MEDICOLEGAL REPORT

ON

RACHEL FERGUSON

Deceased

dob: February 4th 1992

Died: June 10th 2001

Prepared for: John L Leckey LL.M
HM Coroner
Coroner's Office
Courthouse
37 Church Road
Newtownabbey
County Antrim BT36 7LA

By: Edward Sumner MA, BM, BCh, FRCA
Consultant Paediatric Anaesthetist
[redacted]

February 2002

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098-043e-150

My name is Edward Sumner and I am a consultant in Paediatric Anaesthesia with an interest in Intensive Care.
I was consultant at the Great Ormond Street Hospital for Children, London, from 1973 until June this year. I am the author of several textbooks on the subject and am the Editor-in-Chief of the Journal, *Paediatric Anaesthesia.*
Currently, I am the President of the Association of Paediatric Anaesthetists of Great Britain and Ireland.

In the preparation of this report I have carefully perused all the medical and nursing notes and statements presented to me, together with the reports of Dr Herron and Dr Loughrey.

I understand that my overriding duty is to the Court on matters which are within my expertise. I also believe that the facts I have stated in this report are true and that the opinions I have expressed are correct.

Rachel was born on 4th February 1992 and was a previously fit and well little girl with normal development.

On 7th June 2001 she was admitted to Altnagelvin Area Hospital via the Accident and Emergency Department complaining of sudden onset, acute abdominal pain with increasing severity at around 8 pm. She had eaten dinner at 5pm but after that had no appetite.

She was nauseated but was not vomiting. Her temperature was normal. The physical signs were of acute appendicitis with tenderness over Mcburney's point. Her weight was approximately 26kg.

Preoperative haematology and biochemistry was normal, notably the serum sodium was normal at137mmol.l^{-1}
The urine analysis showed proteinuria++
Consent for surgery and for rectal analgesia was taken from Mrs Ferguson in the theatre area. No premedication was administered and anaesthesia was induced at approximately 1130 pm. The anaesthetists were Drs Gund and Jamison and the surgeon Mr Makar.

The anaesthesia was routine and involved analgesia administered by the intravenous, rectal and local routes and a relaxant technique with intubation. She was also given an antiemetic. The anaesthetic form shows that she was given one litre of Hartmann's solution, but a witnessed, retrospective note states that only 200ml of this was actually infused.
Surgery finished after midnight on 8th June and postoperatively there seemed to be prolonged sedation from opioids, though she was awake in recovery by 0115. The IV infusion was to be recommenced in the ward.

The appendicectomy was routine. The peritoneum was clear and the appendix itself was mildly congested with an intramural faecolith. There was no Meckel's diverticulum.

Later on that day Rachel was noted to be apyrexial and free of pain, but she had vomited at 0800 and at 1015 she had a large vomit and again at 1300 and 1500. At 2115 the nurses noted "vomiting ++ (coffee grounds), colour flushed to pale, complaining of headache" and at 2300 there were three more small vomits. In spite of the vomiting Rachel had been able to walk during the day.

During this time she was receiving an intravenous infusion of solution 18 (0.18% saline with 4% dextrose) at a rate of 80ml per hour with a total of 540ml between leaving recovery and 0800 and a further1680ml between 8am and 4am the following morning (9th June) giving a total of 2220ml in 24hours. The fluid balance chart is confusing as the IV input is in the wrong column and I am not sure what is the significance of the AMT (150ml every hour). There is no note of any urine output or oral fluid intake, though it does say she was fasting during the night of surgery. There was no nasogastric tube at that stage.

On 9th June 2001 at 0315 Dr Johnson was called because Rachel had had a fit and had been incontinent. The seizure activity eventually responded to rectal and IV diazepam after 15 minutes. Oxygen was given. Although she was unresponsive, the vital signs were normal and the blood sugar normal at 9.7mmol.l^{-1} An electrolyte disorder was suspected and this was urgently checked. The electrolyte results from 0330 were: sodium 119, potassium 3, chloride 90, CO_2 16 and magnesium 0.59mmol.l –1 These were repeated at 0430 when the serum sodium was found to be 118, potassium 3 and CO_2 15mmol.l^{-1}

At 0630, the paediatric SHO noted that Rachel looked very unwell with pupils that were fixed and dilated. Her face was flushed with a rash and petechiae on the neck, probably from the vomiting. The chest was "rattly" and they wondered whether there had been aspiration into the lungs. The differential diagnosis at that stage was between the biochemical disorder and a cerebral lesion such as meningitis.

There is also an untimed note from the surgical registrar mentioning that Rachel was unresponsive with fixed, dilated pupils that she was intubated and that an emergency CT scan was organised.

At 0830 the anaesthetist was urgently summoned as Rachel had stopped breathing. He found her to be cyanosed and still vomiting. She was intubated without the need for any drugs, given antibiotics, intravenous 0.9% saline with magnesium and catheterized. Suctioning down the tracheal tube produced copious dirty secretions.

Later, the CT scan showed evidence of subarachnoid haemorrhage with raised intracranial pressure and at the request of the neurosurgeons a second, enhanced scan showed no evidence of a subdural collection of pus.

She was transferred to the intensive care unit and then to Belfast at 1110 at a time when she was hypothermic and with a negative fluid balance of one litre.

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Rachel eventually died the following day at 1209.

The postmortem examination was carried out on 11th June by Drs Al-Husaini and Herron. They found diffuse swelling of the brain with flattening of the gyri and effacement of the sulci. There was bilateral uncal swelling and uncal necrosis, plus evidence of diffuse hypoxic ischaemic necrosis due to perfusion failure. Their conclusion was that Rachel died from cerebral oedema due to hyponatraemia.

