood pressure low; dry mucous membranes, sunken eyeballs and fontanel, loss of skin turgor
600–1000	60–100	Severe shock, systolic blood pressure very low, uremia. Plasma sodium low. Plasma osmolality tends to be low

conserving plasma volume is of greater importance and hyponatremia occurs.

Degrees of sodium depletion and associated symptoms are indicated in Table 10.12.

Thirst tends to be absent since this symptom is primarily due to an increase in intracellular osmolality. Packed cell volume, hemoglobin and plasma proteins are often increased; but again may be masked by pre-illness deviations from normality.

MIXED WATER AND SODIUM DEPLETIONS

A loss of isotonic sodium-containing fluid results in the ECF bearing the reduction in volume, whereas a loss of (hypotonic) water is shared by both ECF and ICF. Since many of the clinical manifestations of dehydration are reflections of a reduction primarily in ECF volume, sodium deficiency and its attendant fluid loss produce more marked clinical signs than water deficiency of comparable magnitude.

One of the commonest forms of sodium and water depletion is that due to the loss of intestinal secretion (Table 10.13). This may be occasioned by intestinal obstruction, fistulae of the small intestine, enterocolitis, or an ileostomy. The composition of the fluid lost will vary, but with the exception of saliva all normal alimentary secretions are isotonic with plasma. Diarrheal fluid tends to be hypotonic and, as such, is liable to cause hypernatremia in infants where the fluid intake is relatively low, and the kidney is unable to produce a very concentrated urine. Conversely older children and adults suffering from diarrhea often present with hyponatremia since they ingest large quantities of hypotonic fluids and their kidneys can produce concentrated urine.

In an infant weighing 3 or 4 kg, the volume of alimentary secretions per 24 h is approximately 400 ml.

POTASSIUM DEPLETION AND INTOXICATION

After intestinal absorption, potassium is borne via the plasma and interstitial fluid compartments to its predominantly intracellular location. When cellular function is normal the content of potassium per gram of noncollagen nitrogen (K/N) can be shown to be fairly constant. For specimens of skeletal muscle the value is approximately 2.8 mmol potassium per gram of noncollagen nitrogen. Since about 70% of the body content of potassium resides in muscle tissue, it is obvious that any muscle wasting will result in loss both of potassium and water from the body. Naturally the exchangeable potassium (K_e) will diminish as muscle wasting progresses, but the K/N ratio often remains within normal limits. This state does not normally qualify for inclusion in the term 'potassium deficiency' (depletion) which is reserved rather for situations where the K/N ratio is reduced. It is doubtful whether a significantly increased K/N ratio ever exists for other than brief periods.

Extreme variations in the plasma concentration of potassium (1.0–12.0 mmol/l) have been observed in the living state, ranging both above and below the normal limits of 3.8–5.0 mmol/l in adults and 3.8–5.5 mmol/l in infants. Less than 1% of the potassium in the body is in the plasma and about 2% is in the ECF so that rapid changes in plasma concentration may occur following transfer between the extracellular and intracellular compartments. In many situations it is not valid to assume that hypokalemia indicates potassium deficiency, and hyperkalemia may be found with normal or reduced K/N ratios. In general, however, where the clinical condition justifies assuming potassium deficiency to be likely, hypokalemia can be used as an index for regulating potassium therapy.

Potassium deficiency occurs in a variety of situations but most often involves excessive loss via the intestinal and/or renal routes. Such conditions as vomiting, fistulae, diarrhea, Cushing's syndrome, primary hyperaldosteronism and the use of glucocorticoids or diuretics are particularly liable to induce potassium depletion with or without concomitant hypokalemia. An acidosis tends to cause renal excretion of hydrogen ions in exchange for sodium, whilst conserving potassium. Associated with this is the transfer of hydrogen into the intracellular space and the movement of potassium into the ECF. The final result is an increase in the plasma concentration of potassium. Conversely, an alkalosis will promote the production of hypokalemia without any immediate intracellular potassium deficiency occurring.

One situation where the result of these changes may manifest itself with potential danger to the patients is where intravenous therapy is employed to correct severe dehydration and possible associated acidosis. The initial plasma potassium concentration can be normal but falls rapidly as normal glomerular function is restored and potassium migrates intracellularly. In such circumstances, the use of potassium-free fluids should be avoided.

Table 10.13 Approximate volume and composition of alimentary secretions (adult, 70 kg)

Secretion	mmol/l					Liters/24 hours	pH
	H^+	Na^+	K^+	Cl^-	HCO_3^-		
Saliva	–	15	20	32	3	1.0–1.5	6.3–6.8
Gastric	80	50	20	150	–	2.0–3.0	1.0–1.5
Pancreatic	–	140	10	80	70	0.5–1.5	7.1–8.2
Biliary	–	140	10	120	30	0.5–1.0	7.0–7.6
Succus entericus	–	135	15	125	25	1.0–3.0	7.0–7.5

Most of the clinical upsets resulting from potassium depletion can be attributed to associated hypokalemia. One possible exception is the condition of adynamic ileus which seems to be due to intracellular potassium depletion. Balance studies have shown that in the adult, of a total body potassium of 3500 mmol, it is possible to have a deficiency as great as 1500 mmol (> 20 mmol/kg) following prolonged vomiting. Even with this magnitude of deficiency the membrane potential is affected less than where a change from 5 to 2 mmol/l occurs in the plasma concentration. The clinical symptoms of potassium depletion include muscular weakness, hypotonicity and paralysis, although tetany is occasionally observed. Polyuria due to renal tubular dysfunction is another well-recognized sign of potassium deficiency. Cardiac upsets occur including arrhythmias, tachycardia, hypotension and even ventricular fibrillation. Electrocardiographic changes observed comprise ST depression, prolonged Q–T interval and prominent U wave, T wave inversion and the appearance of bifid U waves.

Potassium intoxication is caused by the hyperkalemia almost invariably present rather than by an excess of total body potassium. This can be demonstrated by employing measures designed to cause the movement of potassium from the ECF to the ICF. Clinical improvement follows such therapy. The occurrence of hyperkalemia is always associated with a failure or overwhelming of the renal excretory mechanisms. The renal failure may be real as in glomerular or tubular disease or related to the lack of steroid hormones in Addison's disease. On the other hand it may be relative and due to excessive entry of potassium into the ECF. The latter situation is found in states of hemolysis, acidosis, infections, and injudicious potassium therapy. Cardiac arrest may occur if the plasma concentration exceeds 7.5 mmol/l, but this is very variable.

The electrocardiographic changes associated with hyperkalemia include peaked T waves, a prolonged P–R interval and ventricular slowing. These are valuable confirmatory signs of potassium intoxication but the clinical decision to employ treatment for hyperkalemia should be based largely on the result obtained for the plasma potassium determination. An exception to this statement is the description of electrocardiographic changes normally associated with hyperkalemia, but found in the presence of a normal plasma potassium concentration. These findings can be induced by administering potassium supplements too rapidly to patients with severe intracellular potassium depletion and hypokalemia. The hypokalemia is quickly corrected, but the intracellular potassium depletion persists. The membrane potential is altered to a value similar to that obtaining where a normal intracellular potassium concentration and hyperkalemia coexist.

THE LOW SODIUM SYNDROME AND WATER INTOXICATION

Whilst sodium depletion is often associated with hyponatremia, the occurrence of hyponatremia does not necessarily imply sodium depletion; it can be due to dilution. Unless this fact is appreciated, serious errors in therapy will result. The following are causes of hyponatremia:

1. true sodium depletion
2. water intoxication
3. diuretic-resistant edema, with or without uremia
4. inappropriate secretion of ADH
5. new 'steady states'
6. dilution of plasma sodium by excess glucose, fat or protein.

Categories 1 and 6 have already been mentioned.

Water intoxication

Water intoxication is the state which results when excretion of water is unable to keep pace with intake and production of water. It is especially liable to occur when prompt diuresis by the kidney fails to occur. It may be found in infants (relatively inadequate tubular function), following operations (increased ADH), in adrenocortical insufficiency (lack of cortisol), or when parenteral hypotonic fluid is given in excess of requirements. Clinical manifestations may appear when water amounting to between 5 and 10% of the body weight has been retained. The excess water is distributed between the ECF and ICF causing reductions in the concentration of plasma sodium and in plasma osmolality. Hypokalemia is also likely to be present unless renal failure is severe. The PCV is theoretically normal, whilst hemoglobin level and plasma protein concentration tend to be low. Anorexia, weakness, nausea, vomiting, headache, confusion and coma have all been reported in this syndrome. That these upsets are due to intracellular overhydration can be proved by infusing a small volume of hypertonic saline, following which water moves from the ICF to the ECF, and clinical improvement results. The finding of a normal plasma sodium concentration excludes the presence of water intoxication.

Edema

Diuretic-resistant edema may occur in renal or cardiac failure and implies that renal excretion is impaired in spite of the possible presence of an increased ECF volume. The total body sodium may be increased but water retention is even more marked, resulting in hyponatremia. The sequence of events leading to this syndrome is complicated and involves excessive secretion and/or diminished inactivation of aldosterone and ADH together with possible changes in capillary permeability. The outcome is an increase in the volume of interstitial fluid but a decrease in plasma volume. This results in pre-renal azotemia which is further aggravated by sodium restriction. The therapeutic use of hypertonic saline in this syndrome often causes improved renal function with a decrease in the uremia even if the edema may not be diminished.

The syndrome of inappropriate antidiuretic secretion (SIADH)

Inappropriate secretion of ADH produces a clinical picture similar to that observed in water intoxication. The fundamental fault is that the secretion of ADH persists even in the presence of a reduced osmolality. This may be associated with head injury where the ADH is of posterior pituitary origin, or it may be found with certain tumors where a vasopressin-like polypeptide is elaborated by the neoplastic tissue.

Treatment of this condition involves water restriction together with any specific therapy against the tumor if such is practicable.

Steady states

New 'steady states': there are some patients, especially in the older age group, whose plasma sodium concentrations are habitually about 132 mmol/l. They have no signs of sodium deficiency and are not suffering from water intoxication. Given hypertonic saline they neither achieve normal plasma sodium concentrations nor do they derive any benefit.

TREATMENT OF FLUID AND ELECTROLYTE DISTURBANCE

To sustain life, the basic principles involve the correction of deficit or excess of fluid and electrolytes, compensation for abnormal loss by whatever route and maintenance of a physiological balance. Where a patient has severe dehydration with attendant shock and peripheral vascular collapse the first move is to expand the plasma volume. To this end, immediate intravenous infusion of citrated plasma or suitable plasma substitute at 20 ml/kg may be life saving. Even in the above dramatic situation all fluids supplied to a patient will basically contain water, solutes and calories.

Water and solutes are usually required in more than normal amounts but when parenteral nutrition is limited to a few days an intake of 20–35% of the average calorie requirement will normally suffice.

The treatment of mild dehydration of the hypotonic, isotonic or hypertonic type does not necessarily require intravenous therapy. Mild gastroenteritis will yield to gastric lavage followed by oral half-strength physiological saline in liberal amounts, and the water-deprived hypernatremia to adequate oral intake of quarter-strength physiological saline. Moderate diarrhea may be treated away from hospital by reconstituted oral solutions such as those of WHO, Armour (Dioralyte) or Searle (Rehidrat). Their composition in mmol/l is shown:

	Sodium	Potassium	Chloride	Bicarbonate	Glucose (g/l)
WHO	90	20	80	30	20
Dioralyte	35	20	37	18	40
Rehidrat	50	20	50	20	16.4

Rehidrat also contains sucrose (32.3 g/l), citric acid (1.76 g/l) and fructose (1.76 g/l). The higher sodium concentration (WHO) is good for high sodium loss as in cholera, but less good for young infants for whom it may be diluted. Monosaccharides are beneficial but disaccharides less so and lactose may be harmful.

Provided that renal function is good it is relatively difficult to overload these infants and 150–200 ml/kg body weight/day are necessary save for the neonatal period when less is required. The widespread use of such 'stock' solutions has produced a dramatic fall in mortality in malnourished infants suffering from diarrhea. Recourse to the intravenous route demands knowledge of daily requirements of fluid and electrolytes. Normal daily requirements for water and electrolytes are listed in Table 10.14 but provided renal function is good, wide tolerance exists.

In a few patients parenteral magnesium (as magnesium chloride) at 0.5 mmol/kg/day in 5% dextrose is required, therapy being governed by measuring plasma magnesium concentration.

Over and above the need to maintain hydration is the need to correct existing deficits. In a considerably dehydrated child the water deficit will lie between 30 and 150 ml/kg body weight and the deficit of sodium and/or potassium between 5 and 20 mmol/kg body weight.

Table 10.14 Average daily requirements for maintenance of fluid and electrolytes per kg body weight at various ages

Age	Water (ml)	Potassium (mmol)	Sodium (mmol)
Day of birth	50	0	0
Infant	150	3	3
Older child	50	2	2

Solutions for fluid and electrolyte replacement are given in Table 10.15.

ISOTONIC AND HYPOTONIC DEHYDRATION

These are by far the commonest patterns of dehydration resulting from sodium and water loss, with or without concomitant potassium loss. Fluids lower in sodium content are usually administered to infants as quarter- or half-strength physiological saline. Physiological (isotonic) saline tends to be used for older children. Energy is generally provided as 5% dextrose and a stock solution of half-strength physiological saline in 5% dextrose solution is a very useful fluid. The rate of infusion will be calculated to counteract continuing losses, to maintain fluid and electrolyte balance and to replace existing deficits. The aim should be to restore water and sodium deficiencies in 24–36 h and potassium deficit, if large, in 5 days.

It is important to stress to all attendants that because a target for administration over a 24-h period may be calculated this must not be divided by 24 and given at a standard rate for the first 24 h. Severe dehydration may require urgent partial correction, continuing diarrhea or vomiting increase the rate of flow required, or renal dysfunction mandate the reverse. The infusion should be started at a relatively fast rate for several hours at least, and progress reviewed on the clinical and biochemical findings.

