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Your Ref: DJD/NH/8/FER/0028

Our Ref: AD-0111-09

Date: 4th December 2009

Dear Mr Doherty,

Re Your Client(s): Mr & Mrs Ferguson
Privilege issue in respect of Raychel Ferguson's documents

Thank you for you letter of 2nd December 2009.

As requested, I enclose for your attention copies of medical reports dated 12th November 2002 and 27th January 2003 prepared by Dr John Jenkins, together with a report dated January 2003 prepared by Dr Declan Warde.

Yours sincerely,

Anne Dillon
Solicitor to the Inquiry

Secretary: Raymond Little **Deputy Secretary:** Bernie Conlon
Arthur House, 41 Arthur Street, Belfast, BT1 4GB
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Rachel Ferguson (Deceased) - Inquest at Belfast Coroner's Court 26 & 27/11/02

Date of birth: 04.02.92 **Date of death: 10.06.01**

This report has been prepared at the request of the Directorate of Legal Services, Central Services Agency following review of photocopied material from the casenotes relating to the admission of this girl to Altnagelvin Hospital in June 2001, together with other material.

Rachel was admitted with abdominal pain suggestive of acute appendicitis on 07.06.01 and subsequently underwent emergency appendicectomy. She was healthy and well with approximate weight 26 kgs and her preoperative blood investigations were normal (serum sodium 137mmol/l). Post-operatively she was initially felt to be making good progress but had vomiting and headache. At approximately 03.00 on 09.06.01 she began to have severe seizure activity with further subsequent deterioration despite resuscitation and intensive care. Unfortunately she subsequently died and evidence on CT scan and at post-mortem was consistent with the diagnosis of cerebral oedema related to hyponatraemia.

Her sodium was found to be 119 at 03.30 on 09.06.01 with a repeat specimen at 04.30 giving a result of 118, associated with low levels of potassium and magnesium. Rachel had received Hartmann solution during her surgery but subsequently this was changed to solution 18 (0.18% saline with 4% dextrose) at a rate of 80 mls/hr. She received a total of 400 mls between 02.00 and 07.00 and a further 1680 mls between 07.00 and 04.00 the following morning (09.06.01).

Comment

Solution 18 has been routinely used in Paediatric medical practice for a very long time and is rarely associated with any acute electrolyte disturbances such as were seen in this tragic case. However, this is largely related to the range of conditions commonly seen by Paediatricians and cared for within the medical (as opposed to surgical) environment. By and large these are not associated with the syndrome of inappropriate secretion of antidiuretic hormone. It has become increasingly recognised in recent years that a regime utilising solution 18 may not provide the right balance of sodium and free water for children with some clinical conditions, and particularly where there is an increased likelihood of failure to excrete water. This would include situations of stress, pain and nausea, and may be particularly common in the post-operative period.

This was well described in an editorial in the Journal "Paediatric Anaesthesia" in 1998 by Dr Arieff, but did not receive widespread publicity in journals likely to be read by most Paediatricians or Surgeons caring for children at that time. The potential dangers were highlighted to a wider clinical community in an article published in the British Medical Journal of 31.03.01 by Halberthal et al. However, it has to be said that this topic is not well covered in a number of standard paediatric texts. Many Paediatric Units were still using their traditional regimes based on solution 18 until further concerns were raised within Northern Ireland in September 2001 as a result of two deaths. Steps were taken to convene a Working Group who have subsequently prepared and distributed guidance on the prevention of hyponatraemia in children under cover of a letter from the Chief Medical Officer dated 25.03.02. This highlights the dangers of this condition and gives guidance as to how these can be minimised in everyday clinical practice.

While it is possible in retrospect to form the opinion reached by Dr Sumner that Rachel must have suffered severe and prolonged vomiting, this does not seem to have been the assessment of her

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Rachel Ferguson (Deceased) - Inquest at Belfast Coroner's Court 26 & 27/11/02

condition made by experienced staff at the relevant time. Sr E Millar records in her statement that "During the morning Rachel became increasingly more mobile and was able to walk to the bathroom with her dad. Rachel also was sitting up on the side of the bed colouring in and generally being bright and happy. Rachel vomited undigested food at 10.30a.m. and again at 1.00p.m. and 3.00p.m. but not large amounts. Rachel continued to be stable and in good form and gave no cause for concern." It seems that some individuals can develop a severe form of this condition in circumstances which are clinically no more severe than those experienced by many children in the post operative period and the reasons for this degree of susceptibility are currently not understood. It is for this reason that guidance has now been prepared and issued to increase awareness of this previously poorly recognised condition and to ensure that Units providing care for children take steps locally to introduce care pathways and/or fluid management regimes which take account of this possibility and minimise the risks of occurrence.