I would like to make the following comments:

1. Rachel was a previously fit and healthy little girl suffering from mild appendicitis.
2. Postoperative vomiting is very common indeed and has a variety of causes notably as a reaction to anaesthetic agents particularly the opioids such as fentanyl and morphine, but also after interference with the peritoneum. Vomiting is also a sign of rising intracranial pressure. Rachel was given antiemetic drugs, but suffered very severe and prolonged vomiting. We know this because of the presence of "coffee grounds" which is a sign of gastric bleeding and also the petechiae seen on her neck from straining.
3. It has been known for many years that after surgery there an accumulation of fluid in the extravascular space and that some degree of fluid restriction is necessary postoperatively for 24 to 48 hours. This known to be caused by the inappropriate secretion of Antidiuretic Hormone (ADH). The commonest regime to cope with this and prevent the deleterious effect of the excess water is to give 2ml per kilo body weight per hour for the first 24 hours of a solution such as 0.18% saline with 4 or 5% dextrose and then a little more the following day. During this time it is essential to replace gastrointestinal losses with an equal volume of 0.9% saline (normal saline) together with a potassium supplement until the patient is back to a normal feeding regime. Rachel was given approx 4ml per kilo per hour of the no 18 solution and no saline replacement for the vomiting losses.
4. Vomiting causes a severe loss of both water and electrolytes. Sodium and acid are lost from the stomach in the vomiting and as a compensatory mechanism the kidneys in trying to conserve sodium allow a net loss of potassium. If these dual electrolyte losses are not replaced with normal saline, but only a fluid containing 30mmol.l^{-1} then a state of hyponatraemia will develop acutely. The extent of the severe electrolyte losses seen in this case is reflected in the very low level of serum magnesium.
5. There is no doubt that Rachel suffered severe and prolonged vomiting. In my opinion there should have been fluid supplements administered, probably as early as 1030 on 8th June after the large vomit. It would also have been very prudent to check the electrolytes in the evening of that day, as the vomiting had not settled down by that stage. It is very uncomfortable, but with prolonged and severe vomiting after an abdominal operation, a nasogastric tube to drain the stomach and allow the gastric losses to be accurately quantified should have been passed. There is no evidence of any attempt to measure the gastrointestinal losses or the urine output – both essential for correct fluid therapy.

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6. By the late evening of the 8th June, Rachel had become extremely hyponatraemic, hypokalaemic and hypomagnesaemic. Hyponatraemia is usually defined as a serum sodium of less than 128mmol.l^{-1} so the levels found in Rachel were very low indeed and the changes from the normal values found preoperatively had occurred very quickly.
7. The brain is very sensitive indeed to acute changes in serum sodium levels and cerebral oedema from hyponatraemia with catastrophic consequences is very well documented in the medical literature. Although the skull is a rigid structure,as the brain swells, the intracranial pressure does not rise at once because CSF and blood are displaced from the cranium, but when this mechanism cannot cope, then the pressure rises rapidly and the brain is forced down into the foramen magnum – a situation known as "coning". At this stage there would be seizures and vomiting with the rise in intracranial pressure followed by changes to the pupils and loss of consciousness. Brain death follows if steps to reduce the cerebral swelling are not taken immediately as the intracranial pressure exceeds that of the blood supply. Rachel's clinical course vividly illustrates this.

To conclude and summarize, I believe that Rachel died from acute cerebral oedema leading to coning as a result of hyponatraemia. I believe that the state of hyponatraemia was caused by a combination of inadequate electrolyte replacement in the face of severe postoperative vomiting and the water retention always seen postoperatively from inappropriate secretion of ADH.

References:

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CHAPTER 8

Fluid balance: all aspects

LUCINDA HUSKISSON

This chapter aims to cover the basics of fluid balance. It is loosely divided into four sections. The first outlines fluid physiology. The use of crystalloid maintenance fluids is dealt with in the second. The third section deals with colloids and touches on the crystalloid:colloid debate for volume replacement. The final part covers the use of blood and its components in paediatric anaesthesia.

FLUID PHYSIOLOGY

TOTAL BODY WATER AND FLUID BALANCE

Total body water varies with degree of adiposity (90% of muscle weight is water vs 10% of fat), disease state and age. Ninety-four per cent of the body weight of a 12-week fetus is water and this falls to 80% by 32 weeks' gestation and 78% by term. There is a further reduction of about 5% in the first week of life, followed by a gradual fall to adult levels of 50–60% by 18 months of age.[1]

Body water is conventionally divided into three compartments: intracellular, extracellular and transcellular. In adults 60% of water is intracellular, 10% transcellular and 30% extracellular, of which 7.5% is intravascular. Although it is well recognized that disease states may affect the distribution of water in the body, it is less well known that age will also influence it. Extracellular water decreases from 60% in a 20-week fetus to 45% at term and falls a further 5% in the first 5 days of life.[1] Adult levels are reached by the end of the second year of life.

THE STARLING EQUATION

The extracellular compartment is further subdivided into the interstitial and intravascular spaces, with the interstitial space being three and a half times larger than the intravascular.[2] Fluid flux between the two was first described by Starling, who noted that the rate of fluid movement into or out of a capillary was related to the net hydrostatic pressure minus the net osmotic pressure.[3] The Starling equation:[4]

$$J_v = K_{fc}[(P_c - P_t) - \delta_c(\pi_c - \pi_t)].$$

where J_v = rate of fluid movement into/out of

capillary: K_{fc} = capillary filtration coefficient: P_c = capillary hydrostatic pressure: P_i = tissue fluid hydrostatic pressure: δ_c = reflection coefficient: π_c = capillary colloid osmotic pressure: π_i = tissue colloid osmotic pressure. has been further modified to incorporate coefficients which represent the permeabilty of the capillary membrane to small solutes (K_{fc}) and the reflection coefficient which describes the membrane's ability to prevent large molecules such as plasma proteins from crossing it: the Starling coefficient (sc).[5] If sc is 1, then a fluid can realize its full osmotic pressure: if sc for a membrane is 0 then fluids will pass freely across it and no pressure will be exerted. The coefficients vary between different organs of the body and are altered by disease. Burns, sepsis and cardiopulmonary bypass, in particular, reduce sc, resulting in capillaries which are increasingly 'leaky'. This has two effects: it allows water to leak out causing tissue oedema and it allows osmotically active particles to escape into the interstitial space. If sc then increases again, these particles will remain in the interstitial space, increasing its osmotic pressure and altering the balance of the Starling equation until they can be removed by the lymphatic system.

Most of the components of the Starling equation can be measured only with difficulty in the laboratory but the intravascular osmotic pressure and the capillary hydrostatic pressure can be measured clinically.[6,7] Guyton *et al.* describe certain 'oedema protection factors', such as increased lymphatic flow, which prevent the accumulation of oedema until the capillary hydrostatic pressure has increased by more than 15 mmHg.[8] This is supported clinically by the observation that in the absence of pulmonary capillary damage, the left atrial pressure (equivalent to hydrostatic pressure) must be increased to 15–20 mmHg before pulmonary oedema is seen.