Thus, in moderately severe dehydration a 1-year-old infant given 150 ml/kg body weight of half-strength 5% dextrose–saline in the first 24 h receives in this 150 ml water, 11.5 mmol sodium and 30 calories per kg body weight. For each of the first 6 h this infusion might run at 10 ml/kg body weight/h, being reduced to 5 ml/kg body weight as hydration improves. In addition, one might add potassium chloride to provide 3.0 mmol of potassium/kg/day. Severe diarrhea or vomiting would modify the rate of infusion in one direction and renal failure the reverse. Very young infants may require slower infusion and paradoxically more concentrated solutions (e.g. isotonic saline) initially to correct existing sodium depletion.

Clinical assessment of progress, tissue turgor, periorbital hydration, fontanel tension, urinary output and general appearances continues to yield remarkably helpful information.

In all but the most severe cases, continued small amounts of oral fluids to moisten the buccal mucosa are advised, and this route will gradually take over and replace the drip, which should be discontinued or reimplanted within 72 h.

The treatment of the basic cause of such dehydration will be undertaken synchronously with antimicrobials parenterally as indicated. When intravenous infusion is impracticable or impossible, smaller amounts of fluid may be given subcutaneously (with hyaluronidase) or intraperitoneally to supplement oral fluid therapy.

Table 10.15 Solutions for fluid and electrolyte replacement

	Approximate calories/liter	(MJ/l)
Dilute		
Dextrose 5%	200	(0.84)
Physiological / Isotonic } sodium chloride 0.9% (154 mmol/l)	–	
Dextrose 5% in sodium chloride 0.45% (77 mmol/l)	200	(0.84)
Dextrose 5% in sodium chloride 0.225% (38 mmol/l)	200	(0.84)
Fructose 5%	200	(0.84)
Dextrose 4% in sodium chloride 0.18% (31 mmol/l)	160	(0.67)
Water	–	
Concentrated		
Potassium chloride (10%) 1.35 mmol/ml	–	
Sodium chloride (11.7%) 2 mmol/ml	–	
Ammonium chloride (5.35%) 1 mmol/ml	–	
Magnesium chloride (10.20%) 0.5 mmol/ml	–	
Calcium gluconate (10%) 0.22 mmol/ml	310	(1.3)
Sodium bicarbonate (8.4%) 1 mmol/ml	–	
Sodium lactate (11.2%) 1 mmol/ml	340	(1.4)
Fructose 10%	400	(1.7)
Fructose 20%	800	(3.3)
Dextrose 50%	2000	(8.4)

(1 kcal = 4.18 kJ)

HYPERTONIC DEHYDRATION (HYPERNATREMIA)

The association of hypertonic dehydration with hypernatremia, convulsions and brain damage has long been recognized. The dangers of overconcentrated feeds have been highlighted. Although infantile gastroenteritis with associated fever, hyperventilation and diarrhea may predispose to the development of hypertonic dehydration, the risk is enhanced by the use of overconcentrated feeds. All such infants displaying significant diarrhea should have solid food and milk removed from their diet for the first 24 h. Hypertonic dehydration is much less obvious than is hypotonic or isotonic dehydration and is now uncommon.

The condition usually affects infants aged less than 1 year and the serum sodium very often exceeds 150 mmol/l. Elevation of chloride, urea and potassium along with hypocalcemia and possibly hypomagnesemia are also usually present. Brain shrinkage and fall in CSF pressure while arterial pressure remains essentially intact are probably responsible for the dilation and rupture of cerebral vessels which is a pathological feature of hypernatremia. Approximately one-third of such cases are noted to convulse, generally within 24 h of admission to hospital and sometimes in relation to treatment. Most infants have diarrhea at some point of the illness and the plasma pH tends to be low, averaging 7.1–7.2. The cause of the convulsions is almost certainly related to renal dysfunction which leads to metabolic acidosis and to a continuing hyperosmolar state from a combination of the raised sodium and urea concentrations. Sudden alterations effected by treatment may trigger convulsions. This is probably due to cerebral edema which may also prove fatal. Rose (1984) stresses that as hyperosmolality develops, the brain responds in two ways. Initially water leaves the brain producing brain shrinkage and the symptoms described above. If this were the only response, rapid lowering of plasma osmolality would restore brain size to normal and there would be little danger to the patient. Unfortunately, the brain can also adapt to the hyperosmolal state by increasing intracellular osmolality by accumulating osmotically active Na^+, K^+ and Cl^- from the plasma and previously inactive cell stores. He stresses that in hypernatremia, brain size decreases initially and then returns to normal in 24 h. Once such adaptation has occurred, an acute reduction in plasma osmolality causes water to move into the brain and cerebral edema results. A low pH and a high blood urea level (or BUN) should alert the clinician to the likelihood of fits during the early treatment of hypertonic dehydration. Whilst the absolute level of serum sodium may not be related to the tendency to convulsions the same is not proven for subsequent brain damage.

Hypertonic dehydration is frequently present with and predisposes to renal venous thrombosis. Fluid orally or by intravenous infusion for hypertonic dehydration in infants should avoid the use of 50% dextrose solution alone since the cerebral disturbance during therapy seems likely to be due to intracellular overhydration from additional intracellular osmotic particles due to deranged metabolism.

A polyionic preparation may be employed as first suggested in 1968 by Bruck et al. Their suggestion was a fluid containing 57 mmol of sodium, 25 mmol of potassium, 3 mmol (6 mEq) of magnesium, 50 mmol of chloride, and 25 mmol of lactate together with 7 mmol of phosphate and 100 g glucose per liter. In practice, a solution of half- or quarter-strength saline in 5% dextrose is usually employed. If there is acidosis present, however, it may be desirable to give the sodium as $NaHCO_3$ and not as NaCl, and the number of mmol of sodium should be calculated. Thus 1 l of 5% dextrose plus 39 mmol of sodium bicarbonate is osmotically equivalent to quarter-strength saline in 5% dextrose.

The rate of infusion is variable but is usually 100–150 ml/kg body weight/day, provided that abnormal losses have ceased and renal handling of water is adequate. Ideally the water deficit should be restored slowly over a period of 3–5 days. It is thought that the added sodium prevents some of the water migrating osmotically into the cells. As normal cellular function returns, the smaller osmotic particles reunite and cellular volume tends to decrease to normal. The use of peritoneal dialysis for the grossly uremic, hypernatremic hyperosmolar cases offers what may prove to be the most successful therapy at present available.

reduced skin turgor, sunken eyes and sunken fontanel; and with increasing dehydration these symptoms become progressively more severe, with increasing apathy until the child is more than 10% dehydrated, when shock and anuria develop. Children on high solute diets, who develop diarrhea, may develop hypernatremic dehydration in which case the signs of dehydration are less obvious although lethargy and irritability may develop earlier.

Loss of bicarbonate and potassium in the stool, poor tissue perfusion, hypoglycemia, ketosis and renal failure may all lead to severe metabolic derangement. Symptoms of lethargy and irritability are particularly marked in hypernatremic dehydration and its rapid correction with i.v. fluids may lead to cerebral edema as a result of fluid shifts across the blood–brain barrier. This can result in convulsions or even death.

MANAGEMENT

The management of children with acute infective diarrhea hinges upon the treatment of their dehydration. The type of treatment required will depend very much on the severity of their dehydration and on the facilities which are available. Most mild cases can be treated at home by their family practitioner with oral rehydration therapy but where dehydration is more severe, social circumstances are poor or where other complicating medical factors are present, hospital admission is required. In developing countries where hospital facilities may be poor, the use of oral rehydration therapy has revolutionized the management of dehydrated infants (Lancet 1983). In addition to the management or the prevention of dehydration one has to consider how best to feed these infants during both the acute and recovery phase of their illness and whether drugs have any role in their therapy.

Dietary therapy

In patients with very mild or absent dehydration it is important that an adequate fluid intake is maintained to prevent the development of dehydration. In well-nourished children feeds may be reduced in strength for 24 h and fluids given as an oral rehydration solution. It has been suggested, however, that in children over the age of 9 months there is no need to reduce the strength of feed, as complications due to persistent diarrhea are relatively uncommon, and feeds should also be continued in breast-fed infants (Brown 1994). In poorly nourished children it is important to try to maintain food intake so that malnutrition is not further exacerbated, as fasting may prolong the diarrheal illness.

The use of oral rehydration solutions (ORS) has led to a large fall in mortality from acute infective diarrhea and has made it possible to treat moderate and severe dehydration without needing always to resort to intravenous therapy (Table 11.18). In 1980 the World Health Organization (WHO) published guidelines for the use of an ORS which contained a sodium concentration of 90 mmol/l and a glucose concentration of 110 mmol/l. There is some concern that this sodium concentration is too high for children living in developed countries and a concentration of 60 mmol/l has been suggested as ideal for ORS solutions used in temperate climates. Where solutions need to be reconstituted with water prior to use, errors in dilution may lead to the production of hyperosmolar solutions which may further exacerbate the diarrhea.

Table 11.18 Major constituents of oral rehydration solutions (mmol/l)

	Sodium	Chloride	Potassium	Bicarbonate	Glucose
WHO–UNICEF	90	80	20	30	110
Dioralyte	60	60	20	10	90
Rehydrat	50	50	20	20	111

When giving ORS solutions to prevent the development of dehydration, maintenance fluids should be given as a solution with a sodium concentration of 35–60 mmol/l or a 90 mmol/l sodium solution (WHO) in a ratio of 1 : 1 with water or breast milk. It has been suggested that in moderate and severe dehydration twice the estimated total fluid deficit is given orally over 6 h with two-thirds of this being given as 90 mmol/l ORS over the first 4 h and the remaining one-third as water over the next 2 h. If the infant is now rehydrated maintenance therapy should be started, as outlined above for the prevention of dehydration; if still dehydrated the regime should be repeated and if the dehydration is worse intravenous therapy should be started. This therapy is likely to be successful in up to 95% of cases. Where ORS solutions with a lower sodium concentration (i.e. 60 mmol/l) are used, all fluids should be given as ORS.

If the infant is shocked, is unable to tolerate the oral fluids or has evidence of an ileus, intravenous fluids should be initiated.

Intravenous therapy

If a child is severely shocked intravenous fluid in a volume of 15–20 ml/kg should be given rapidly over 10–15 min. In less severe cases it is more usual to give 40–80 ml/kg of fluid over 4 h as 0.45% sodium chloride/2.5% dextrose, although some of this fluid may be given as a plasma protein solution.

Once the child has been resuscitated, rehydration should be planned over the next 24 h for 5% dehydration and over 48 h for 10% dehydration. The volume required for rehydration = % dehydration/100 × weight (kg) × 1000 ml. The rehydration solution should be given as 0.45% sodium chloride/2.5% glucose and maintenance fluid should be given as 0.18% sodium chloride/4% dextrose. Potassium should only be added to the infusions once urine flow has started. Requirements for potassium will vary greatly (2–10 mmol/kg/24 h) depending on the extent of the losses.

In children with hypernatremic dehydration, rehydration should continue over 48 h to prevent sudden fluid shifts which might precipitate convulsions. The rehydration is similar to that described above for normonatremic infants except that after an initial period of half strength (0.45%) saline this can be reduced to one-fifth strength (0.18%). Where hyponatremia is present the total body sodium may paradoxically not be depleted and therefore 0.18% normal saline can be used for both rehydration and maintenance. However, when the sodium falls below 115 mmol/l extra sodium may be required. In severe cases intravenous bicarbonate should be given while in milder cases it is likely that the acidosis will correct spontaneously as the child is rehydrated. If urine flow is not re-established once circulatory volume has been restored, the possibility of acute renal failure should be considered.

Drug treatment

As a general rule drug treatment has very little part to play in the treatment of the majority of children with acute infective diarrhea. Opiate-derived antisecretory and antimotility drugs may under

Table 11.19 Antibiotics used in the treatment of acute infective diarrhea

Infective condition	Antibiotic
Campylobacter enteritis	Erythromycin
Pseudomembranous colitis (*C. difficile*)	Vancomycin
Shigella dysentery	Co-trimoxazole
Typhoid	Co-trimoxazole or
Systemic salmonellosis	Chloramphenicol
Enteropathogenic *E. coli*	Gentamicin
Giardiasis	Metronidazole
Amebic dysentery	Metronidazole

some circumstances be harmful and should not be used. With certain pathogens treatment with antibiotics may be helpful although in the majority of cases treatment of the dehydration is all that is required. A list of antibiotics and their indications is shown in Table 11.19.

COMPLICATIONS

Acute infective diarrhea may be complicated by the extraintestinal spread of the infection with the development of intestinal perforation, abscess formation, septicemia and meningitis. If dehydration is pronounced acute renal failure may develop and if hypernatremia is corrected too rapidly cerebral edema with convulsions is likely to occur. This complication is less common when ORS is used as fluid shifts occur more slowly. In some children severe damage to the intestinal mucosa will lead to the development of diarrhea which persists long after the original pathogen has been cleared.

PROTRACTED DIARRHEA IN EARLY INFANCY

Protracted diarrhea may be defined as the passage of four or more watery stools per day persisting for at least 2 weeks. This definition encompasses a wide variety of disorders (Table 11.20) and a definitive diagnosis may only be made in approximately 70% of cases. In most instances, the protracted diarrhea appears to follow an episode of acute gastroenteritis in early infancy, although by the time of presentation to hospital it may be impossible to isolate an infective agent from the stool (Walker-Smith 1994). Furthermore, unless the situation is managed effectively at an early stage, a vicious cycle of malabsorption and malnutrition may result which will further compromise intestinal function and perpetuate the diarrhea.