The deterioration in Rachel's condition occurred rapidly. The possibility of an electrolyte disturbance being the cause of the fit was considered by Dr Johnston and efforts made to obtain electrolyte results from the laboratory urgently. However, even by the time these became available her condition had further deteriorated and her pupils were found to be dilated and not reacting to light (evidence that increased intracranial pressure due to cerebral oedema had already caused pressure damage within the brain). Despite prompt resuscitation and further investigation and management this damage proved irreversible and led to the tragic outcome of her death.

Conclusion

Having carefully studied the statements provided by the doctors and nurses involved in Rachel's care my impression is that they acted in accordance with established custom and practice in the Unit at that time. It is however important that further details are obtained of relevant nursing and medical procedures and management in relation to fluid administration and post-operative monitoring of fluid intake, urine output and other losses such as vomiting. In particular information needs to be obtained regarding the local policy for post-operative fluid administration in children. Was the prescribed regime in this case in keeping with this guidance? If it can be confirmed that the frequency and severity of Rachel's vomiting was not outwith the degree expected by experienced staff in these circumstances and that the staff involved acted in accordance with local policies and guidance then, in my opinion, their actions do not amount to negligence.

The tragic outcome in this case rather highlights the current situation whereby one sector of the medical profession can become aware of risks associated with particular disease processes or procedures through their own specialist communication channels, but where this is not more widely disseminated to colleagues in other specialties who may provide care for patients at risk from the relevant condition. In the circumstances relating to this incident, it was only the tragic deaths of two children in Northern Ireland which alerted the wider clinical community to these concerns. These have subsequently been assessed and relevant guidance prepared and disseminated as outlined above.

Dr J G Jenkins MD FRCP FRCPCH
Senior Lecturer in Child Health & Consultant Paediatrician

Date of report: 12.11.02

Queen's University Belfast

united

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Directorate of Legal Services
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25 Adelaide Street
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BT2 8FH

DIRECTORATE OF LEGAL SERVICES
31 JAN 2003
Insp. CC.

27th January 2003

Dear Sirs

Re Rachel Ferguson (Deceased) IND TSO 19/15

Thank you for your further letter dated 23.01.03 and enclosed copy report from Dr Declan Warde, the Consultant Paediatric Anaesthetist retained to advise the Trust. These documents reached me today 27.01.03. In view of your request for an urgent reply I have not had the opportunity to consider the report in great detail, or to consult the references quoted. My initial impressions are that in many aspects Dr Warde's report does not differ significantly from previously available information. There is a slight difference in his calculation of the total amount of fluid given. He calculates this at 2160 mls whereas the figure given in Dr Sumner's report is 2220 mls and my calculation from the hospital chart was 2080 mls. These differences are largely due to the difficulty each of us have had in trying to interpret the figures given in the chart. It may be that you would wish to clarify this with those responsible for Rachel's care at the relevant time.

Dr Warde again makes reference to the significance of the vomiting. I pointed out in my report of 12.11.02 the importance of seeking further information regarding the frequency and severity of Rachel's vomiting in the opinion of senior staff, given the comments in the report by Sister E Millar. I have also not been provided with any further details of relevant nursing and medical procedures and management in relation to fluid administration and post-operative monitoring of fluid intake, urine output and other losses such as vomiting.

With regard to the involvement of the Paediatric Medical Staff, it must be remembered that Dr Jeremy Johnston only became involved as he happened to be in the ward with a Paediatric medical admission when Rachel's condition deteriorated. He immediately responded and provided appropriate treatment for her convulsion. This was successful in stopping the seizure. He contacted the surgical PRHO Dr Curran and advised him to contact his surgical Registrar and SHO urgently. Unfortunately it appears that it was some significant time before the senior members of the surgical team became available. In the interim Drs Johnston and Curran suspected the possibility that an electrolyte abnormality could be the cause of the fit and electrolyte profile and other blood tests were sent urgently to the Laboratory. Dr Johnston did his best to ensure that these results became available as quickly as possible. In the absence of the Surgical team he discussed the situation with Dr Trainor who was the Paediatric second term SHO on-call and busy in the Neonatal Unit at the time.