One of the main differences between the fluid balance of adults and infants is the relatively large water turnover in the infant. The water contained within the extracellular space of a 70 kg man is about 14 l. Just under 3 l day^{-1} is lost in urine, faeces, sweat and during respiration (20%). In a 7 kg infant, the extracellular space contains about 1.6 l and obligatory losses are around 0.7 l day^{-1} (44%).[2] Any relatively small increase in losses will therefore have a much greater effect on a small child and this explains why diarrhoea remains such an important cause of infant mortality world-wide.

MAINTENANCE CRYSTALLOID REQUIREMENTS

MAINTENANCE WATER REQUIREMENTS

Although there are numerous formulae for calculating maintenance fluid requirements, it is important to stress that these are all guidelines only. They may be used as a starting point but the individual child's response to the fluid given must be monitored and appropriate adjustments made to the regimen.

The formulae available for calculating fluid requirement have as their basis body surface area (BSA), calorie requirement and the weight of the child.

Body surface area

Various nomograms are published which calculate BSA from height and weight. In older children the calculation of BSA is relatively easy and accurate because it is possible to obtain an accurate height. Measurements of the length of a neonate or small infant are not as reliable and errors of up to 20% in BSA are well recognized in babies less than 3 kg.[9] Because the height/length measurement is inaccurate, most centres now use a formula based on weight alone to calculate fluid requirement and the use of BSA has fallen from favour.[10]

Calorie requirements

The metabolism of 1 calorie requires 1 ml of water because, although 0.2 ml of water is produced, a further 1.2 ml is consumed. Therefore, 100 calories will require 100 ml of water for metabolism and knowing the calorie requirement of a child will also reveal the water requirement.[11] In 1911 Howland calculated the calorie requirement of an infant from 3–10 kg

to be 100 cal kg^{-1}, with older children needing 75 and adults 35 cal kg^{-1}. The extra calories metabolized by the younger children he attributed to proportionally larger surface area and growth.[12] In infants of less than 10 kg body weight, 50 cal kg^{-1} will be needed for basal metabolic requirements and the rest for growth. Children of less than 20 kg body weight need 1000 calories for the first 10 kg but only 50 cal kg^{-1} for the next 10 kg because of slower growth rate, and larger children and adults need only three times the calories of a neonate (1500 calories for the first 20 kg and 20 cal kg^{-1} thereafter).[11]

Normal maintenance fluid requirements calculated by weight

Whatever mechanism is used to calculate fluid requirements, it must be simple and foolproof because small miscalculations can result in significant errors in fluids administered. It has already been said that 100 calories requires 100 ml of water. A 25 kg child, therefore, requires 100 ml kg^{-1} for the first 10 kg (1000 ml), 50 ml kg^{-1} for the next 10 kg (500 ml) and 20 ml kg^{-1} thereafter (100 ml), making a total of 1600 ml per day or 66 ml h^{-1}. This can be simplified by assuming that there are 25 h in a day. The child then needs 4 ml kg^{-1} h^{-1} for the first 10 kg (40 ml), 2 ml kg^{-1} h^{-1} for the next 10 kg (20 ml) and 1 ml kg^{-1} h^{-1} thereafter (5 ml), giving an hourly total of 65 ml, which can be computed at the bedside without a calculator.

Neonates have greater fluid requirements than infants. As a general rule, most neonatal units allow 60 ml kg^{-1} for the first day, increasing by 30 ml kg^{-1} day^{-1} to 150 ml kg^{-1} for a term neonate and 180 ml kg^{-1} day^{-1} for a preterm. This requirement will be affected by environment (overhead heaters increase water loss compared with incubators), by whether the baby is ventilated (when there will be a humidifier in the circuit) and by the general state of the neonate. A premature baby with a patent ductus arteriosus may close the duct in response to fluid restriction and this may avoid the need for more aggressive management.

Table 8.1 Normal maintenance fluid requirements

Weight (kg)	Maintenance fluid requirement (cumulative values) (ml kg^{-1} day^{-1})
< 10	100
11–20	50
> 20	20

Dextrose requirements

In the UK, intravenous dextrose infusions are usually supplied as 4%, 5%, 10% and 20% strengths. In addition, 50% dextrose is available for management of hypoglycaemia. Most infants and children require 4% or 5% dextrose. A recent study from Germany, however, suggests that when these infusions are given peroperatively, children may become hyperglycaemic with dextrose concentrations as low as 2.5% although these were administered at large volumes equivalent to 200 ml kg^{-1} day^{-1}.[13] Neonates have poor glycogen stores and require higher glucose infusions to maintain their blood glucose levels. The majority of neonates, therefore, are traditionally managed using infusions of 10% dextrose which can be given through a peripheral cannula. Sick neonates on the intensive care unit, particularly in the presence of sepsis, may require higher infusions than this. Such patients may also need fluid restriction and it is not uncommon for small septic babies to need 20% infusions of dextrose. A neonate who cannot be fed enterally for more than a couple of days will require parenteral hyperalimentation rather than simple dextrose saline solutions. Hyperglycaemia can develop in response to stress. Both hypoglycaemia and hyperglycaemia can occur and blood sugar levels should be regularly monitored.

Electrolyte requirements

Electrolyte requirements vary with prematurity, losses and disease states. There is some debate as to whether a neonate needs sodium on the first day of life. Some units use a dextrose solution without added electrolytes, whilst others add sodium, potassium and calcium, particularly for premature infants. Preterm breast milk contains higher concentrations of sodium, calcium and phosphorus for the first 2–

4 weeks of lactation, and meets the preterm infant's increased requirements for these elements. Preterm formulae with similar electrolyte composition are also available. As a guide for prescribing, most children require 2–3 mmol kg^{-1} of sodium and 2 mmol kg^{-1} of potassium. This means that a 10 kg infant who needs 1000 ml of fluid per day will also need around 30 mmol of sodium. In the UK, fluids are available as dextrose (4% or 10%) and 0.18% saline (i.e. containing 30 mmol l^{-1}). For most patients, a ready-made bag of fluid will provide their electrolyte needs. In some instances, additional sodium will be required. This can be added to a standard bag using a strong sodium (30%: 5000 mmol l^{-1}) solution, or Normal saline (0.9%: 150 mmol l^{-1}) or half Normal saline (0.45%: 75 mmol l^{-1}) can be used in its place.