Table 11.20 Causes of protracted diarrhea in early infancy

1. Food sensitivity/postenteritis syndrome
2. Cystic fibrosis
3. Celiac disease
4. Microvillous inclusion disease
5. Congenital chloride losing diarrhea
6. Congenital short bowel
7. Inborn error of carbohydrate absorption
   a. Sucrase–isomaltase deficiency
   b. Glucose–galactose malabsorption
8. Immunodeficiency
9. Hormone-secreting tumor (i.e. VIPoma)
10. Autoimmune enteropathy
11. Bacterial overgrowth
12. Nonaccidental injury (laxative administration)
13. Idiopathic

One proposed sequence of events is that an acute infective insult may sensitize the intestine to foreign proteins (usually cow's milk protein), and subsequent ingestion of the offending food antigen causes further damage to the intestinal mucosa thus continuing the diarrhea. Although the resulting enteropathy may be caused primarily by the protein content of the milk feed, disaccharide intolerance (particularly lactose) may also develop and the institution of an appropriate exclusion diet will often result in resolution of the diarrhea. In this situation, an exclusion diet may be necessary only for 2 or 3 months after which a normal weaning diet can be reintroduced. In some cases, particularly in the developing world, bacterial overgrowth of the small intestine may be an added complication and bacterial toxins themselves may impair mucosal function.

Celiac disease, cystic fibrosis, selective inborn errors of absorption and immunodeficiency states may all present with intractable diarrhea of infancy. These conditions are discussed elsewhere in this chapter.

Although it is not possible to attach a diagnostic label to some 20–30% of cases of protracted diarrhea, over recent years several new causes of the condition have been described. Mirakian et al (1986) reported cases of protracted diarrhea in which autoantibodies to intestinal enterocytes were present as part of a polyendocrinopathy syndrome. In the remainder of cases of protracted diarrhea, a strong family tendency may be present and in these patients there is a high mortality (Candy et al 1981). Many of these children have true secretory diarrhea which may be evident from the large volume of stool passed and from analysis of the stool electrolytes. A proportion of these infants may suffer from congenital chloride losing diarrhea or intestinal microvillous inclusion disease (Phillips & Schmitz 1992). Where intestinal secretion is present in utero there may be a history of polyhydramnios or premature labor.

INVESTIGATIONS

Collection and examination of the stool is vital and a careful search for intestinal pathogens (bacteria, viruses, parasites) should be undertaken at an early stage. The stools should be analyzed for the presence of reducing substances (> 1%) and stool electrolytes and osmolality should be measured to determine whether the diarrhea is osmotic or secretory in nature (Table 11.21). Chromatography of a fresh stool specimen may help in the

Table 11.21 Stool analysis in osmotic and secretory diarrhea

	Osmotic	Secretory
Osmolality (mosmol/l)	400	290
Na^+ (mmol/l)	30	105
K^+ (mmol/l)	30	40
$(Na^+ + K^+) \times 2$	120	290
Solute gap (mmol/l)	280	0

treatment for this should alert the attending pediatrician to the possibility of septic meningitis.

Hirschsprung's disease

Some infants with Hirschsprung's disease present with severe and recurrent enterocolitis, starting in the neonatal period. The infant becomes extremely dehydrated and toxic, and the mortality rate is about 80% if the condition is unrecognized and not properly treated. Between attacks there is usually constipation with abdominal distention. The cause of these episodes of diarrhea is unknown but may be related to the high intraluminal pressure proximal to the aganglionic segment, facilitating infection by bacteria and viruses. A rectal biopsy will establish the diagnosis, and colostomy will prevent further attacks.

Thrush diarrhea and other superinfections

This may occur in premature and feeble neonates who initially contract infantile gastroenteritis. Overenthusiastic use of antibiotics may favour excessive growth of the fungus *Candida albicans*. There is often perianal erythema and ulceration with the typical whitish monilial plaques. The whole gastrointestinal tract may be infected and the perianal skin secondarily infected by the fungus-laden stools. Recognition of the condition and the timely use of oral nystatin, 500 000 to 2 million units per 24 h, may save the infants's life.

Antibiotic usage may also result in overwhelming growth of resistant bacteria, particularly resistant staphylococci, in the alimentary tract with a resulting infective enterocolitis.

Ankylostomiasis

Normally, hookworm infestation is rare in infancy, but when it occurs it is usually severe. Hookworm infestation in the older child does not usually result in diarrhea but in the infant it leads to severe diarrhea with consequent anemia and dehydration. Failure to diagnose the condition in time results invariably in death.

Chronic refractory diarrhea

There is a group of chronic refractive diarrheas which include cow's milk intolerance, disaccharidase and monosaccharidase deficiency, immunodeficiency, short-gut syndrome, familial chloride diarrhea, vipomas, intestinal lymphangiectasia, acrodermatitis enteropathica and other malabsorptive states.

TREATMENT

Preventive

Prevention of infantile gastroenteritis can be achieved by paying close attention to hygienic measures in the preparation of feeds and maintaining a high standard of hygiene generally. In the hospital nursery, strict observance of measures to prevent cross-infection is necessary, because the disease, once it has occurred, can spread very rapidly. Strict isolation of cases and of contacts and barrier nursing are necessary in the ward. Breast-feeding is an important preventive measure. Oral vaccines against rotaviruses are now available and they are given in three doses. They are safe and effective (Griffiths et al 1995).

Parenteral fluid therapy (see also Ch. 10)

The treatment of the infant with gastroenteritis depends on the etiology, if known, and on the degree of dehydration. Patients with moderate and severe dehydration need parenteral fluids and hence must be hospitalized, while infants with mild dehydration may be treated at home or on a hospital outpatient basis.

Mild cases

The majority of infants with gastroenteritis suffer mild attacks. It is best to take such cases off milk and solids for 12–24 h. This period of 'starvation' will ensure some degree of rest for the intestines. In place of milk or solids, watery fluids are given in small amounts frequently, e.g. hourly feeds, and this will obviate vomiting. A total daily intake of at least 180 ml/kg will be required. 5% dextrose with 0.4% sodium chloride drinks have been used for many years with good results. Higher concentrations of dextrose should not be given as this may aggravate the diarrhea. After 12–24 h, when diarrhea is reduced, quarter-strength milk is given for 24 h, then half-strength and so on till gradually full-strength milk is taken without recurrence of the diarrhea. Solids can then be reintroduced at this stage.

Moderate or severe cases

When the dehydration is moderate or severe, the infant should be hospitalized as parenteral fluid therapy will be necessary. Fluids should be given intravenously, as fluids given by the subcutaneous route are absorbed erratically and too slowly for efficacious treatment. In infants, intravenous fluids can be given by scalp vein infusion or into a limb vein (pp. 1836–1838).

There are three main aspects of fluid therapy in infantile gastroenteritis, namely the type of repair fluid, the amount, and the rate at which it is administered. There are many regimes in use but there is little substantial difference between them. The following regime is simple and has been used for many years and found to be satisfactory. It is desirable, but not necessary, to have serum electrolyte estimations before starting off intravenous therapy.

First, the degree of dehydration is gauged from the history and clinical examination (using the criteria in Table 23.13), and the baby is weighed. If dehydration is very severe, with signs of peripheral vascular failure, it can be assumed that the infant has lost fluid equivalent to 10% of his bodyweight, with less severe dehydration 7–8%, and with moderate dehydration 5%. These assumptions are made because in most instances the weight of the baby just prior to the onset of the diarrhea is unknown so that the actual amount of fluid lost cannot be calculated. For example, if the baby weighs 10 kg and has moderate dehydration (i.e. 5% dehydration), the fluid lost is 5% of 10 kg = 500 ml. This amount must be replaced and is termed replacement fluid.

At the same time, the baby will have continued with normal fluid losses, and the amount necessary to replace these may be calculated as shown in Table 23.14.

This amount is termed maintenance fluid, and in our example above, the 10-kg baby (aged between 1 and 2 years) will need approximately 1000 ml. The total amount to be given intravenously over the first 24 h is the sum of the replacement and maintenance fluids, i.e. 500 + 1000 = 1500 ml. Half of this amount (i.e. 750 ml) is to be given in the first 8 h, and the other

Table 23.14 Approximate daily fluid requirements related to patient age

Age	Approximate daily fluid requirement
Up to 12 months	150 ml/kg body weight (70 ml/lb) up to a maximum of 1000 ml
1–2 years	100 ml/kg body weight (45 ml/lb)
2–4 years	90 ml/kg body weight (40 ml/lb)

half over the next 16 h. The rates at which these fluids are to be delivered will have to be adjusted so that these amounts will be administered in the stated times. Microdrip sets are readily available now and volumes delivered over a set period of time can be determined, preset as required beforehand and expressed as drops per minute. The continuing pathological fluid loss should be roughly assessed during the first 24 h, and this is added to the next 24 hours' therapy.

The next problem is the *type* of repair fluid to be used. For replacement therapy (i.e. over the first 8 h) half-strength isotonic saline containing 0.45% sodium chloride and 2.5% dextrose can be used. This solution contains 77 mmol/l of sodium and is a safe and effective replacement fluid for infantile gastroenteritis. Suitable maintenance fluids are quarter-strength isotonic saline containing 0.23% sodium chloride and 3.75% dextrose or one-fifth-strength isotonic saline containing 0.18% sodium chloride and 4.3% dextrose. In the example quoted above, 750 ml of half-strength isotonic saline solution would be given over the first 8 h and the other 750 ml of maintenance fluid (one-fifth or one-quarter strength) given over the next 16 h.

In addition to the fluid and electrolyte replacement therapy outline above, there are the problems of metabolic acidosis and potassium needs. In severe dehydration with marked acidosis, it is desirable to have the plasma bicarbonate estimated on admission so that the acidosis can be corrected with sodium bicarbonate solution. The deficit of bicarbonate in mmol/l can be calculated from the difference between the plasma bicarbonate in the patient and normal: the amount of sodium bicarbonate needed to correct the acidosis is calculated from the formula: 0.058 g of sodium bicarbonate per kg body weight would raise the plasma bicarbonate concentration approximately 1 mmol/l. Based on this, the amount necessary is given intravenously within the first 8-h period, and the volume given is subtracted from the volume calculated for replacement fluid. For example, if in the patient above the plasma bicarbonate is 10 mmol/l, the deficit is approximately 10 mmol/l (20 mmol/l–10 mmol/l). If 4.2% $NaHCO_3$ solution (one millequivalent or 0.84 g/2 ml) is used the volume needed to raise the plasma bicarbonate by 1 mmol/l is 1.4 ml/kg bodyweight. Therefore, the volume needed is $1.4 \times 10 \times 10$ = 140 ml, and the amount of replacement fluid to be given is modified to 750 ml – 140 ml = 610 ml. Alternatively, if base excess measurements are available the procedure described on page 421 can be used.

Although the serum potassium is usually within normal limits during the acute stage of dehydration, the total body potassium is reduced. After rehydration has been effected and urine passed, the serum potassium may fall. It is probably better not to give potassium at the beginning, but when urine is passed, potassium in the form of Darrow's solution can be given intravenously in a volume of approximately 100 ml/kg bodyweight, given over the first 24 h. When Darrow's solution is given the volume is subtracted likewise from the total volume of fluids to be given over the first 24 h. Otherwise potassium can be given as indicated on page 411.

Although the above regimen is outlined in some detail, the amounts, rates and types of fluid recommended serve only as a working guide. The pediatrician cannot leave the patient totally to the care of the nursing staff with instructions as to amount and rates of fluid to be delivered within 24 h. He must reassess the infant time and time again especially during the critical first hours and be prepared to make changes in the intravenous fluid therapeutic regime as and when necessary to suit changing needs.

In moderate and severe cases no oral feeds should be given during the first 24 h since the fluid needs are being met intravenously. Another reason for omitting oral feeds is that these infants are often drowsy, and may be immobilized to some extent by restrainers because of the drip, so that if vomiting should occur, aspiration into the lungs is a distinct possibility.

Oral rehydration solutions (ORS)

Following experience in the use of ORS in the treatment of cholera, it was later found that mild and moderate degrees of dehydration in infantile diarrhea can be treated totally by oral dehydration therapy. The standard World Health Organization ORS enjoyed widespread use in developing countries and this confirmed the usefulness of ORS. However, the use of WHO ORS met with certain problems such as cost, inability to dilute the salts correctly, use of contaminated water, deterioration of the packet contents, etc. A cheaper and equally effective method of ORS using boiled rice water was advocated as an alternative (Wong 1981), and since then rice-based ORS have been shown to be sterile, cheap, effective and acceptable in developing countries (Mehta & Subramaniam 1986, Roesel & Schaffter 1989). It is considered that the use of ORS in infantile diarrhea may substantially reduce the large number of deaths in developing countries.

Antibiotics and chemotherapy

The place of antibiotics and chemotherapy in the treatment of infantile gastroenteritis is a matter of some controversy. Although most of the agglutinable *E. coli* isolated from cases of gastroenteritis are sensitive to a wide range of antibiotics there is little evidence that antibiotics benefit such cases and may merely lead to drug resistance which has already proved to be a problem in certain epidemics.

Likewise in cases of viral origin chemotherapy will be ineffective and may be harmful in producing superinfection. In general, antibiotics may be justified in the treatment of cholera, *Shigella* dysentery, some cases of *E. coli* diarrhea, *Clostridium difficile* diarrhea, *Salmonella* diarrhea with parenteral infection and a few other bacterial diarrheas.

Other symptomatic treatment

The use of the so-called absorbents such as kaolin and pectin in infantile gastroenteritis is of doubtful value and similarly opiates and allied drugs to reduce gastrointestinal mobility can be only symptomatic in their effect. They may, however, reduce the number of stools without affecting the primary lesion itself and may serve a purpose in allaying unnecessary worry on the part of parents of infants with mild gastroenteritis.