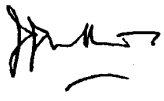
Dr Trainor went to Ward 6 and was informed that the electrolyte results had just become available showing a low sodium of 119 with potassium of 3. She immediately suspected that this might be an erroneous result if the blood sample had been taken from the same arm where the IV drip was running, but was told that this was not the case. It would be standard practice to arrange to urgently repeat electrolytes in this situation and this was performed. At this stage the seizures were under control. The main finding when Dr Trainor examined Rachel was of a petechial rash around her face, neck, upper chest, and her trunk appeared flushed although her temperature was normal. She was also unresponsive with dilated and non reactive pupils. Dr Trainor contacted Dr McCord the Consultant Paediatrician on-call and asked him to come to ward immediately. In view of the possibility of meningococcal infection Rachel was given intravenous antibiotics. Shortly after this her condition deteriorated again and Dr Trainor commenced resuscitation while the Anaesthetic Registrar was fast-bleeped. The Registrar arrived very quickly and assisted with resuscitation. Following this the results of the repeat electrolytes confirmed severe hyponatraemia and fluids were changed to 0.9% sodium chloride at reduced rate of 40 mls per hour.

Dr Warde questions why, upon receipt of the initial electrolyte results, IV therapy was not immediately changed to 0.9% sodium chloride. It is always easy to ask a question like this in retrospect, but the clinical picture had raised the possibility of meningococcal infection and this would be uppermost in the mind of someone whose experience was mainly in the medical aspects of the care of children, where this is a relatively common and immediately life threatening condition. The IV fluid was changed to 0.9% sodium chloride on receipt of the results of the repeat electrolytes (at approximately 04.30). In my opinion it is very unlikely that the continuation of the previous IV fluid for the relatively short period concerned is likely to have significantly worsened the prognosis, given that we now know that cerebral oedema must have already been present at that time.

Dr Warde raises the possibility that some would argue that faced with a symptomatic patient with acute severe hyponatraemia it would have been appropriate to be more aggressive and commenced treatment with hypertonic (3%) sodium chloride combined with a diuretic such as Frusemide. This would certainly not be indicated in a situation where a doctor was unsure as to the accuracy of the electrolyte results, and so would only be considered when the diagnosis had been confirmed by a repeat electrolyte check. Even then this is a treatment which requires specialist knowledge and experience and I would not expect it to have been commenced by a doctor of this level of seniority.

Finally, I wish to confirm my availability all day next Wednesday, 5 February 2003, but to point out that, as stated in my letter to you of 29 November 2002, I am not available on 06 February 2003 as I have a prior commitment to attend and speak at a meeting in London on that day. I will therefore be grateful if you can confirm details of my expected involvement as a matter or urgency as I have heard nothing further regarding this despite the request in my letter of November.

Yours faithfully



Dr J G Jenkins MD FRCP FRCPCH
Senior Lecturer in Child Health & Consultant Paediatrician

MEDICOLEGAL REPORT

Re: Rachel Ferguson, deceased.

Prepared for: **Ms Donna Scott**
Assistant Director of Legal Services
Directorate of Legal Services
25 Adelaide Street
Belfast
BT2 8FH

By: **Dr Declan Warde MB, BCh, BAO, FFARCSI**
Consultant Paediatric Anaesthetist
The Children's University Hospital
Temple Street
Dublin 1

January 2003

My name is Declan Warde and I am a Consultant Paediatric Anaesthetist with an interest in Paediatric Intensive Care.

I have been a Consultant in the Children's University Hospital, Temple Street, Dublin since 1986 and have served at various times since then as Chairman of the Department of Anaesthesia and Director of the Paediatric Intensive Care Unit in that institution. I am the author of several articles and book chapters on Paediatric Anaesthesia / Intensive Care. I served as representative for Ireland on the Executive Committee of the Association of Paediatric Anaesthetists of Great Britain and Ireland between March 1994 and March 1998 and as Honorary Treasurer of the Association from March 1998 until March 2002.