The amount of sodium administered to neonates in the immediate postoperative period needs to be monitored. Krummel *et al.* studied 20 surgical newborns and found that hypernatraemia occurred in 64% of term babies and 67% of preterms. In all cases this appeared to be due predominantly to an administered sodium load of more than 400% of the estimated maintenance requirements. This was compounded by a slightly reduced ability to excrete sodium and a short period of postoperative sodium retention.[14]

SPECIAL REQUIREMENTS

Gastrointestinal losses

Gastrointestinal surgery is relatively common in small infants. Ileus may also occur in a sick child: therefore, gastrointestinal losses are of importance. Nasogastric aspirates should be replaced volume for volume with normal saline containing 10 mmol potassium chloride per 500 ml. This sodium load allows the patient's kidneys to correct the hydrogen deficit incurred by the loss of gastric secretions. Stoma losses may also need to be replaced. High stomas in particular may be associated with significant sodium losses leading to as much a 6-fold increase in sodium requirements. Losses of more than 40 ml kg^{-1} day^{-1} are likely to require parenteral replacement using the same solution of normal saline with 10 mmol potassium per 500 ml. Usually, 0.5 ml is replaced for every 1 ml lost but, depending on the volume of losses and the site of the stoma, anything from one-third to three-quarter replacement may be required. Children who have ileus, either from gastrointestinal surgery and pathology or secondary to another cause, can secrete large amounts of fluid within the gut and peritoneal cavity. Babies with abdominal distension due to Hirschsprung's disease (colonic aganglionosis) may be intravascularly depleted in the presence of steady weight or even weight gain and such patients may need intravascular volume replacement despite normal indices.

Pyloric stenosis

Hypertrophic pyloric stenosis is a common condition with an incidence of about 1 : 200. All babies with this condition vomit and will require preoperative intravenous fluids. About 50% of patients will have a significant derangement of their electrolytes and acid: base status as a result of vomiting. The most useful electrolyte to gauge the seriousness of the metabolic upset is chloride, which can be used to calculate the chloride deficit and this must often be specifically requested as it is no longer performed routinely in most hospitals. While the serum chloride remains low, the infant will be alkalotic.[15] The vomiting of HCl, together with the kidney's attempts to conserve sodium, results in a metabolic alkalosis and a depletion of total body potassium. Because potassium is an intracellular ion, the serum potassium is a poor guide to potassium requirements and will usually be within the normal range.

To correct the metabolic alkalosis, the infant must be given sufficient sodium and potassium so that the kidneys can conserve hydrogen ions and correct the acid: base status. Chloride is also given as the anion to both sodium and potassium. Most babies with mild derangement (sodium bicarbonate < 35 mmol) will be corrected within 24 h using a solution of 5% dextrose plus 0.45% saline with 15 mmol of potassium chloride per 500 ml bag at 150–180 ml kg^{-1} day^{-1}. In extreme cases, normal saline (or 4.5% albumin which contains 150 mmol NaCl l^{-1}) may be required and 2–3 days of parenteral fluids may be needed preoperatively. It is also important to remember

that any gastric distension results in more gastric juices being secreted and lost. A wide-bore nasogastric tube should be left on free drainage with regular aspirations and any nasogastric losses should be replaced millilitre for millilitre with normal saline with added potassium (10 mmol in 500 ml).

Posterior urethral valves

Posterior urethral valves cause a congenital obstruction of the male posterior urethra and affected infants may also have a degree of renal dysplasia. Nowadays, the condition is increasingly diagnosed antenatally and most other children present within the first month of life. The initial management of a neonate with valves is to catheterize the patient and then confirm the diagnosis by cystogram and or cystoscopy. Surgery consists of valve ablation via the cystoscope. A significant number of these children will have renal impairment which may be long-term, and almost all have a diuresis in response to catheterization and relief of the obstruction. Urine output must be closely monitored in these patients and their fluid intake will be based on their creatinine and their output. Most of these babies are well enough to receive oral feeds but will also require parenteral supplementary fluids to keep up with urinary losses. This diuretic phase can last for between 24 h and a couple of weeks, again emphasizing that strict criteria cannot be laid down for neonatal fluid infusions.

Congenital diaphragmatic hernia

The fluid handling of a neonate with congenital diaphragmatic hernia merits special mention. There appears to be only a narrow path between hypovolaemia and fluid overload, either of which may have catastrophic effects resulting in a worsening spiral of acidosis and hypoxia. Rowe *et al.* studied the urine output and osmolarity of both urine and serum in 22 infants with diaphragmatic hernia vs 12 control infants undergoing laparotomy for some other reason. They found that although all controls responded appropriately, 64% of the diaphragmatic hernia group inappropriately retained fluid in the first 16 h after surgery and one-third still had an inappropriate urine output 24 h after surgery.[16] Fluid management of these children involves strict crystalloid restrictions (30 ml kg^{-1} day^{-1} for the first 24 h) and colloid boluses to maintain normovolaemia. Close monitoring of urine output and serum and urine osmolarity will help in the management.

Phototherapy

Neonatal 'physiological' jaundice is relatively common and is worsened by dehydration. Some neonates with a rising unconjugated hyperbilirubinaemia can be managed simply by liberalizing their fluids. This may need to be via a nasogastric tube or parenterally as the jaundice tends to make the baby sleepy and therefore less able to feed, which compounds the problem. If phototherapy is required, environmental water loss is increased significantly and an extra 25 ml kg^{-1} day^{-1} should be added to the fluids to compensate.

Effects of surgery

In 1968 Reid showed in adult patients that fluid accumulation occurred in the early postoperative phase and that this occurred entirely within the extravascular space. There was no change in the intravascular volume even when large fluid increases were seen extravascularly.[17] Seven years earlier, Shires *et al.* had studied fluid shifts peroperatively and noted an acute contraction of the functional extracellular fluid which, in the absence of blood loss, they presumed to be due to internal redistribution. They noted that the magnitude of the internal redistribution was related to the degree of surgical trauma and particularly to the duration and degree of retraction. They concluded that this was a major stimulus to the fluid and sodium retention seen postoperatively.[18] Certainly, after major abdominal surgery there is a fall in the serum sodium and evidence of fluid retention with periorbital and dependent oedema, which can be reduced by restricting fluids for the first 24–48 h following surgery. Following minor procedures patients are allowed their full maintenance fluids, but after any major surgery their intake is reduced to 50% of requirements for the first postoperative day, and if additional fluid is required it may be better to be given as colloid.