FROM RBHSC MEDICAL GUIDELINES JULY 1999 SECOND EDIITON EDITION -CURRENT IN 2000

Subsequent management of moderate to severe dehydration

Maintenance fluids may be calculated as

Body weight	Fluid requirement per day
First 10 kg	100 ml / kg
Second 10 kg	50 ml / kg
Subsequent kg	20 ml / kg

5% dehydration = maintenance + 50 ml /kg
10% dehydration = maintenance + 100ml /kg
15% dehydration = maintenance + 150 ml /kg
In addition, remember to replace any ongoing losses.

Try oral rehydration therapy by mouth although nasogastric or intravenous fluids may be necessary.

<u>With normal serum sodium,</u>
treat shock if present. Then use 0.18% saline + 4 % dextrose as the intravenous fluid. Fluid may be replaced over 24 hours.

<u>With low serum sodium,</u>
treat shock if present. Then use 0.45 % saline + 2.5 % dextrose as the intravenous fluid over 24 hours.

<u>With hypernatraemic dehydration,</u>
treat shock if present. Then, use 0.45% saline + 2.5% dextrose. Calculate maintenance and replacement fluid requirements. Rehydration must take place slowly. Give maintenance fluids over 24 hours but replace lost fluid over 48 hours or longer.The serum sodium should be reduced slowly (< 5 mmol / 12 hours.)

Note, unless there is anuria, KCl is added to the intravenous fluid according to electrolyte results.

Royal Belfast Hospital for Sick Children- Managing Medical Problems For Children 2003 third edition

The advice on fluid was changed as follows

Choice Of Maintenance Fluids (Not Neonate)

This must reflect the anticipated sodium and glucose requirements. 0.45% saline +2.5% dextrose is usually suitable added KCl is often necessary.

The 1999 Guidelines used in my own Clinical Directorate were similar but changed in late 2001 in paediatrics medicine then and in surgical specialties by summer 2002 when No 18 solution was restricted only to consultant prescription with routine fluids being 0.45 % saline

PINDERFIELDS AND PONTEFRACT DISTRICT GENERAL HOSPITALS

Admissions in year 2000/2001 were:

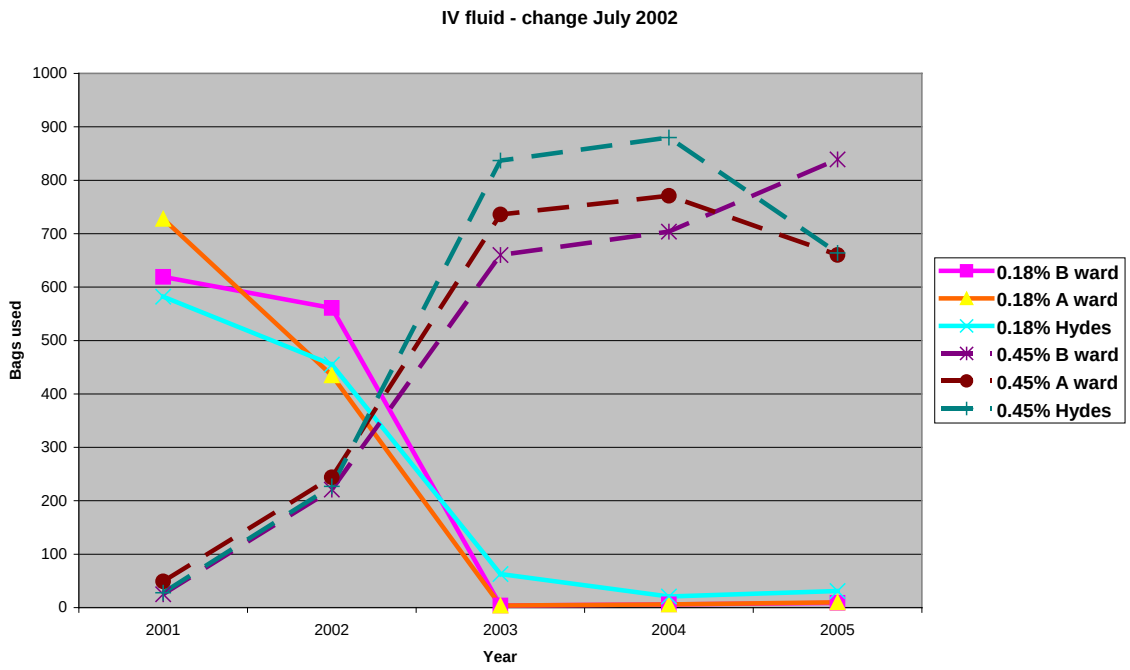
Paediatric 5300

Children's Surgical 2630 (Pinderfields 2140 and Pontefract 490)

Evidence of change in usage of IV fluid after change in policy January 2002

As a measure of change in practice the issue of IV fluids bags to the stock on the wards over the periods was analysed by the paediatric pharmacist and the results presented in Table and Figure which show the changes

Solution	Ward	Hospital	2001	2002	2003	2004	2005
0.18%	B ward	PGH	619	561	4	6	9
0.18%	A ward	PGH	728	435	4	6	10
0.18%	Hydes	PGI	582	455	63	21	31
0.45%	B ward	PGH	25	221	660	704	839
0.45%	A ward	PGH	49	244	736	771	660
0.45%	Hydes	PGI	28	227	837	880	664



ANNEX D RELEVANT GUIDANCE ON COMPLETION OF DEATH CERTIFICATES.AND REPORTING TO CORONER and consent in hospital autopsy

From Northern Ireland *Guidance On Death, Stillbirth And Cremation Certification*

Deaths that must be reported to the coroner

There is a general requirement under section 7 of the Coroners Act (Northern Ireland) 1959 that any death must be reported to the coroner if it resulted, directly or indirectly, from any cause other than natural illness or disease for which the deceased had been seen and treated within 28 days of death. The duty to report arises if a medical practitioner has reason to believe that the deceased died directly or indirectly:

• as a result of violence,misadventure or by unfair means;

• as a result of negligence, misconduct or malpractice (e.g. deaths from the effects of hypothermia or where a medical mishap is alleged);

• from any cause other than natural illness or disease e.g.:

- homicidal deaths or deaths following assault;

- road traffic accidents or accidents at work;

- deaths associated with the misuse of drugs (whether accidental or deliberate);

- any apparently suicidal death;

- all deaths from industrial diseases (e.g. asbestosis).

• from natural illness or disease where the deceased had not been seen and treated by a registered

medical practitioner within 28 days of death;

• death as the result of the administration of an anaesthetic (there is no statutory requirement to

report a death occurring within 24 hours of an operation – though it may be prudent to do);

• in any circumstances that require investigation;

- the death, although apparently natural, was unexpected;

- Sudden Unexpected Death in Infancy (SUDI).

• doctors should refer to the Registrar General's extra-statutory list of causes of death that are referable to the coroner (see pages 8 - 14).

The Coroner's Decision

Following the report of a death the coroner may adopt one of three courses:

1. Direct that the doctor should issue a Medical Certificate of Cause of

Death (MCCD).

After discussion the coroner and doctor may agree that the cause of death does not need investigated and the MCCD can be completed. You should record the discussion in the patient's notes.

2. Allow the death to be processed under the "pro-forma" system.

Coroner's Pro-forma

This is a special form for stating the cause of death and providing brief particulars of the background circumstances. Normally, the coroner will agree to use the *"proforma"* system where:

• it is a natural death and the only reason a death certificate cannot be issued is that the doctor has not seen and treated the deceased for the condition from which they died within 28 days of death;

• the cause of death is not a natural one but there are no suspicious circumstances e.g. a simple fall by an elderly person resulting in a fractured neck of femur and leading to the onset of bronchopneumonia as the terminal event;

• the cause of death is not a natural one but a post-mortem examination is unnecessary as a definite diagnosis had already been made eg asbestosis in a shipyard worker.

A doctor should not proceed to use the "pro-forma" system for a death without having first agreed that course with the coroner.

The pro-forma should be sent immediately by fax and followed by hard copy to the Coroner's Service. It should not be given to the family as they may confuse it with an MCCD and try to take it to the registrar.

DEATH CERTIFICATION

There is limited attention given in paediatric textbooks to this topic. Mainly it is related to the management of sudden unexpected death in infancy as part of the enquiries into "cot death" in the past.

The Paediatric Vade Mecum 14th edition does advise that a death should be reported to the Coroner if it cannot readily be certified as being due to natural causes.Or, the death may be related to medical procedure or treatment or,the case has any other unusual or disturbing features and that it may be wise to report any death where there is an allegation of medical mismanagement.

However the General Medical Council issues guidance and signposts to the specific guidance provided by the Office of National Statistics as well as that provided by government departments especially in Northern Ireland and Scotland. For reference therefore I provide therefore extracts from the Northern Ireland guidance and from the Office of National statistics. The guidance that I refer to is relatively contemporary but equally applied in 2000 and therefore is relevant to the circumstances pertaining to Lucy Crawford's death.

Guidance for doctors completing Medical Certificates of Cause of Death in England and Wales
From the Office for National Statistics' Death Certification Advisory Group, Revised July 2010

The purposes of death certification

Death certification serves a number of functions. A medical certificate of cause of death (MCCD) enables the deceased's family to register the death. This provides a permanent legal record of the fact of death and enables the family to arrange disposal of the body, and to settle the deceased's estate.

Information from death certificates is used to measure the relative contributions of different diseases to mortality. Statistical information on deaths by **underlying cause** is important for monitoring the health of the population, designing and evaluating public health interventions, recognising priorities for medical research and health services, planning health services, and assessing the effectiveness of those services. Death certificate data are extensively used in research into the health effects of exposure to a wide range of risk factors through the environment, work, medical and surgical care, and other sources.

After registering the death, the family gets a certified copy of the register entry ("death certificate"), which includes an exact copy of the cause of death information that you give. This provides them with an explanation of how and why their relative died. It also gives them a permanent record of information about their family medical history, which may be important for their own health and that of future generations. For all of these reasons it is extremely important that you provide clear, accurate and complete information about the diseases or conditions that caused your patient's death.

3 Who should certify the death?

When a patient dies it is the statutory duty of the doctor who has attended in the last illness to issue the MCCD. There is no clear legal definition of "attended", but it is generally accepted to mean a doctor who has cared for the patient during the illness that led to death and so is familiar with the patient's medical history, investigations and treatment. The certifying doctor should also have access to relevant medical records and the results of investigations. There is no provision under current legislation to delegate this statutory duty to any non-medical staff.

In hospital, there may be several doctors in a team caring for the patient. It is ultimately the responsibility of the consultant in charge of the patient's care to ensure that the death is properly certified. Any subsequent enquiries, such as for the results of post-mortem or ante-mortem investigations, will be addressed to the consultant.

In general practice, more than one GP may have been involved in the patient's care and so be able to certify the death. If no doctor who cared for the patient can be found, the death must be referred to the coroner to investigate and certify the cause.

If the attending doctor has not seen the patient within the 14 days preceding death, **and** has not seen the body after death either, the registrar is obliged to refer the death to the coroner before it can be registered. In these circumstances, the coroner may instruct the registrar to accept the attending doctor's MCCD for registration, despite the prolonged interval. In contrast, a doctor who has not been directly involved in the patient's care at any time during the illness from which they died cannot certify under current legislation, but he should provide the coroner with any information that may help to determine the cause of death. The coroner may then provide this information to the registrar of deaths. It will be used for mortality statistics, but the death will be legally "uncertified" if the coroner does not investigate through an autopsy, an inquest, or both.

5 How to complete the cause of death section

Doctors are expected to state the cause of death to the best of their knowledge and belief; they are not expected to be infallible. Even before any changes to the law, it is likely that there will be increased

scrutiny of death certification and patterns of mortality by local and national agencies as a result of the Shipman Inquiry. Suspicions may be raised if death certificates appear to give inadequate or vague causes of death. For example, if a patient dies under the care of an orthopaedic surgeon, it might be expected that some orthopaedic condition contributed to the death and so this condition would be mentioned in part I or part II of the certificate. Similarly, it would be surprising if a patient was being treated in an acute hospital, but no significant disease or injury at all was mentioned on their death certificate.

The level of certainty as to the cause of death varies. What to do, depending on the degree of certainty or uncertainty about the exact cause of death, is discussed below.

5.1 Sequence leading to death, underlying cause and contributory causes

The MCCD is set out in two parts, in accordance with World Health Organisation (WHO) recommendations in the International Statistical Classification of Diseases and Related Health Problems (ICD). You are asked to start with the immediate, direct cause of death on line Ia, then to go back through the sequence of events or conditions that led to death on subsequent lines, until you reach the one that started the fatal sequence. If the certificate has been completed properly, the condition on the lowest completed line of part I will have caused all of the conditions on the lines above it. This initiating condition, on the lowest line of part I will usually be selected as the **underlying cause of death,** following the ICD coding rules. WHO defines the ***underlying cause of death*** as **"a) the disease or injury which initiated the train of morbid events leading directly to death, or b) the circumstances of the accident or violence which produced the fatal injury". From a public health point of view, preventing this first disease or injury will result in the greatest health gain.** Most routine mortality statistics are based on the underlying cause. Underlying cause statistics are widely used to determine priorities for health service and public health programmes and for resource allocation. Remember that the underlying cause may be a longstanding, chronic disease or disorder that predisposed the patient to later fatal complications.

You should also enter any other diseases, injuries, conditions, or events that contributed to the death, but were not part of the direct sequence, in part two of the certificate.

THE FOREGOING GUIDANCE IS ECHOED IN THE GUIDANCE PROVIDED IN NORTHERN IRELAND WHICH FOLLOWS NORTHERN IRELAND *GUIDANCE ON DEATH, STILLBIRTH AND CREMATION CERTIFICATION*

MCCD= medical certification of cause of death

SUDI= Sudden Unexpected Death in Infancy

Registered Medical Practitioners have a legal duty to provide, without delay, a certificate of cause of death if, to the best of their knowledge, that person died of natural causes for which they had treated that person in the last 28 days. This is a statutory legal duty on all doctors based on Births and Deaths Registration (Northern Ireland) Order 1976, independent of any employment contract.