In preparing this report at the request of Ms Donna Scott, Directorate of Legal Services, 25 Adelaide Street, Belfast BT2 8FH, I have carefully perused all the medical and nursing notes and statements presented to me, together with the reports of Drs Herron, Loughrey and Sumner and have also reviewed the medical literature available to me on perioperative fluid and electrolyte management in children..

I believe that the facts I have stated in this report are true and that the opinions I have expressed are both correct and within my area of expertise.

Rachel Ferguson was born on February 4th 1992 and died on June 10th 2001.

Having been fit and well until earlier that evening, she was brought to the Accident and Emergency Department of Altnagelvin Area Hospital at 8 p.m. on June 7th 2001. She was complaining of sudden-onset and increasingly severe abdominal pain since 4.30 p.m. approximately. She had eaten dinner at 5.10 p.m. but had no appetite subsequently. She complained of nausea but had not vomited. Her temperature was normal. Her weight, perhaps estimated, was 26 kg. Clinical examination, which revealed right iliac fossa tenderness, guarding and rebound tenderness was consistent with a diagnosis of acute appendicitis.

Preoperative haematology and biochemistry were normal. In particular, her serum sodium was 137 mmol.l^{-1} (normal laboratory range for Altnagelvin Area Hospital 135 -145 mmol.l^{-1}). Urinalysis showed proteinuria +++.

Mr Makar, Surgical Senior House Officer, obtained consent for surgery. At 8.20 p.m. Rachel was given Cyclimorph 2mg by intravenous (i.v.) injection for relief of pain. She was subsequently admitted to Ward 6, kept fasting and commenced on i.v. fluids to maintain adequate hydration prior to surgery. Mr Makar initially prescribed Hartman's solution (sodium content 130 mmol.l^{-1}). He later changed this to Solution 18 (sodium content 31 mmol.l^{-1}) at Staff Nurse Noble's request after she informed him that his initial prescription was not in keeping with common practice on the ward. Solution 18 was

infused at 80 mls/hour until Rachel went to the Operating Theatre at which point in time it was discontinued, 60 mls having infused.

No premedication was administered. Consent for rectal analgesia was obtained from Rachel's mother in the Operating Theatre area. Anaesthesia was induced at 11.30 p.m. approximately. Intravenous Hartman's solution was commenced prior to induction. The exact amount infused in theatre is in some doubt. Dr Jamison's retrospective note of June 13th 2001 on the anaesthetic record indicates that this was 200 mls while her statement of February 3rd 2002 refers to a volume of 300 mls. The infusion was discontinued prior to Rachel's return to Ward 6. The anaesthetic and surgical techniques were routine and surgery proceeded uneventfully. Rachel was somewhat slow to awaken afterwards, presumably because of opioid administration, but was awake by 1.15 a.m. on June 8th.

Following her return to Ward 6, the Solution 18 infusion was recommenced at 80 mls/hour. Later on June 8th Rachel was noted to be apyrexial and free of pain. She vomited at 8 a.m. and had a large vomit at 10 a.m., at which time she also passed urine (volume not measured). She vomited "++" at 1 p.m. and again at 3 p.m. At 9 p.m. she vomited coffee grounds "++", had three more small vomits at 10 p.m. and a small coffee ground vomit at 11 p.m. She had been given i.v. injections of the anti-emetics Ondansetron and Cyclizine at 6 p.m. and 10.15 p.m. respectively. The vomitus volume was not measured and there was no nasogastric tube in place. At 9.15 p.m. Staff Nurse Gilchrist noted that her colour was "flushed to pale" and that she was complaining of headache.

Throughout the day the prescribed Solution 18 was infusing. I have calculated from the fluid balance chart that Rachel received 1840 mls from the time she left recovery until midnight on June 8th, and a further 320 mls between that time and 4 a.m. on June 9th, a total of 2160 mls. There is no record of any urine output other than at 10 a.m. Rachel does not appear to have taken any oral fluids or solids although Dr Zaffar, Surgical Senior House Officer had been happy for her to have small amounts of clear fluids orally from morning onwards.