Sepsis

Hyponatraemia in sepsis is well recognized and is associated with a worse prognosis. Hannon and Boston looked at fluid and ion redistribution in an animal model of sepsis and found significant shifts of sodium, chloride and water into cells compared with sham controls.[19] They found this trend to be exacerbated by infusing 5% dextrose compared with normal saline with fluid shifts occurring when the volume infused was less than the estimated fluid requirement. They suggested that the hyponatraemia and plasma hypo-osmolality were caused by a combination of intracellular shift of sodium and water, and a dilution of the extracellular space, probably caused by physiological anti-diuretic hormone (ADH) secretion. Their conclusion was that, in the presence of sepsis, 4% dextrose + 0.18% sodium chloride is inappropriate, potentially dangerous and should be avoided.

Burns (see also Chapter 15)

Significant burns cause large fluid losses and burns patients require large volumes of fluid resuscitation. In addition to normal maintenance fluids, such patients need resuscitation fluid administered as Normal saline, Ringer's solution or Gelofusine* given over at least the first 36 h after injury. The 36 h are divided into six periods: three of 4 h, two of 6 h and one of 12 h. The timing starts from the moment of injury so that the first infusion is inevitably delayed. During each period the child needs an average of 0.5 ml kg^{-1} per % burn. The precise volume given is adjusted on the basis of urine output, urine and plasma osmolality, perfusion and the calculated plasma deficit. This can be calculated from the formula:

$$\text{plasma deficit} = \text{blood volume} - \left(\text{blood volume} \times \frac{\text{normal haematocrit}}{\text{observed haematocrit}}\right)$$

Deep burns result in red cell destruction and the usual blood requirement is of 1% of normal blood volume per 1% burn for deep burns of more than 10% surface area. Because the haematocrit is a useful guide to the plasma deficit, blood is usually best administered during the last 12 h of fluid resuscitation.[20]

Clinical assessment of dehydration

Although thirst appears with the loss of approximately 2% of total body water, the state of the peripheral circulation is the most sensitive guide to more serious levels of clinical dehydration in children. Core–peripheral temperature difference becomes clinically detectable and mucous membranes dry at around 5% loss of total body water. With 10% dehydration the peripheries are cold and capillary refill, normally complete within 2 s, is delayed. Pulse and respiratory rates increase, consciousness may be clouded, and in the neonate the fontanelle is sunken. Blood pressure may fall, although because of the increased cardiac output caused by the tachycardia this is not an early or reliable sign. Urine output is decreased. At 15% dehydration capillary refill may be incomplete even after 10 s, the mouth is parched and the eyes are sunken. The pulse is rapid and thready and blood pressure low. The child is stuporose and oliguric, and may show signs of respiratory distress. Losses in excess of 20% may be fatal.

COLLOIDS

THE CRYSTALLOID VERSUS COLLOID DEBATE

The superiority of colloid over crystalloid for volume replacement remains controversial.[21,22] Whilst crystalloids are generally more popular in the USA, colloids are preferred in Europe.[23] The debate centres on which fluid space needs replenishing and the importance or otherwise of colloid osmotic pressure.

Colloids theoretically remain within the intravascular space, therefore expanding the intravascular volume more efficiently, producing the same increase in cardiac output for a smaller volume of fluid. The proponents of crystalloid argue that the whole extracellular fluid space is reduced in hypovolaemia because of fluid movement from the interstitial compart-

undergone a circulating volume transfusion of red cells. In babies and infants, a transfusion of 5–10 ml kg^{-1} is usually sufficient, whilst unit transfusions are appropriate in older patients.

FRESH-FROZEN PLASMA AND CRYOPRECIPITATE

FFP contains 1 unit of factor activity per millilitre of plasma. A decision to use FFP or cryoprecipitate should be based on a combination of clinical and laboratory findings. An INT of less than 1.4 or a partial thromboplastin time of less than 60 s is unlikely to cause significant bleeding problems and correction is not required. Laboratory values greater than these levels or significant bleeding will require correction. An empiric dose of 5–10 ml kg^{-1} is usually adequate or the dose can be calculated by body weight, plasma volume and desired increment of clotting factors.

Cryoprecipitate is a poor source of factors II, V, IX, X, XI and XII but contains factors VIII : C, VIII:vWF, XIII, fibrinogen and fibronectin. Indications for its use include haemophilia A, von Willebrand disease, fibrinogen deficiency, massive transfusion and uraemic platelet dysfunction. Its advantage over FFP is that it is concentrated and one bag (of 15–20 ml) is the dose per 10 kg body weight.

SPECIAL SITUATIONS

Jehovah's witnesses

The Royal College of Surgeons of England have produced a code of practice for the surgical management of Jehovah's Witnesses. This acknowledges that the children of Jehovah's Witnesses requiring blood transfusion present a most difficult management problem.[84] There are some mitigating factors, however. Either parent may sign a consent form permitting a transfusion. Most operations on children do not require or involve blood transfusion, but it is unethical to let a child die for want of a blood transfusion. The surgeon and anaesthetist must, however, respect the beliefs of the family and should make every effort to avoid the perioperative use of blood or blood products. For children under 13 years of age who require or may require a transfusion but whose parents refuse to give consent, legal advice should be sought. Such children will normally be made a temporary ward of court. This subject is covered more fully in Chapter 2.

Sickle cell disease

Traditionally, patients with sickle cell disease have been routinely transfused before elective surgery. There has, however, been little consensus as to whether simple correction of the anaemia is sufficient or whether the level of HbS should be reduced to less than 30%. Vichinsky *et al.* compared a conservative regimen (transfusing to a haemoglobin level of around 10 g dl^{-1}) with an aggressive regimen (haemaglobin of around 10 g dl^{-1} and an HbS level of less than 30%). They found the conservative regimen to be as effective in preventing perioperative complications and this group had half as many transfusion-associated complications.[85] Similarly, immediately preoperative transfusion to a haematocrit of more than 36% was as efficacious as two-volume exchanges beginning 2 weeks prior to surgery, with less disruption to the family.[86] Patients with HbSS disease should receive transfusions to correct their anaemia. They should be given adequate perioperative hydration with crystalloid. Postoperatively, they should receive adequate analgesia in addition to oxygen and physiotherapy to prevent atelectasis. For more information, see Chapter 1.

CONCLUSION

Fluid management in paediatrics is an art as well as a science: clinicians need to monitor the response to therapy and change the regimen appropriately. This chapter contains guidelines and suggestions for safe fluid administration but cannot replace clinical experience.