In hospital, there may be several doctors in a team caring for the patient who will be able to certify the cause of death. It is ultimately the responsibility of the consultant in charge of the patient's care to ensure that the death is properly certified. Foundation level doctors should not complete medical certificates of cause of death unless they have received training. Discussion of a case with a senior colleague may help clarify issues about completion of an MCCD or referral to a coroner.

A doctor who had not been directly involved in the patient's care at any time during the illness from which they died cannot certify the cause of death, but he should provide the coroner with any information that may help to determine the cause of death

Recording the Cause of Death.

The Cause of Death section of the MCCD is set out in two parts, in accordance with World Health Organisation (WHO) recommendations in the International Statistical Classification of Diseases and Related Health Problems (ICD).

Part I - Sequence leading to death, underlying cause You have to start with the immediate, direct cause of death on line I (a), then to go back through the sequence of events or conditions that led to death on subsequent lines, until you reach the one that initiated the fatal sequence. If the certificate has been completed properly, the condition on the lowest completed line of part I will have caused all of the conditions on the lines above it.

Part II - Contributory causes You should enter any other diseases, injuries, conditions, or events that contributed to the death, but were not part of the direct sequence, in part II of the certificate. Single condition causing death A single disease may be wholly responsible for the death. In this case, it should be entered on line (a) and the other lines left blank. More than three conditions in the sequence The MCCD has 3 lines in part I for the sequence leading directly to death. If you want to include more than 3 steps in the sequence, you can do so by writing more than one condition on a line, indicating clearly that one is due to the next.

More than one disease led to death If you know that your patient had more than one disease or condition that was compatible with the way in which he or she died, but you cannot say which the most likely cause of death was, you should include them all on the certificate. They should be written on the same line.

Results of investigations awaited If in broad terms you know the disease that caused your patient's death, but you are waiting for the results of laboratory investigation for further detail, you need not delay completing the MCCD. For example, a death can be certified as bacterial meningitis once the diagnosis is established, even though the organism may not yet have been identified. Similarly, a death from cancer can be certified as such while still awaiting detailed histopathology. This allows the family to register the death and arrange the funeral. However, you should indicate clearly on the MCCD that information from investigations might be available later. You can do this by circling "Yes" under section A on the back of the MCCD. It is important for public health surveillance to have this information on a national basis; for example, to know how many meningitis and septicaemia deaths are due to meningococcal or to other bacterial infections.

Terminal events, modes of dying, clinical signs and other vague terms. Terms that do not identify a disease or pathological process clearly are not acceptable as the cause of death. Description of terminal events such as cardiac or respiratory arrest, syncope or shock describe modes of dying not causes of death. Signs such as oedema, ascites, haemoptysis, haematemesis and vague statements such as debility or frailty are equally unacceptable.

The MCCD can only be completed by A DOCTOR who has seen and treated the patient for their cause of death within 28 DAYS before the death in any circumstances that require investigation; - the death, although apparently natural, was unexpected; - Sudden Unexpected Death in Infancy (SUDI). • doctors

should refer to the Registrar General's extra-statutory list of causes of death that are referable to the coroner (see pages 8 - 14).

RBHSC GUIDANCE ON CONSENT FOR AUTOPSY 2nd EDITION JULY 1999 APPLICABLE IN 2000.

AUTOPSY PROCEDURES FOR CHILDREN DYING IN THE RBHSC

Consultant paediatric pathologists Dr.Claire Thornton and Dr. Denis O'Hara should be contacted as soon as it is practical as they will give advice about how to proceed with arranging the autopsy. They are available by phone throughout the week and over the weekend at the following numbers.

Dr. C. Thornton
RVH: Ext. [redacted]
Home: [redacted]

Dr. M.D.O'Hara
RVH: Ext. [redacted]
Home: [redacted]

If the pathologists are unavailable at the above numbers, call their mobile phone [redacted]

General Information

There are two forms of autopsy - the Coroner's autopsy and the hospital autopsy.

The Coroner's autopsy is requested by the Coroner when the death falls into one of the following catagories

1. sudden, unexpected death (at home or hospital)
2. unnatural cause of death
3. anaesthetic death
4. when there is a possibility of litigation

Parental consent is not required in these cases and the family cannot prevent the autopsy.

If the death falls in to one of the above catagories, telephone the Coroner's office [redacted] and give the Coroner or his officer a short summary of the clinical history. Remember that both paediatric and forensic pathologists do autopsies for the Coroner so ask which pathologists are to perform the autopsy.

If the paediatric pathologists are to carry out a Coroner's post mortem, this will be done in the RVH

weeks later.

A hospital autopsy is requested by the clinicians and requires written consent from the next of kin of the deceased. It is performed by the paediatric pathologist or by their junior staff in the RVH mortuary. For a hospital autopsy, the pathologist requires the written consent form and the clinical summary on a completed request form. When it is complete, the pathologist will telephone the ward with the result and a death certificate can be issued if this has not already been done. A provisional summary is issued the following day by the pathologist and the final report is sent to the consultant clinician several weeks later.

When the patient has been under the care of the paediatric neurologists or neurosurgeons the autopsy, whether Coroner's or hospital, is generally carried out by the neuropathologists. To arrange this, contact Dr.M.Mirakhur at RVH ext. [redacted] Out of hours, call his mobile phone [redacted]

ANNEX E RESOURCES FOR ENQUIRY INTO CHILDHOOD DEATHS IN N.I. IN 2000

This Annex contains material from various sources and covers :

- **Enquiries Into Childhood Deaths : Processes In England**
- **From CESDI Report NI 2001**
- **MacFaul proposal from DH ENGLAND 1999 Audit of deaths of children in hospital: a national audit proposal (to NICE resulting in establishment of CEMACH)**
- **NCAS in Northern Ireland**
- **From Northern Ireland Guidance On Death, Stillbirth And Cremation Certification**
- **Relevant guidance on completion of death certificates.**

ENQUIRIES INTO CHILDHOOD DEATHS : PROCESSES IN ENGLAND

FROM WORKING TOGETHER TO SAFEGUARD CHILDREN Dept Children, Schools and Families (England) 2010 Chapter 7

The Regulations relating to child deaths

7.13 One of the LSCB functions, set out in Regulation 6 of the Local Safeguarding Children Boards Regulations 2006, in relation to the deaths of any children normally resident in their area is as follows:

(a) collecting and analysing information about each death with a view to identifying –

(i) any case giving rise to the need for a review mentioned in Regulation 5(1)(e);

(ii) any matters of concern affecting the safety and welfare of children in the area of the authority; and

A guide to inter-agency working to safeguard and promote the welfare of children **211**

(iii) any wider public health or safety concerns arising from a particular death or from a pattern of deaths in that area; and

(b) putting in place procedures for ensuring that there is a co‑ordinated response by the authority, their Board partners and other relevant persons to an unexpected death.

7.14 As explained in Chapter 3, the child death review functions became compulsory on 1 April 2008.

Supply of information about child deaths by registrars

7.15 Registrars of Births and Deaths are required by the Children and Young Persons Act 2008 to supply LSCBs with information which they have about the deaths:

●● of persons aged under 18 in respect of whom they have registered the death; or

●● of persons in respect of whom the entry of death is corrected and it is believed that person was or may have been under the age of 18 at the time of death.

Registrars must also notify LSCBs if they issue *a Certificate of No Liability to Register* where it appears that the deceased was or may have been under the age of 18 at the time of death.

7.16 Registrars are required to send the information to the appropriate LSCB no later

than seven days from the date of registration, the date of making the correction/ update or the date of issuing the certificate of no liability as appropriate. (The appropriate LSCB is the Board established by the children's services authority in England within whose area is situated the sub-district for which the register is kept). These requirements only apply in respect of deaths occurring on or after 1 April 2009.

A review of practice was reported in January 2009 of the period January-March 2008 to determine progress. Preventing Childhood Deaths Supplementary Survey 2008 Research Brief Dcsf By Peter Sidebotham University Of Warwick

JOINT REPORT RCPCH AND RCPath 2004

Because of deaths occurring in children from abuse and neglect, a more structured process has been put in place for the examination of all factors which may contribute a child's death and this process is in hand now over the country following recommendations of joint working party of the Royal Colleges of Pathologists and Paediatrics And Child Health chaired by Baroness Kennedy. *Sudden unexpected death in infancy-a multiagency protocol for care and investigation. 2004.*

RCPCH EMBARKS ON MAJOR REVIEW OF CHILD DEATHS 2012

15 May 2012 RCPCH news release:

Retrospective review of child mortality across the UK
Additional 'themed' study on mortality and morbidity in children and young people with epilepsy

With child mortality in the UK amongst the highest in Europe and limited current data on the causes of death, the national clinical outcome review programme - **Child Health Reviews-UK -** led by the Royal College of Paediatrics and Child Health will provide new insights into the conditions with which children die. It is hoped that the information will lead to improved outcomes for children and young people in the future.

The 2 year programme, funded by the Healthcare Quality Improvement Partnership, will cover two key areas, a retrospective review, looking at the characteristics of children who have died, and a themed case review focusing on mortality and morbidity in children and young people with epilepsy.

Retrospective review

Currently the only comprehensive national overview of underlying conditions and causes of death in children is based on death certificates which provide limited ancillary information. Data on co-morbidities, patterns of previous hospital admissions, and whether children with terminal illnesses are enabled to die at home are lacking. The Royal College of Paediatrics and Child Health review, led by Professor Ruth Gilbert, Professor of Clinical Epidemiology at the Institute of Child Health, UCL, will be the most comprehensive of its kind, and will:

- Link the information on children's hospital admission records across years and to death certificates

- Reclassify causes of death using information from death certificates and from the diagnostic codes recorded for hospital admissions immediately preceding death
- Categorise underlying chronic conditions, based on the child's entire hospital care record

It will seek to answer the questions:

- What are the characteristics of children who die?
- How are these characteristics changing over time?

From CESDI Report NI 2001

"A Survey of Risk Management in the HPSS Organisations" Report by Healthcare Risk Resources International – February 1999

Methodology

1. The survey assessed the 26 HPSS bodies against 12 specific risk management areas. The consultants graded the level of compliance on a score of 1 to 10 for each area in each organisation. A mark of 7 or more was equated to achieving full compliance. An overall average mark for each area was awarded, but the consultants emphasised that the averages, in some cases, disguised wide variations between organisations.

Assessment of Issues and Ratings Awarded

Issue 1 – Risk Management Strategy Documents – Rating: 5

"Almost all Trusts have produced a risk management strategy document. However, most are limited in their contents and a variety of models have been developed. It appears that greater efforts need to be made in order to ensure that the Strategy is endorsed fully by the Board of the Trust concerned and that **all** managers, clinicians and other professionals are fully aware of its contents. With regard to the four Boards and three Agencies, none of them has a contemporary, formal risk management strategy document."

Issue 2 – Risk Profiling – Rating: 6

"There is evidence of a reasonable amount of risk assessment activity with Health and Safety issues in all the organisations, but a limited amount of risk profiling of clinical and care services on a regular basis in Trusts. Where clinical risk assessments have been made, these have tended to be one-off focused risk reviews of particular, worrying clinical services (eg maternity) where there have already been indications of the need for investigation. The emphasis required is for a rolling programme of proactive risk assessments, as part of the organisation's normal business plan, covering every clinical, care and support service in a three-year cycle."

Issue 3 – Incident Reporting – Rating: 7

"There is generally a good level of reporting of incidents relating to Health and Safety issues, slips, trips and falls, with a great deal of data accumulated. Whilst in some of the organisations this is converted into meaningful management information, there is an inconsistent patchwork of manual and data processing systems in use for doing so. The major deficiency relates to the very limited and, therefore, probably significant under-reporting o*f clinical incidents and "near misses". A major effort is needed in almost all Trusts to improve in this area."

Issue 4 – Patient Records – Rating: 5

"There was a low level of compliance with this issue amongst the majority of Trusts. There is no doubt that inadequately prepared patient records, or records which are unavailable when needed, contribute to unsafe clinical care and indeed, can lead to claims of negligence being lost. Accordingly, there is a real need for most Trusts to develop an explicit policy document incorporating all of the elements shown, and for there to be a system in place for the routine audit of compliance with the policy."

Issue 5 – Clinical Audit – Rating: 5

"The consultants identified very few examples of multi-disciplinary clinical audit being used as a robust tool for risk reduction and risk control. However, there were many more instances of uni-disciplinary audit (for example, medical audit and nursing audit) and limited progress

towards the development of integrated care management."

Issue 6 – Complaints – Rating: 7

"In almost all the HPSS organisations, there were excellent systems for managing complaints from patients, their relatives and the public. Furthermore, the consultants found a lot of evidence to show that the systems are used effectively. This is not considered to be a high priority for improvement. However, because of the widening management agenda generally, it is necessary for the organisations to take steps to avoid complacency in this crucial area of risk management."

Issue 7 - Policies and Procedures – Rating: 6

"In all the organisations visited, there were many examples of excellent policies and procedures. However, in some cases, these were noted to be out-dated and, in a few instances, related to the predecessor organisation. Whilst there is much good practice in this arena, the importance of up-to-date, easily understood, clinical and other policies, procedures, guidelines, treatment protocols and agreed standards cannot be over-emphasised in relation to risk reduction. Often, a major cause of risk is that members of staff are individually uncertain of which is expected of them, particularly in emergency situations. This can be compounded when other members of the same team have different understandings about what actions should be taken in such situations."

Issue 8 – Communications – Rating: 6

"Generally, the HPSS organisations performed well under this heading. The majority visited had developed detailed communication strategies. Nearly all organisations visited had identified a senior manager to act as a focal point for overseeing external communications with relevant organisations and individuals. The approach…with combined healthcare and social service organisations, provides a significantly improved opportunity for interface between professionals engaged in clinical or social care input."