At 3 a.m. on June 9th Staff Nurse Noble was informed by N/A Lynch that Rachel was fitting. She asked Dr Johnston, Paediatric Senior House Officer, who was in the ward, to attend urgently. Rectal and i.v. diazepam were administered, and were successful in controlling the seizure. Oxygen was administered via a face mask. Rachel's vital signs and airway were satisfactory. Dr Johnston suspected an electrolyte abnormality as a likely cause of the fit and asked Dr Curran, Surgical Pre-registration House Officer, to send blood to the laboratory for urgent analysis for electrolyte profile, calcium, magnesium and full blood count. Prior to obtaining the electrolyte results he went to the Neonatal Intensive Care Unit to discuss the scenario with Dr Trainor, Paediatric Second Term House Officer. During their discussion he was bleeped and informed that Rachel's condition had deteriorated. Dr Trainor went to Ward 6. The electrolyte results were now available and she noted that the sodium was 119 and the potassium 3 $mmol.l^{-1}$. She asked the Surgical Junior House Officer to urgently repeat the electrolytes and to send blood for

culture and gas analysis. On clinical examination she found that Rachel looked very unwell, was unresponsive with dilated and non-reacting pupils and that her breathing sounded "rattly". Her oxygen saturation was normal and her heart rate 160/minute. She had a petechial rash around her face, neck and upper chest, her trunk appeared flushed and her limbs were floppy. She spoke to Dr McCord, Consultant Paediatrician, by telephone and asked him to come to the ward immediately. She asked Dr Johnston to come and assist her – he inserted a second i.v. line and gave Rachel i.v. antibiotics in view of the petechial rash. Shortly afterwards the arterial oxygen saturation fell to 70% and Rachel became apnoeic. Dr Trainor commenced bag and mask ventilation and the Anaesthetic Registrar (Dr Date) was fast-bleeped at 4.00 – 4.15 a.m. He arrived quickly and intubated without the need for any drugs. The second electrolyte analysis revealed sodium 118 and magnesium 0.59 $mmol.l^{-1}$. Intravenous fluid therapy was altered to 0.9% sodium chloride at 40 ml/hour
nd Rachel was given intramuscular magnesium sulphate because of her low magnesium level.

At 5.30 a.m. approximately she was brought to the X-Ray Department for an emergency CT Brain Scan. The examination revealed evidence of cerebral oedema. There was a suggestion of associated subarachnoid haemorrhage although this was subsequently considered to be simply secondary to reduced brain density. A repeat examination was performed to outrule possible subdural empyaema.

Rachel was transferred to the Intensive Care Unit for continuing care and later that morning to the Paediatric Intensive Care unit at the Royal Belfast Hospital for Sick Children. She died at 12.09 p.m. on June 10th 2001.

An autopsy was performed by Drs Herron and Al-Husaini on June 10th. The major findings were of cerebral oedema and of diffuse hypoxic ischemic necrosis in the cerebral cortex. Specialist opinion was sought from Dr Loughrey as to the likely cause of the cerebral oedema. It was concluded that Rachel died from cerebral oedema secondary to acute hyponatraemia.

SUMMARY AND COMMENTS

Rachel Ferguson was a previously fit and healthy girl who was admitted to hospital suffering from mild acute appendicitis. Following appendicectomy, she experienced severe and protracted vomiting despite the administration of anti-emetics. During this time she was receiving Solution 18 intravenously. Some 27 hours postoperatively she

developed seizures secondary to acute cerebral oedema, itself secondary to acute hyponatraemia. She died approximately 60 hours following surgery.

Vomiting following appendicectomy is very common. Causes include administration of opioid drugs, traction on the peritoneum during surgery and side-effects of certain anaesthetic agents e.g. nitrous oxide. However vomiting as severe and sustained as that experienced by Rachel is rare – it is not clear why this occurred. It may be that in the later stages rising intracranial pressure was a contributory factor. Vomitus contains 70-100 $mmol.l^{-1}$ of sodium – this is less than that contained in plasma. However, if the sodium and other electrolytes lost through vomiting are replaced with fluids containing minimal electrolytes, such as Solution 18, , the net effect is sodium depletion with resultant hyponatraemia.

The "Syndrome of Inappropriate Antidiuretic Hormone secretion (SIADH)" has long been known to be associated with stress (e.g. anaesthesia and surgery), vomiting and pain. It leads to accumulation of fluid in the extravascular space and inhibition of excretion of excess free water – this syndrome is the most likely explanation for Rachel's apparently low urine output in the postoperative period. While accurate "fluid balance" (volume administered by all routes vs volume excreted by all routes) records are not available, I strongly suspect that Rachel was in significant "positive balance" (administered fluid in excess of that excreted) by the time she became acutely ill. In addition, the administered fluid (Solution 18) was low in sodium whereas much of that excreted (vomitus) had a significantly higher concentration. The inevitable end-result was acute hyponatraemia.