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Editorial
Postoperative hyponatraemic encephalopathy following elective surgery in children

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Introduction

In the United States, there are an estimated 15 000 deaths per year as a consequence of postoperative hyponatraemia (1) (Figure 1). There have been a number of recent studies which have described postoperative hyponatraemic encephalopathy with death or permanent brain damage (2–6). From these studies, it appears that brain damage associated with postoperative hyponatraemic encephalopathy primarily affects menstruant women (1) and prepubertal children (6).

Postoperative hyponatraemic encephalopathy in prepubertal children

There are multiple reports of prepubertal children suffering brain damage from postoperative hyponatraemic encephalopathy (6–9). The aetiology of the hyponatraemia usually involves a combination of: a) intravenous hyponatraemic fluids; b) elevated plasma antidiuretic hormone (ADH); c) respiratory insufficiency secondary to hyponatraemic encephalopathy. It has been demonstrated in several series that plasma levels of ADH (vasopressin, antidiuretic hormone) are elevated in virtually every postoperative child (7,10–13). If such patients are given intravenous free water (any solution with a sodium concentration below 140 mmol·l^{-1}), there will always be a tendency towards postoperative hyponatraemia (14). When compared with other groups, prepubertal children are far more susceptible to brain damage from hyponatraemia than are adults (6), and recent experimental evidence demonstrates why this may be the case.

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Effects of hyponatraemia on the paediatric central nervous system

Nattie & Edwards (15) studied the effects of acute hyponatraemia on the brain of puppies. They found that acute lowering of plasma sodium from 140 to 120 mmol·l^{-1} resulted in severe hypoxaemia (arterial PO_2 fell from 11.4–6.9 kPa (88 to 53 mmHg)) and cerebral oedema. In contrast to adults, the brains of paediatric animals (three day old puppies and neonatal rats) were unable to adapt to hypo-osmotic stress by extrusion of cation (15,16).

Adaptation of the brain to hyponatraemia occurs as a consequence of the following sequence of events. First, hyponatraemia leads to a movement of water into brain cells as a result of osmotic forces. In addition, vasopressin which is usually elevated in the plasma of hyponatraemic patients [7] may lead to a direct movement of water into brain cells independent of the effects of hyponatraemia (15). The early response of the brain to hyponatraemia-mediated oedema is the loss of bone and cerebrospinal fluid, followed by extrusion of sodium from brain cells by several pathways (19). Loss of potassium and possibly organic osmolytes follows later, in an attempt to decrease brain cell osmolality without a gain of water (20).

Effects of hormones and physical factors on brain adaptation to hyponatraemia

There is a significantly higher intracellular brain water content in prepubertal rats in comparison with adult rats, suggesting that the brain occupies a greater percent of the available intracranial volume in young rats (16). Such physical factors may be important determinants of outcome in hyponatraemic rats. As individuals age, there is a progressive decline in the volume of brain, while skull size remains constant in adult life (21). Thus, elderly individuals of both

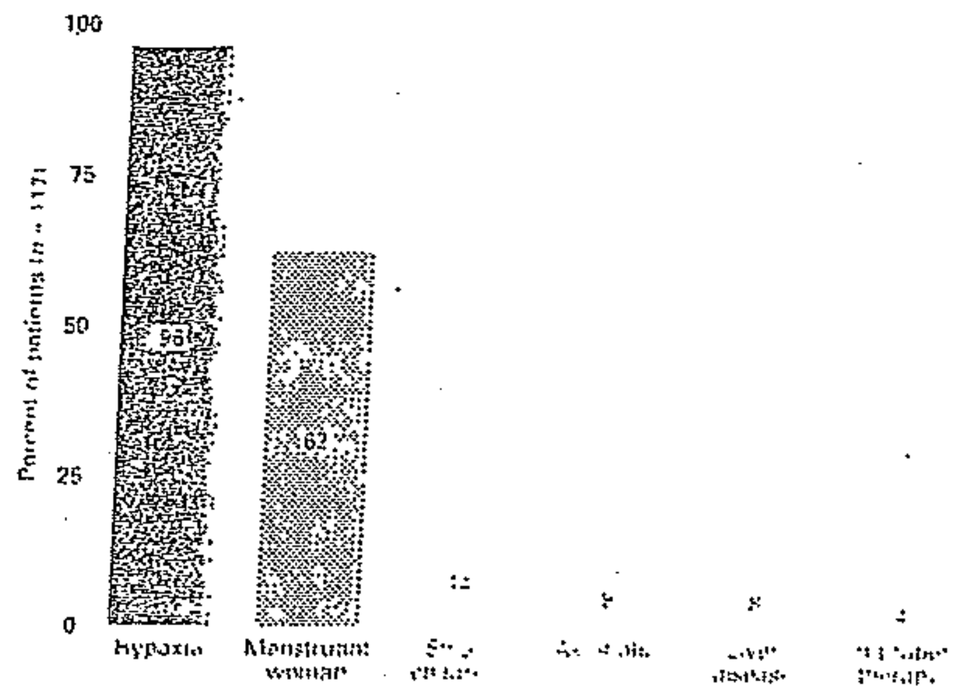

Figure 1
In nine published series from our laboratory comprising 84[illegible] hospitalized patients with postoperative hyponatraemia, [illegible] (158/847) developed hyponatraemic encephalopathy and 117 developed permanent brain damage or died. The major risk factors associated with permanent brain damage or death in these 117 patients with hyponatraemic encephalopathy are shown. Most patients (96%) suffered an hypoxic episode because of failure to initiate active therapy in a timely manner. In 4% of patients suffering permanent brain damage, improper therapy for hyponatraemia was implicated in the outcome.

genders have more room in the rigid skull for the brain to expand than do younger ones. This finding is more marked in males (21).

If adaptation of the brain is not adequate, pressure of the swollen brain on the rigid skull leads to a decrease in cerebral blood flow (22) and cerebrospinal fluid production (23). If the ability of the brain to adapt is impaired, there will be increasing oedema, with eventual tentorial herniation and secondary cerebral ischaemia (24). This often leads to respiratory insufficiency (4), with reduced delivery of oxygen to brain because of the further decrease of cerebral blood flow, thereby exacerbating the existing cerebral ischaemia (22).