Issue 9 – Supervision of Junior Staff – Rating: 6

"In general, with regard to most non-clinical junior staff, there are effective systems in place for supervising their activities. However, consultants found few examples of formal, written procedures for ensuring that clinical staff have ready access to advice and support from their seniors. This does not imply that such processes are not in place, but these do need to be made more explicit. This is a particularly vulnerable arena in the context of clinical risk and needs more focused attention."

Issue 10 – Assessing Competence – Rating: 6

"This is an area which HPSS organisations are taking increasingly seriously and many areas are being addressed and reviewed. In addition, all organisations appear to have effective arrangements for individual performance review for staff. However, the consultants are concerned in particular about issues (dealing with procedures to verify the qualifications, references, police checks, health status and competence of all locum and agency staff to fulfil the duties required by the HPSS organisation, and the procedure for informing all staff of their responsibility to limit their actions to those for which they are competent), where they saw very limited evidence that the appropriate methodologies and procedures had been formulated. These are matters which need to be addressed urgently, as they can have a major impact on enhancing the risks to patients/clients in particular, but also to the organisation generally."

Issue 11 – Health and Safety and Related Issues – Rating: 8

"The consultants found examples of good work having been undertaken in all organisations regarding Health and Safety and related issues. Indeed, it is from these foundations that many of the risk management programmes have been built. The only point of concern with this issue is the possibility that some organisations may lose sight of the need to be continually vigilant in meeting on-going statutory and legislative requirements in this arena. Organisations cannot afford to become complacent in their pursuit of the wider challenging agenda, and should build on and maintain their current successes with Health and Safety and related issues."

Issue 12 – Claims Management – Rating: 6

"The consultants found few examples of a claims management policy in accordance with the detailed and helpful framework set out in (the Department's circular). It is likely that, because of the generally under-developed claims management function in most organisations, there is an excessive reliance on solicitors to manage claims of negligence. This incurs many costs which could be avoided if claims managers were given suitable training and more status within their organisation to genuinely manage the claims and the solicitors too. It is also important to note that, because of the central funding mechanisms for claims, there appears to be little financial or other incentive for HPSS organisations to pay more attention to this function."

FROM: CONSTITUTION AND STRUCTURE OF THE CONFIDENTIAL ENQUIRY

INTO MATERNAL AND CHILD HEALTH N.I.APPENDIX 6

VISION

This paper proposes that the key elements of the vision for CEMACH are that it should:

- be an enquiry with a nationally focused programme of work
- cover a wide programme including mortality, morbidity and near misses for mothers, babies and children
- be making a clear difference to clinical practice
- be part of the everyday working lives of the clinical professionals to whom its work relates
- have a robust study methodology which enables its results to play a part in the development of national clinical guidelines
- continue to produce epidemiological analyses of deaths of mothers and babies and, in the future, children
- carry out major projects and also minor studies designed to answer specific questions
- develop mutually supportive relationships with local perinatal surveys and equivalent local bodies
- attract wider sources of funding for work which is compatible with its core enquiry programme and ethos.

INFORMATION MANAGEMENT AND TECHNOLOGY

Information Management and Technology will also be an important part of headquarters activity. CEMACH will manage the software requirements of the enquiry and the arrangements for data security. This will be essential if it is to meet the increasingly stringent legal and policy requirements in this area. CEMACH will need to submit a section 60 application for exemption from the requirement to obtain consent for the capture and storage of patient identifiable information. Approval will only be possible if CEMACH can demonstrate that it is in control of the systems which support its activity.

RELATIONSHIP OF CEMACH REGIONAL OFFICES WITH REGIONAL PERINATAL SURVEYS

CEMACH will, though, want to work in partnership with local health services. There are clear benefits in the CROs working at a local level in a close alliance with related local bodies such as Perinatal Survey Units. Local survey units can gain from an association with a national programme. CEMACH will gain from easier access to local networks and dissemination of findings.

From CESDI (NI) 2001 report

Table 4.1 Sudden Unexpected Deaths in Infancy 1993-2001

Year	Total	Occurring in Neonatal Period
1993	24	5
1994	17	1
1995	10	1
1996	17	0
1997	18	1
1998	14	0
1999	11	3
2000	12	4
2001	10	2

Table Scale of child deaths after age 12months (MacFaul from ONS)

Age (E&W) 2001	***Mortality rate per 100,000 population***
Infants with birthweight ≤1500g Age ≤7 days	**18,000**
All infants Up to 28 days	390
28 days to 12 months	200
1 year to 4 yr	27
5-14 yr	14
15-34yr	61
35-64yr	400
65-74yr	2555
75 and over	8979

REF: NATIONAL INSTITUTE FOR CLINICAL EXCELLENCE: CONSULTATION DOCUMENT NOV. 2002 APPENDIX 7

PRINCIPLES OF RECORD KEEPING

- legible and neat using black ballpoint pen to facilitate photocopying
- to be clear, unambiguous and concise
- to be contemporaneous, accurate, relevant and complete
- no blank spaces (if information not requested draw line through space)
- identify time of day or night 24 hour clock
- correct mistaken entries promptly and properly
- state observations and action taken clearly
- document conversations between staff or staff to patients (including advice and discussions relating to care)
- clearly record care given by another member of the health care team
- record patient/health care worker non-compliance
- sign and date every entry in professional capacity

MACFAUL PROPOSAL FROM DH ENGLAND 1999 Audit of Deaths of Children in Hospital: a national audit proposal Deatinq4

1] NICE is asked to consider support for the following proposals, which contribute to the audit of care of critically ill children. A detailed submission from RCPCH is attached.

Background

2] Implementation of the recommendations of the SoS report on Paediatric intensive care has formed a prototype National Service Framework in which audit is integral. Care of critically ill children encompasses level 1 PIC much of which is provided in acute general hospitals rather than in PIC units. PICU s should provide all level 2 and 3 intensive care and a proportion of level 1 care. For PIC units an MRC research proposal from ICNARC into mortality and morbidity, corrected for patient severity, has been submitted. ICNARC itself provides an audit of PIC activity. However a child may die in hospital on children's wards or A&E or theatres and will have been critically ill though may not have been treated on a PIC. Such deaths should also be audited.

Proposed audit

3] The following proposal constitutes a broad audit of the care of children and is cross cutting in respect of the specialities and clinical services treating children.

4] A structured means of inquiry into each childhood hospital death has been proposed. This offers

(a) critical incident review for local action aiming firstly to ensure that all that was necessary had been done in a timely and satisfactory way and, secondly to identify what remedies should be put in place should deficiencies be identified within the service.

(b) central return of such data to enable an overview to be provided allowing both feed back to individual hospitals and a means of monitoring of services. This requires set up and maintenance of a central database supported by an expert committee to refine means of inquiry and to provide reports and feedback.

5] A small project was funded from DoH clinical effectiveness budget in 1998 to pilot such a standardised inquiry although this was set up initially to develop a validated audit instrument to develop for professionals and for use by regions as they chose. Dr Rob Ross Russell at Addenbrooke's hospital is developing a guideline for a structured inquiry into each hospital child death. This includes deaths in A&E, the wards, theatres and intensive care units. The pathway of care for each child is examined from referral into the hospital all the way through the course.

6] Following Secretary of State's announcement in respect of "*First Class Service*" that all specialist doctors should take part in a national audit, consideration was given to the range of current audits. This included NCEPOD, the maternal death inquiry and CESDI The opportunity was then taken to consider extending the clinical effectiveness work . and to establish a national audit into childhood hospital deaths. The proposal aims to audit clinical services. Thus a population based approach as has been proposed in Trent region by Prof Michael Clarke, examining the entire spectrum of child deaths with a focus on injury deaths¸ at this stage is not thought appropriate although it could form a model for later consideration.

7] in March 1999 , RCPCH made a submission which in summary proposes :

> Phase 1 - to establish a methodologically robust system for the audit of deaths of all children over 1 year of age and under 17 years occurring in hospital in England and to use this system to audit deaths over a pilot 6 months.
>
> Phase 2- To establish an enquiry mechanism for reviewing a sample of cases, using the results of the national audit, the East Anglian study and the experience of other Confidential Enquiries. The legal and administrative processes required to give the project the legal status of a confidential enquiry would be addressed in this phase of the project.
>
> RCPCH proposes working with other Colleges and Faculties involved in the care of children : RCS, RCN, RCAnaes, FPHM and GP. There will be a need to work closely with CESDI which is considering a similar study on infants below 12 m of age.

Numbers involved - is the proposal feasible ?:

8] In childhood the highest mortality rates are seen in children upto one year of age and after this age the rate is low until adolescence. After age 1 year the commonest cause of death is accident and about 40 % of the 1800 or so annual deaths in England occur out of hospital.

9] A standard district hospital covering a population of 250,000 will have 2,500 admissions a year for paediatrics of whom about 1% (25) will require paediatric intensive care. From that population in the age group 0 to 1 year there are likely to be about 20 to 25 deaths each year including perinatal and infant deaths. In the age group 1 to 14 years there will be 12 deaths each year for that population of which 9 will result from medical illnesses - the others are accidents - some will be unavoidable (cancer or congenital malformation) and others will occur prior to hospital admission. Numbers of deaths will be greater in regional centres where larger numbers of children with serious illness are cared for.

10] In 1995 there were 1765 deaths in children in England aged 1-14 years in England –from all causes in 1995 age 1-4 years deaths were 737 ; age 5-9 years were 470 and 10-14 years were 558. About 60 % take place in hospital. With these numbers an audit seems feasible. CESDI is considering an inquiry into post neonatal hospital deaths under age 12m which will be some fraction of the 1200 post neonatal deaths occurring each year from age 1m to 12m

Interim action

11] A step could be taken to “kick start” this exercise. The annual estimated cost of £70,000 for the central function could for its first year be requested as a contribution from regional PIC allocations 1999/2000. The additional local level costs should be absorbed by the service itself as a critical incident review should in any event be conducted into each in-hospital death.

12] Thus NICE is being asked to consider supporting this audit. Further work is needed to evaluate feasibility and costs. There are also some issues to be resolved surrounding confidentiality and a review of methodology will be necessary.

Dr Roderick MacFaul

Medical Adviser Paediatrics and Child Health Department of Health England

NCAS in Northern Ireland

Evaluation of the National Clinical Assessment Service

NCAS was set up as a Special health Authority following recommendations made in reports by the Chief Medical Officer for England[1,] 2. There had been concerns that tackling problems with medical issues required specialist skills not always available in individual NHS trusts3. In April 2005 NCAS became a Division of the National Patient Safety Agency (NPSA)4.

NCAS provides free, confidential, expert advice and support to the NHS in situations where the performance of individual doctors and since 2003 dentists, is giving cause for concern. It does not take on the role of an employer, nor function as a regulator. Prior to the establishment of NCAS concerns could be referred to the General Medical Council (GMC) even when not serious enough to justify regulatory action[3].

NCAS currently receives approximately 750 referrals a year5. Support can range from advice over the telephone, followed up by letter, through more detailed and ongoing support involving meetings between the adviser, case manager and practitioner, to a detailed NCAS assessment of a practitioner's performance. Employing organisations, managers or practitioners themselves can contact NCAS for advice. The referrer retains the responsibility for handling the case throughout the process[3].

1.1.1 NCAS in Northern Ireland

NCAS has provided services to the NHS in Northern Ireland since March 2005 under a Service Level Agreement with the Northern Ireland Department of Health, Social Services and Public Safety (HPSS). Discussion with NCAS regarding the way forward in particular cases and consideration of an NCAS assessment are two of the key actions when a performance concern first arises, and the involvement of NCAS should be considered at any stage in the handling of a case as employers or practitioners see fit. There is however a statutory obligation in the HPSS framework for handling performance concerns6 ('the Framework document') to involve NCAS in the course of disciplinary proceedings: NCAS must be notified when an employing body is considering exclusion or restriction of practice, so that alternatives to exclusion can be considered.

In July 2006 NCAS reported that the casework in Wales and Northern Ireland was running at half the anticipated rate7. This may indicate a different level of need, or that the later introduction of the service has resulted in the service not yet being fully used and further growth likely. There are currently two advisers dealing with cases in Northern Ireland.

The context of the NHS in Northern Ireland is distinct from that of England and Wales in being smaller and more self-contained. The majority of doctors in the service have qualified at Queen's University Belfast, and remain in Northern Ireland to complete their training. This leads to a familiarity between clinicians, especially at a senior level, which may not be found in the rest of the UK. The NHS in Northern Ireland also has a larger remit than its counterparts, with social services being part of the NHS rather than local authorities. There are related legal and procedural differences which add to the distinctive organisational and cultural context in which clinicians work, and so in which NCAS referrals will take place.

The NHS in Northern Ireland has also undergone recent organisational change, which may have a more temporary effect on the working environment. Eighteen NHS Trusts merged to form five Trusts in 2007, with some personnel changing roles as a result.

• Referrers' perceptions of NCAS as an organisation

Respondents had become aware of NCAS through a variety of means including direct publicity from NCAS, attendance at meetings and workshops, employers, the HPSS framework for handling performance concerns ('the Framework document') and journals. Two respondents (a medical director and a consultant) mentioned that their human resource departments had brought NCAS to their attention. The majority of respondents were aware of the referral process and the advice, support and assessment stages of an NCAS referral. The website had been helpful in providing 'step-by-step' guidance. Prior conceptions of NCAS were of a broad-ranging independent service mediating between employer and doctor, providing additional expertise and an additional tool where referral to the regulatory body (General Medical Council or General Dental Council) was not deemed appropriate.