Administration of low-sodium solutions to children in the perioperative period is not uncommon. Their use, I believe, stems largely from the fact that it has been known for many years that sodium excretion in the presence of sodium loading is far less efficient in infants and young children than in adults. "Ward policies" regarding i.v. fluid administration in children's wards were developed, in part at least, to ensure that children were not given too much sodium, which can itself lead to major problems. Unfortunately such policies rarely took maturation of body organs with age into account. In reality, assuming appropriate volumes were given, the perioperative fluid and electrolyte requirements of Rachel, a previously healthy nine year old child, were far closer to those of a full-grown adult than to those of an infant or young child. In recent years there has been a steady move away from the perioperative use of such hypotonic fluids in children. There are a number of suitable alternatives available including 0.45% sodium chloride ("half-strength saline", sodium content $77mmol.l^{-1}$) in 2.5% or 5% glucose

Excess gastrointestinal losses in the postoperative period should be replaced with 0.9% sodium chloride (normal saline) with added potassium. If a hypotonic fluid such as Solution 18 is used for maintenance requirements, this necessitates the administration of two different i.v. fluid solutions simultaneously, which may be difficult. An acceptable alternative approach is to use a single fluid with intermediate electrolyte content e.g. Ringer's Lactate or Hartman's Solution for all perioperative requirements with added glucose and electrolytes as indicated by regular measurement of blood glucose and serum

electrolytes. I note that Mr Makar prescribed Hartman's Solution preoperatively but that this was not administered because of ward policy, and also that the anaesthetist used this fluid in the operating theatre.

Appropriate fluid and electrolyte management in the postoperative period in a patient with abnormal losses cannot, in my opinion, be achieved without electrolyte measurement and accurate estimation of fluid balance. Gastrointestinal losses could have been measured either by passage of a nasogastric tube to drain the stomach contents, or by ensuring that Rachel vomited into a container with volume markings. Urinary output could have been measured by bladder catheterisation or other means.

In my opinion, Rachel died as a result of developing acute cerebral oedema secondary to acute hyponatraemia, which was itself caused by a combination of severe and protracted postoperative vomiting, SIADH, and administration of intravenous fluid with a low sodium content. The relative contribution of each of these factors is impossible to quantify. Many cases of hyponatraemic encephalopathy occur because initial non-specific symptoms such as nausea, vomiting or headache are ignored; an alternative view is that many cases occur because the initial symptoms ***are*** non-specific. Nevertheless, a high degree of vigilance, accurate fluid balance recording and regular electrolyte evaluation in postoperative patients with these symptoms may help prevent future tragedies.

REFERENCES

Viliunas V. Postoperative hyponatraemia. WFSA Distance Learning Paper No. 19 (reprinted from Australian Anaesthesia 1998)

Arieff AI. Postoperative hyponatraemic encephalopathy following elective surgery in children. Paediatric Anaesthesia 1998; 8: 1-4

Liu L. Fluid management. In: A practice of Anesthesia for Infants and Children, 2nd Ed. W B Saunders Co., 1992

Rodney G. Fluid and Electrolyte Balance for Children . Anaesthesia and Intensive Care Medicine 2002; 3: 456-63

ADDITIONAL COMMENTS

Rachel's medical management from the time she began fitting was, in my opinion, in most respects entirely appropriate. I believe that many doctors of Dr Johnston's relative lack of seniority would not have suspected from the outset that an electrolyte abnormality was the root cause of the problem. One could question why, upon receipt of the initial electrolyte results (revealing sodium 119 $mmol.l^{-1}$), Dr Trainor did not immediately alter the i.v. fluid therapy to 0.9% sodium chloride but instead asked for a repeat estimation. Whether or not this would have made a difference to the ultimate outcome we do not know, but it may have been beneficial. Some would argue that faced with a symptomatic patient with acute severe hyponatraemia it would have been appropriate to be more aggressive and to commence treatment with hypertonic (3%) sodium chloride. However this solution may not have been readily available and once again one can only speculate as to the possible effect.