Sex steroid and certain neuropeptide hormones may influence brain adaptation to hyponatraemia. Male rabbits and cats are more efficient than females in extruding sodium to decrease brain cell osmolality during hyponatraemia, resulting in significantly less brain swelling in male than in female hyponatraemic animals (16,25). Oestrogens have also been reported to stimulate, and androgens to suppress, vasopressin release (26,27). Virtually all hyponatraemic patients have increased plasma levels of vasopressin (17,28), a neuropeptide which may exert multiple potentially deleterious cerebral effects. In normonatraemic animals vasopressin results in water accumulation in the brain (18), a significant decline in brain synthesis of ATP (29), and a decline of brain pH (29,30). Vasopressin also impairs the function of several important adaptive pathways to hyponatraemia (31,32).

Recent studies have demonstrated that the brains of prepubertal rats are unable to adapt to hyponatraemia (16). The greater mortality with hyponatraemia in prepubertal rats is associated with a greater accumulation of water in the intracellular space of the brain than in rats belonging to other age groups, as well as an inability of the prepubertal brain to extrude sodium from brain cells. The baseline intracellular sodium content in the prepubertal rats was greater by almost 50% than in control adult rats, a finding consistent with previous studies in newborn dogs (15,33).

Biochemical differences in paediatric vs adult brain with hyponatraemia

There are several possible reasons for the increased brain intracellular sodium in prepubertal rats. The Na^+-K^+ ATPase system appears to be the major early adaptive pathway for extrusion of sodium from brain cells during hyponatraemia (19,34) and its impairment results in decreased ability to pump sodium out of the brain. In prepubertal rats, the brain Na^+-K^+ ATPase activity is significantly lower than that observed in adults, both *in vitro* (35) and *in vivo* (36). Coupled with the higher brain sodium, these differences may reflect a limited ability to pump sodium out of the prepubertal brain. The increased intracellular sodium content may be a consequence of limited cerebral Na^+-K^+ ATPase function in young rats compared to adults. The decreased cerebral Na^+-K^+ ATPase activity may be responsible for the impaired adaptation to hyponatraemia in prepubertal rats. Testosterone stimulates Na^+-K^+ ATPase activity in rat brain (37,38). Pretreatment of prepubertal rats with testosterone resulted in a significant decrease in the brain intracellular content of both sodium and water while also reducing the mortality associated with acute hyponatraemia from 84% to zero (16).

Clinical effects of hyponatraemia in children vs adults

If one can extrapolate the above experimental findings to paediatric patients, then the implications would be that children are more susceptible to brain damage from postoperative hyponatraemia than are adults. The reasons include: a) decreased available

room for swelling of the paediatric brain in the rigid skull, leading to a propensity for brain herniation with what might appear to be a small decrement of plasma sodium (39); b) impaired ability of the paediatric brain to adapt to hyponatraemia when compared with adults (15,37); c) severe systemic hypoxaemia secondary to respiratory insufficiency frequently occurs in children with only modest hyponatraemia (6,15,39). The respiratory insufficiency is a consequence of increased intracranial pressure (3).

Gomola *et al.* have described a prepubertal (10 years old) female child with middle face hypoplasia who underwent elective maxillary reconstruction (40). The surgery went well and postoperatively, she was given primarily free water intravenously (280 mM glucose in 51 mM NaCl) at a rate of 2 l per [illegible]. The child weighed 30 kg with estimated total [illegible] water of 18.5 l. On the first postoperative day, the child became confused and developed headache and vomiting. Renal function was apparently normal on the basis of normal plasma urea and creatinine. The plasma sodium was found to be 117 mmol·l^{-1}. She was initially treated with sodium supplementation, but on the second post-operative day, the plasma sodium was still low at 120 mmol·l^{-1}. The urine and plasma osmolalities were 342 and 255 mOsm·kg^{-1}. An MRI of the brain was normal.

The authors proposed three possible explanations for the hyponatraemia: a) dilutional hyponatraemia secondary to IV hypotonic fluid; b) pituitary insufficiency; c) inappropriate secretion of ADH. Pituitary insufficiency was ruled out by normal values for ACTH, cortisol, thyroid hormone and growth hormone. The ADH was 4 to 5 pg·ml^{-1}, which is 'normal' but inappropriately high for the [illegible]cellular hypoosmolality (41) and is essentially a [illegible]rsal finding in both paediatric and adult postoperative patients (7–13). The child received 2 l per day of hypotonic IV fluid in the presence of elevated plasma ADH. Although neither initial plasma sodium, urine output or total volume of IV fluids are provided, given the child's weight and rate of infusion, the plasma sodium of 117 mmol·l^{-1} appears very likely to have been the consequence of retention of about 3 l of IV hypotonic fluid over two days (6). The expression inappropriate secretion of ADS (SIADH) was originally used for elevated plasma ADH related to lung cancer (42) and has become a catch all term for virtually any patient with elevated plasma ADH. In particular, postoperative patients as well as those with heart failure or hepatic cirrhosis have elevated plasma ADH levels but are functionally hypovolaemic as well (41). Postoperative subjects are functionally hypovolaemic, so that the term SIADH may not be appropriate in this patient (11). There is also a perception that ADH, and by association SIADH, can somehow lower the plasma sodium. Although ADH leads to increased retention of ingested or infused water, in the absence of increased water intake, ADH by itself will have no effect upon the plasma sodium. Thus, the most likely explanation for the hyponatraemia in this patient is infusion of hypotonic fluid (51 mM NaCl/280 mM glucose) in the presence of the expected postoperative increase in plasma ADH. Adrenal insufficiency is ruled out by the normal plasma cortisol and the fact that she remained normal for six months without any steroid replacement therapy. Exactly why the plasma sodium rose following IV hydrocortisone is uncertain, but may have been related to the expected decline of ADH values to normal after four to five postoperative days. Pituitary insufficiency is ruled out by normal values for ACTH, IGF1 and growth hormone.

Symptomatic postoperative hyponatraemia carries a mortality of at least 15% (43), particularly in children and respiratory arrest is a frequent occurrence, but once this complication occurs, the morbidity is substantial (6,7). There is no obvious rationale for the administration of hypotonic fluid to a postoperative patient, unless the individual is hypernatraemic (14). If the patient becomes symptomatic, therapy with hypertonic NaCl is indicated (39). The syndrome can be prevented by administration of primarily isotonic fluids to postoperative patients.