• Perceptions of the role of NCAS and its relevance to them

The majority of referrals were related to clinical competence, although behavioural, health and probity issues were also mentioned. Respondents noted that issues often overlapped. Reasons for seeking input from NCAS, apart from referral being automatically triggered as part of disciplinary procedures, centred on: following local governance procedures, seeking reassurance, seeking objective and independent advice, seeking expertise, trusts reaching as far as they could with a case, and seeing NCAS as filling a governance gap. Respondents saw their role in the referral process predominantly as providing appropriate supporting information and felt that their role was clearly defined in the Framework document. They noted their responsibility in being supportive to the process and acting upon NCAS advice and recommendations.

• Feedback regarding the usefulness of NCAS in cases already referred by Northern Ireland

All respondents found NCAS to be approachable, accessible and neutral and expressed satisfaction with the service, supporting earlier findings. The majority of referrers had worked with a local adviser and, whilst some were unaware of the option to have an adviser from outside Northern Ireland, this would still not have been their preferred option as they valued local advisers' knowledge of the local, organisational and legislative context within Northern Ireland.

Advice stage

Advice had generally been considered useful, for example in terms of helping clarification of issues, providing assurance on the appropriateness of involving NCAS or other organisations, providing assistance with decision-making, in setting out actions to be taken and external validation of actions in process. The opportunity to make any corrections to the follow-up letter from NCAS was welcomed as was the openness demonstrated by copying letters to the Chief Executive of the trust.

Support stage

At the support stage respondents valued the supportive role of the case manager in collating information, keeping the referrer informed and providing clear correspondence.

Assessment stage

There was satisfaction with the independent NCAS assessments, with some reporting on the transparency of the process and arrangements, although there were some concerns about the reports, such as perceived time delays and perceived conflict in evidence provided by referrers and practitioners. Many respondents felt that additional support from NCAS would have been received if requested.

- The impact of NCAS activity on the management of medical and dental performance

  Some respondents indicated that their contact with NCAS had a wider impact on their practice, such as being able to use the experience and information they had gained to consider more options in future cases, although it was also noted that each case had its own specific issues. Some indicated changes in approaches to governance, such as recognising the role of organisational factors in performance issues. However, the main impact was recognition of NCAS itself as a tool for dealing with performance concerns. Respondents valued the independence of NCAS and the reassurance and confirmation it offered. Changes to the organisation of health services in Northern Ireland meant that organisational learning about NCAS may be limited in recent years.

  Implications for resources were referred to in the costs (and responsibility of those costs) of assessment, and in the necessity to provide service cover during periods of investigation and suspension. There appeared to be a lack of clear policy at a local level regarding payment for re-training.

- The nature of any unmet needs regarding the management of practitioner performance in Northern Ireland

  Many respondents commented on a difficult balance between patient safety, maintaining service delivery and their responsibility to be fair to the practitioner in question. The involvement of multiple stakeholders sometimes led to additional tensions, for example if NCAS recommended rehabilitation and the trust dismissal. A need to bring in expertise to help deal with the problem was seen as valuable. A difficult part of the process was the first contact with the practitioner: a hurdle which NCAS might be able to support via training in the future. Some referred to difficulty when there was a lack of insight on the part of the practitioner. There were particular difficulties in identifying and managing performance concerns in doctors in training and locums. This difficulty was in part that some issues were difficult to identify in short placements or if they were low frequency events and in part because of a lack of jurisdiction and control on the part of the employing trust. Respondents felt that NCAS was most suitable for clinical problems, but no general areas were seen as unsuitable.

- Opportunities for service improvement

  Overall, respondents were satisfied with the NCAS service. Suggestions for improvement included addressing the issue of performance concerns in non-permanent employees; dealing with problems related to short-term employment and subsequently resourcing any remedial action; improving publicity and raising the profile of NCAS as an independent service (potentially increasing self-referrals); providing training on initial engagement with clinicians; providing more information on NCAS timescales and providing anonymised NCAS case summaries as a resource for potential referrers.

Conclusion

- Referrers to NCAS had appropriate expectations of the service. NCAS was valued as an independent service providing expertise not always available locally, and filling a gap in governance between issues dealt with locally at trust level, and those which need to be referred to regulatory bodies. Referrers were largely satisfied with the service provided at all stages.

- There was some evidence that NCAS involvement could lead to changes in the way future cases are dealt with locally, although the unique circumstances of the majority of cases meant referrals were still likely with future cases.
- It was indicated by referrers that governance gaps exist in the current service with regard to practitioners who are not employed by trusts, and so can be lost between different jurisdictions. This applies to self-employed practitioners, trainees and, particularly, locums. Low frequency events which may indicate problems may not emerge when a practitioner moves between posts with any frequency.
- Referrers did suggest ways in which NCAS could potentially improve, both in the way it delivers its current service, and in which it could expand its delivery to a wider community. NCAS could make better use of the experience it is gaining as an organisation through training in referral and action planning, and dissemination of case outcomes, and could improve clarity in process, particularly with regard to timescales and the transparency of reports, including questions of risk associated with a practitioner. It was suggested that NCAS could increase awareness of its service, and ensure its status as an independent resource is clear. This may help medical managers considering options with a clinician presenting problems, and increase self-referrals by clinicians. Opening referral to patients and patient organisations may also increase the reach of the service. A suggested helpline which allows a potential referrer to gain advice without initiating an indexed case may also increase uptake. Conversely some referrers suggested that NCAS might consider how greater leverage can be applied to practitioners who do not engage with the NCAS process.
- Referral to NCAS may carry a stigma for the referring organisation as well as the practitioner involved. While marketing of the service, stressing its non-punitive nature, may address this perception, it may be something which will require deeper cultural shifts.

OTHER AVAILABLE RESOURCES 2001

The NHS publication "*Assuring The Quality Of Medical Practice*" published in January 2001 provided a valuable resource. It focused on the development of clinical governance, improving the complaints procedure and providing advice on how professionals should maintain their competence. It referred to the establishment of the NCAS as a special Health Authority and states that problems were not being dealt with well. Major problems often surface as a serious incident when they have been known about in informal networks for years (paragraph 5 of the introduction) the establishment of the authority NCAA aimed at providing a new performance assessment and support service to which a doctor can be rapidly referred were the concern about their practice will be promptly assessed and an appropriate solution devised. It will see an end to lengthy, expensive suspensions, multiple investigations of the same problem, variable local approaches and delay in acting to protect patients. It emphasises the need to be involved in continuing professional development, appraisal for all doctors underpinned by revalidation and that clinical audit is required of all NHS doctors. Each consultant should have a professional development plan. It quotes that clinical audit is an effective tool for reflecting on and improving care and that significant event reporting can help to celebrate good care while also identifying opportunities for improvement." (BMA"

It refers to "*An Organisation With A Memory*" published in June 2000 which identified 4 key categories of serious recurring adverse events and recommended that a new national mandatory system be established to record and analyse adverse events in healthcare. The document refers to an additional resource-the NHS *Clinical Governance Support Team* which from February 2001 had a website (now defunct but available at the time to Dr Kelly and others). It also made reference to the need to improve the process of complaints. Detail is given about how the problems faced by employers should be addressed. But importantly stresses in paragraph 4.4 that the employer or Health Authority remains responsible for resolving the problem at all stages. "Past experience however shows that local services have difficulty in dealing with complex problems of professional practice and it is likely that a discussion with the medical director of the NCAA might be very helpful in the initial stages of handling such problems. Much emphasis is given to the need to support failing doctors with their professional development and improvement as an alternative to disciplinary measures.

In 2010 the National clinical assessment authority published a bibliography which also indicates the resources which might have been available to a clinical director or medical director at the time. "*Bibliography understanding performance difficulties in doctors*"

The document *Organisation with a Memory* refers to the systems which can be seen as a mechanism for learning from adverse health care events which include the Confidential Enquiries and health and public health statistics and a range of internal and external incident enquiries. It acknowledges in paragraph 14 however that some of the confidential enquiries achieve good coverage but overall coverage is patchy with many gaps.

1. **British Association of Medical Managers (BAMM)** Support for medical managers such as medical directors or clinical directors has in the past largely been through the British Association of Medical Managers (BAMM) and they could have been looked to for advice to Dr Kelly in 2000/2001. BAMM was established by Dr Jenny Simpson in 1990 and closed in 2010. At this point Dr Simpson reported in the BMJ as follows[2]. It was created to improve care for patients "*by changing the way clinical professionals work with managers. We believe that dysfunctional organisations threaten patient safety and care. We set out to change the dialogue and cultivate an environment in which the dynamics of professional organisations are embraced and developed, rather than used as a convenient excuse for tribal warfa*re." Dr Simpson points out that networks of doctors interested in making the interface work between clinical practice and management were set up. Members included well-established medical and clinical directors but also junior doctors and some students. In 2004 standards for medical management and leadership framework were set. Dr Simpson refers to a joint board between the Royal College of Gen Practitioners and BAMM and suggests that other Royal Colleges might set up similar arrangements. *The way I see it. Whither or wither medical management? Jenny Simpson founder of the British Association of Medical Managers .BMJ Careers 04 Aug 2010*

2. ***The National Clinical Assessment Service (NCAS)****. NCAS is a national service. It was established in April 2001 following recommendations made in the Chief Medical Officer for England's reports, Supporting Doctors, Protecting Patients (November 1999), and Assuring the Quality of Medical Practice: Implementing Supporting Doctors, Protecting Patients (January 2001). NCAS works to resolve concerns about the practice of doctors, dentists and pharmacists by providing case management services to health care organisations and to individual practitioners. Our aim is to work with all parties to clarify the concerns, understand what is leading to them and make recommendations to help practitioners return to safe practice. We respond to calls about any aspect of individual or team practice, even where it is not yet clear whether there is evidence of poor practice. We also provide advice on long-standing and complex cases and we can discuss concerns without the need for the practitioner to be identified. Contacting us for initial advice does not commit the caller to making further use of our service. We do not take on the role of an employer so we do not investigate cases ourselves, nor do we function as a regulator. We are established as an advisory body, and the referring organisation retains responsibility for handling the case. Since 2001 NCAS has extended its coverage across the UK and associated states, within both the NHS and the independent health sector. We cover doctors, dentists and pharmacists working in primary and secondary care, including locums and postgraduate trainees. All our services, with the exception of team reviews, are currently free of charge to NHS organisations. http://www.ncas.nhs.uk/home/*

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ANNEXF F : LUCY CRAWFORD REPORT: ON DEVELOPMENT AND PRACTICE IN CLINICAL AUDIT AND GUIDELINES.

CLINICAL AUDIT

This professional activity became part of mainstream practice from the early 1990s following a number of reports. It is expected that clinicians will take part in clinical audit as part of their on-going professional development at all stages by the GMC, the Department of Health, and the Medical Royal Colleges and other professional associations and by professional for Nursing Midwifery and the Professions Allied to Medicine. Additionally NHS employing Trusts are expected to support clinical audit and to ensure it is in place as part of quality management and clinical governance.

The main purpose of clinical audit is to maintain and improve high-quality clinical care of the benefit of the patient. To do so clinical practice is reviewed to ensure that it meets what is required to manage individual or groups of patients and to identify substandard care.

Practice may be reviewed in a number of ways.

- Case note review.
- Topic audit.
- Drug and other therapy provision.
- Review of deaths and other adverse outcomes.

Case note review : this is usually conducted by randomly selecting some inpatient or outpatient case records of the clinical team involved. The quality of the record in its comprehensiveness and accuracy is reviewed and deviations such as omissions or failure to reach pre-set standards are logged. An opportunity is created when doing so in a group meeting of clinicians including those not directly involved in the case as well as the team responsible, to discuss and consider the clinical management of the case under consideration and to challenge and question the clinical care delivered to ensure that it is meeting current standards and, to identify any adverse events.

Topic audit. In this form of audit, a particular diagnosis or series of investigations may be selected and all the case records in a previous set period are collated and reviewed. The reviewer may be either a consultant who was responsible for provision of the care or a junior doctor in the team. The case notes collated will include those from different members of the consultant team. Usually here the audit is conducted by first creating a pro forma based upon the standards (e.g. guidelines) which are expected to be reached and then completing one for each patient. The data is then analysed and quantified often using statistical methods to determine the range of practice and opportunities for measuring improvement. Also adverse events are identified and logged. During the review of the case records errors made can be identified and recorded.

Process audit. Provision of therapy and services. In this form of audit, review of workload such as numbers of patients, length of stay, waiting time, use of medication or other therapies such as requests for imaging or pathology are reviewed with the aim of identifying appropriate use of resources or shortfall in provision.

Use of standards and guidelines

It is implicit in conducting audit that practice is compared with standards which have been set. These standards may have been set by national specialist bodies or colleges or by the Department of Health or, by a hospital itself when it adopts guidance out from elsewhere modified for use locally and local guidance which drawn up by a department in the form of protocols for local use. (See section below on guidelines).

Information system support

It is also necessary that good hospital information systems are available and used to identify patients with diagnoses selected for topic audit or e.g. using laboratory, imaging or pharmacy databases. Accurate diagnostic coding is an important element of this work.

Framework for Audit

Clinical Audit is conducted within the framework of:

- Structure
- Process
- Outcome

Necessarily these three components of audit overlap and the framework is best explained in reverse order.

OUTCOME

Although the ideal would be to examine practice against outcomes, identification of specific outcomes in general paediatrics other than mortality has proven challenging. However some examples include such outcomes as defined complications of a particular illness.

PROCESS

The process of clinical care of an individual patient or group of patients is compared with pre-set standards of care, for example guidelines. This is on the assumption that provision of this care standard will result in good outcomes. Here process is used as a proxy for good outcome.

STRUCTURE

When reviewing individual or groups of patients opportunities arise review whether or not the necessary resources were available in the institution in order to manage patients to the appropriate standard. This includes access to and use of medication, laboratory investigation or imaging and provision of sufficient numbers of staff, beds or isolation cubicles.

AUDIT REPORTS AND ACTIONS

Results of audit should be logged and major adverse events or lack of appropriate facilities should be attended to quickly. Additionally when deficiencies in clinical care are identified, reasons for this can be identified, discussed and actions taken to remedy deficiencies. Such actions include further training, provision of more or more clearly written guidelines, changing or updating existing guidelines or provision of additional guidance agreed by members of the clinical team. Then, in order to complete the “audit cycle” after a suitable period the topic is re-audited to quantify and record the degree of improvement and if this is not sufficient to address the reasons why. These processes may entail discussions with hospital management to ensure appropriate resources are available and additional training and provision of skills amongst clinical staff. Discussions may also be held with the hospital investigation departments and pharmacy. Each year a report should be provided of the topics which have been considered in the year and the actions which are followed. Guidance for 1990s was that the report should be sent to the hospital trust management but this in the event did not widely take place. Clinical staff needed to be able to show that they had been participating in audit and these should be listed in personal portfolios either as trainees or during review of continuing professional development on the part of senior staff. Nursing staff would also need to show and record their involvement in clinical audit.

Audit meetings

Audit meetings should be held regularly-monthly frequency is often adopted. They may form a separate meeting or afternoon session and may be conducted entirely within a hospital or, groups of hospitals may come together at a regional level to contribute to the topic under consideration and to offer opportunities for comparison. Frequently the regular postgraduate meeting which should be held in every paediatric Department is devoted to audit on one of its programmed sessions.

National Audits

In 1998 in *First Class Service* DH advised that all clinicians take part in national audits. For surgeons and anaesthetists this was the Confidential Enquiry Into Perioperative Deaths (CEPOD). For paediatricians in neonatal or general paediatrics one would be the Confidential Enquiry Into Stillbirth And Death In Infancy (CESDI)and from the 2000s to include deaths at all ages in childhood in the Confidential Enquiry Into Maternal And Child Health (CEMACH). Additionally all childhood deaths from mid 2000s are reported to the local social service department and all deaths reviewed in a multi-professional arena with emphasis on deaths which were possibly related to abuse/neglect.

Other national audit systems in place include audit programmes for neonatal intensive care, paediatric intensive care, the newborn bloodspot screening and other screening programs. Many specialties conducted audits through their national associations or colleges.

Milestones : In 1998 Department of Health proposed the establishment of a national audit into in-hospital children's deaths. There were delays in process as a pilot study was funded followed by transfer of identified funds to NICE and then later transfer of the responsibility for the development of this process to the existing Department of Health funded confidential Inquiry into still births and deaths in infancy CESDI one of the earliest national audits already in existence which on deaths in the perinatal period and up to age one year. Under the newly setup arrangements as a result of the negotiation starting in 1999, the Confidential Enquiry Into Maternal And Child Health was set up.

Most obstetricians and some anaesthetists would be involved to a greater or lesser extent in the CESDI. The majority of paediatricians were involved in returns to the British Paediatric Surveillance Unit which assembled information upon rare conditions and produced reports. The paediatric intensive care audit network was set up by the Department of Health and followed shortly by the neonatal intensive care audit network. All paediatric intensive care units and/or neonatal units would be expected to return information and receive reports back from this process. These processes are expected to be multi-professional.

The Department of Health (England) review of paediatric intensive care made recommendations in 1997 including centralisation and set up of networks of care. In 2002 paediatric intensive care audit network PICANET was set up to document and quantify outcome such as death or disability. Paediatric intensive care units form views about the quality of care provided in the district general hospitals which refer to them but from the first the level of feedback was limited. In my own experience up to 2006, feedback would be provided on request in Yorkshire region but it was not proactively offered. In my paediatric department from May 1997 onwards, all resuscitations including those that were referred for intensive care were reviewed in a critical incident process whether or not outcome was good and whether or not concerns had arisen about quality of care. This aimed to recognise good practice and potential for improvement as well as to identify adverse events. My experience was that a similar approach was not adopted in most Trusts at the time which focused on adverse events only and thus the RBHSC review of every death was above standard practice. If a child had been transferred to PICU we invited the treating intensive care specialists from Leeds to attend from 1999/2000 and frequently they did so.

MILESTONES IN CLINICAL AUDIT

Medical audit as a concept was introduced widely in the NHS in 1988/89 after which a range of guidance was issued. Recommendations regarding medical audit were embraced by the professions in a series of reports and recommendations. I provide extracts here from some of the key documents.

One of the earliest formal structures for auditing care was the practice within hospitals of perinatal mortality review meetings and reports (from the late 1960s). These conducted relatively structured evaluations of obstetric care involving midwives, obstetricians, pathologists, anaesthetists, paediatricians and general practitioners often with support from the local public health department. The Department of Health supported the National Confidential Enquiry Into Maternal Deaths. From 1989, the Confidential Enquiry Into Perioperative Deaths (CEPOD) was set up and one of its first reports addressed issues relating to children. The regional Confidential Enquiries into Stillbirth and Infant Deaths were collected into a DH supported national CESDI from early 1990s. The DH National Specialty Commissioning Group monitored outcomes in certain specialty services for children, notably liver surgery and cleft palate and after centralising to a smaller number of units documented clinical outcome improvement. In the 1990s DH funded a number of specialist national audits through the R Colleges and Specialist bodies.

The government *White Paper Working for Patients 1989 and Department of Health circular HC (91) Advice on medical audit of hospital and community health services* stated

a. *"Within the next two years, the government would like to see all hospital doctors taking part in what doctors themselves have come to call "medical audit" a systematic, critical analysis of the quality of medical care, including the procedures used for diagnosis and treatment, the use of resources and the resulting outcome for the patient." And in Medical Audit-Working Paper 6 HMSO. February 1989 defined audit as: "the systematic critical analysis of the quality of medical care, including the procedures used for diagnosis and treatment, the use of resources, and the resulting outcome of quality of life of the patient" it can therefore be seen that the primary purpose of clinical audit is to improve practice"*

Introducing this in 1998 the Health Secretary England stated that:

"… *from next year [1999], all hospital doctors will be required to participate in a national audit programme appropriate to their speciality or subspecialty externally endorsed by the new Commission for Health Improvement*.", and that:

"... individual doctors will be required to share their results confidentially with the Medical Director of their Trust and the Trust 's lead clinician responsible for clinical governance. In turn, doctors on the Commission for Health Improvement will have access to these data …".

Requirement to participate

The NHS Plan set out the requirement - taken forward within the Quality Taskforce - that:

"*All doctors employed in or under contract to the NHS will, as a condition of contract, be required to participate in annual appraisal, and clinical Audit, from 2001. This will underpin, and provide much of the data to support, the General Medical Council's mandatory five-yearly revalidation process, which is likely to begin in 2002. Subject to Parliament, by April 2001 all doctors working in primary care, whether principals, non-principals or locums, will be required to be on the list of a health authority and be subject to clinical governance arrangements. These will include annual appraisal and mandatory participation in clinical audit*". (para 10.10)

This strengthened existing requirement in the HSC1999/065 on clinical governance issued in March 1999 for all NHS hospital doctors to participate in clinical audit programmes, including speciality and

sub-speciality national audit programmes endorsed by the Commission for Health Improvement. NHS Trusts were responsible for ensuring that their doctors meet this requirement. In addition, all NHS organisations were required to report on their participation in, and the impact of, their clinical audit activities in their annual clinical governance reports.

ROLE OF GMC The General Medical Council makes clear in *Good Medical Practice: Duties of a Doctor* 1995 that doctors should "take part in regular and systematic medical and clinical audit." The requirement for clinical audit results were to be part of GMC revalidation, which was expected to begin in 2002.

(From Good Medical Practice 1995)

Para	Statement
3	Keep accurate and contemporaneous patient records which report the clinical findings, the decisions made, information given to patients and any drugs are the treatment prescribed
5	You must maintain the standard of your performance by keeping your knowledge and skills up to date throughout your working life. In particular you should take part regularly in educational activities which relate to your branch of medicine
6	You must work with colleagues to monitor and improve the quality of healthcare. In particular you should take part in regular and systematic clinical audit.
30	You must be satisfied that, when you are off duty, suitable arrangements are made fewer patients medical care. These arrangements should include effective handover procedures and clear communication between doctors.

DH view on mechanisms for ensuring the impact of clinical audit on service quality (2003)

Individual doctors are required to share their clinical audit results with the Medical Director and the lead clinician responsible for clinical governance in their Trust. In turn, doctors from the Commission for Health Improvement (CHI) (now CQC) will have access to these data when they visit the Trust to review each NHS organisation's local standards and clinical governance processes. Where clinical audit identifies problems in service quality, and especially where these have wider implications for resource investment and service management, the NHS Trust and Health Authority, Chief Executives should also have access to the results.

NHS Trusts must show the impact of clinical audit in their annual clinical governance reports. These annual reports are public documents available to the local health community.

As one mechanism for ensuring participation in clinical audit, CHI's 4-year rolling programme of clinical governance reviews was to pick up cases where this is not happening - publishing these within its reports and requiring an action plan to be agreed with the relevant NHS through performance management of clinical governance to identify and act on cases of poor uptake in national confidential enquiries.

Other possible mechanisms could form part of standard processes for performance management of the NHS or, alternatively, could be agreed through the Royal Colleges' own mechanisms.

DEVELOPMENT OF GUIDELINES

"Clinical guidelines are systematically developed statements to assist practitioner and patient decisions about appropriate health care for specific circumstances. "

(Institute of medicine. Guidelines for clinical practice: from development to use. Washington DC. National Academic press 1992.)

The development of guidelines and the next step, the transformation of these into protocols for daily use has been an evolving process.

In order for clinical audit to consider such matters other than structure such as records-quality of completion etc., it is necessary for some form of standard to be adopted by which a clinical topic management may be judged. Thus the presence of guidelines or protocols within the unit is an important component of clinical audit.

Their use was very variable in the early 1990s and 1980s. Although some national bodies were producing or developing guidelines, it is not possible for clinical units to await their delivery because clinical work continues and with an increasing focus on evidence-based medicine in the 1990s, it was often necessary for units to develop their own, not least because the number being produced nationally was addressing only a small subset of the clinical problems presenting daily to clinicians.

By the mid-1990s most hospitals in United Kingdom had some form of guidance provided in written form for junior doctors and nurses, including resuscitation protocols and to be used by all members of the clinical team.

In the mid 1990s it became evident that although guidelines were available upon the management of identified diagnoses, there were very few relating to the presentation of a child with an undiagnosed condition. From studies conducted by my own research group we identified that the commonest presenting problems in acute general paediatrics were breathing difficulty, feverish illness, diarrhoea and vomiting and a fit, and these presenting problems represented 80% of paediatric inpatient and accident and emergency attendance and were replicated in general practice. Consequently a process was set up funded both by DH and a medical charity to develop guidelines using a Delphi consensus process and systematic evidence review. Guidelines on the management of diarrhoea and vomiting and on breathing difficulty were produced by the group based in Nottingham. Because of limitation of resource and time it was not possible to develop a guideline on feverish illness. In my role in the Department of Health England I was able to identify this as a topic to be taken through the process for NICE -one of the first topics dealing with a presenting problem rather than an identified condition. A chronology is presented here on some guidelines relevant to paediatric practice here to indicate the state of evolution in the 1990s. The RCPCH and other R Colleges have produced guidance on Clinical Guidelines over the years.

Topic	***Date process started***	***Date published***	***Source***
Appropriateness of Paediatric Admission Protocol	1992		Not possible to gain consensus DH project
Advanced Paediatric Life Support manual First Edition		1993	
Advanced Paediatric Life Support manual 2nd Edition		1997	
Diarrhoea & vomiting	1996	2001	Nottingham Paediatric A&E group (funded part RCPCH

Topic	*Date process started*	*Date published*	*Source*
			mainly Charity)
Breathing Difficulty	1996	2009	Nottingham Paediatric A&E group
Seizure	1996	2003	Nottingham Paediatric A&E group
Asthma inhalers	1998	2002	NICE
Growth Hormone	1998	2002	NICE
Inquiry into Childhood Deaths	1999	2006	CEMACH via DH
Feverish Child	2001	2007	NICE
Head Injury	~2000	2002	NICE
PICAuditNetwork	2001	2002	DH handed to CQC
Neonatal Audit National		2003	DH handed to CQC
Epilepsy	~2000	2004	NICE
Urinary tract Infection in children	2001	2007	NICE
Evidence-based guideline for the Management of decreased conscious level	~2002	2006	It is noteworthy that the syndrome of inappropriate ADH secretion is mentioned once only in passing. Hyponatraemia receives attention
Diarrhoea and vomiting		2009	NICE: defines hyponatraemia as an electrolyte disturbance in which the plasma sodium concentration is less than 135mmol/l. But refers to studies in which the definition has been variously < 132 or < 130mmol/l

KEY REFERENCES

Medical audit – later clinical audit - was introduced widely in 1988/89.The following are the key documents applying in early 2000s.

White Paper Working for Patients 1989

Hospital Medical Audit, Kings Fund 1989

Medical audit-a first report: What, Why And How Royal College of Physicians 1989

The Quality Of Medical Care. Report of the Standing Medical Advisory Committee Department of Health 1990. HMSO.

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Report of a working party BPA Outcome Measures For Child Health 1992

BPA Paediatric medical audit 1992

Specialty Medical Audit-King's Fund Centre. Charles Shaw. 1992

Medical audit a second report Royal College of Physicians of London 1993

1993 Children first-a study of hospital services. Audit commission

GMC Good Medical Practice: Duties of a Doctor 1995

RCPCH . Clinical Audit in Paediatrics and Child Health – Some Examples. London: Royal College of Paediatrics and Child Health, 1997.

1998. Department of Health. *"First class service"*

NHS Plan 2000

HSC1999/065 on clinical governance issued in March 1999 for all NHS hospital

Organisation with a memory DH 2000

Principles for Best Practice in Clinical Audit a joint publication of NICE , Commission for Health improvement, Royal College of Nursing and the University of Leicester. 2002.