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37 Guerra M, del Castillo AR, Battaner E *et al.* Androgens stimulate preoptic area Na^+, K^+ ATPase activity in male rats. *Neurosci Lett* 1987; 78: 97–100.
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41 Schrier RW. Pathogenesis of sodium and water retention in high-output and low-output cardiac failure, nephrotic syndrome, cirrhosis, and pregnancy. Parts 1 and 2. *New Engl J Med* 1988; 319(16): 1065–1072 and 1127–1134.
42 Bartter FE, Schwartz WB. The syndrome of inappropriate secretion of antidiuretic hormone. *Am J Med* 1967; 42: 790–806.
43 Fraser CL, Arieff AI. Epidemiology, pathophysiology and management of hyponatremic encephalopathy. *Am J Med* 1997; 102: 67–77.

Accepted 4 June 1997

CURRICULUM VITAE

OF

EDWARD SUMNER, MA BM BCh FRCA

May 2001

NAME: Edward Sumner

ADDRESS: [redacted]

DATE OF BIRTH: [redacted]

MARITAL STATUS: [redacted]

DEGREES HELD:

MA, BM, BCH	Oxford 1966
FRCA	London 1971

ACADEMIC DISTINCTIONS:

First Class Honours Degree
Animal Physiology
Oxford 1963

Nuffield Prize
Primary Fellowship
London 1969

DATE OF MEDICAL REGISTRATION: March 1968

PRESENT APPOINTMENT:

Consultant Anaesthetist - Great Ormond Street Hospital for Children NHS Trust
Great Ormond Street
London WC1N 3JH

2 311

RF - PSNI

098-043e-170

Appointed September 1973

Director of the Department of Anaesthetics 1987 - 1992

Director: Cardiac Intensive Care Unit 1988 - 1993

Honorary Senior Lecturer : University of London

I have six operating lists each week with cardiac and general paediatric surgery and I am involved in all aspects of paediatric anaesthesia. Approximately 12,000 anaesthetics per year are administered at Great Ormond Street Hospital for Children. The respiratory support service for the whole hospital developed under my supervision.

I am involved in the training of 40 new residents each year, plus at least 12 seconded residents.

PREVIOUS APPOINTMENTS:

House Officer Medicine and Surgery	University College Hospital London	Jan 1967-Feb 1968
S.H.O. Anaesthetics	University College Hospital London	Mar 1968-Feb 1969
Registrar Anaesthetics	University College Hospital London	Mar 1969-Jan 1971
Senior Registrar Anaesthetics	St Thomas' Hospital London Including 6 monthly rotations to National Hospital for Nervous Diseases, London, and National Heart Hospital, London.	Nov 1971-Sep 1973

After my consultant appointment, I was seconded on a part-time basis to the Nuffield Research Department, Royal College of Surgeons, London, for research experience with Mass Spectrometry and its use for inhaled and expired gases (a prototype breathalyser) for the year 1974.

OFFICES HELD I have been elected to the Executive of the Association of Paediatric Anaesthetists of Great Britain and Ireland, Representing England and am chairman of the Scientific Subcommittee.

President Association of Paediatric Anaesthetists of Great Britain and Ireland

President Elect Federation of European Associations of Paediatric Anaesthesia

TEACHING EXPERIENCE:

Teaching in the Institute of Child Health, London:

Annual Advanced Course in Paediatrics

Annual Course in Paediatric Intensive Care

Twice Annual Final Fellowship Course (part of the London Hospitals' Course)

Annual St Bartholomew's Hospital Final Fellowship Course.

British Council Course, Paediatric Cardiac Surgery - Teaching Respiratory Support - 1978, 79, 80, 81, 83, 84, 85, 86, 87, 90 and 92.

Courses in Paediatric Anaesthesia to Danish Anaesthetists and Norwegian Anaesthetists - 1980, 1982, 1990 and 1995.

Presented papers at the Association of Paediatric Anaesthetists:

London 1974 : Mass Spectrometry - Clinical Applications.

Newcastle 1976 : Wilson-Mikity Syndrome

Edinburgh 1982 : Congenital Diaphragmatic Hernia

Dublin 1985 : Management of Phrenic Palsy in the Infant.

London 1988 : Analgesia for Newborns

Royal College of Anaesthetists Final Fellowship Course 1974, 78, 80, 84, 88, 89 - 1997.

Continuing Medical Education Day - 1991, 1993, 1994

1982 European Congress, London.

Paper - Congenital Diaphragmatic Hernia
Poster - Caudal Analgesia

Royal Society of Medicine

1982 - Congenital Diaphragmatic Hernia
1982 - Paediatric Anaesthesia Symposium

1980 Stockholm, Sweden : Paediatric mechanical ventilation.

1980-1984 Liege, Belgium - 5 visits. Paediatric cardiac anaesthesia.

1982 Bonn, Germany - Cardiac anaesthesia.

Visiting Professor, Sydney, Australia, Royal Alexandra Children's Hospital - December 1981.

Lecturer to the British Council Sponsored Workshop in Neonatal Surgery and Intensive Care: Delhi - March 1983 : Jaipur - 1984.

Prague 1983 - Congenital Diaphragmatic Hernia.

Royal College of Surgeons, London: Symposia:

1983 Paediatric Anaesthesia
1984 Neonatal Emergencies
1985 Paediatric Anaesthesia
1987 Paediatric Anaesthesia in the District Hospital

Paris 1983 - Congenital Diaphragmatic Hernia.

Manila, Phillipines 1984 - Total Intravenous Anaesthesia in Paediatrics. World Congress.

Lectures at Intensive Care Meetings:

Birmingham 1984
Rotterdam 1984
Tubingen 1984

Lectured at Thoracic Anaesthetic Meeting, London 1983, 1984.

Open Heart Surgery Congress - Bombay 1985.

Pulmonary Hypertensive Crisis - Paediatric Intensive Care.
Brussels 1985.

British Council Lecturer in Paediatric Anaesthesia - Kathmandu, Nepal : 1986, 1987.

Association of Anaesthetists, London:

1986 - Neonatal Analgesia
1987 - Cyclopropane

Neonatal Anaesthesia - Gothenburg, Sweden : 1986

Neonatal Anaesthesia - Oslo, Norway : 1986.

Paediatric Anaesthesia, Basel, Switzerland : 1987, 1994, 1996.
Cardiac Anaesthesia, Lucerne, Switzerland: 2000

Lectures:

Barbican - ICU Update
September 1991
: Respiratory Support in Paediatrics

Oporto
September 1991
: Pulmonary hypertension
: Cardiothoracic anaesthesia for children.

Coimbra
May 1992, May 1994, May 2000
: Neonatal topics
: Cardio-respiratory physiology
: Renal physiology
: Fluid management
: Pain management

3rd World Congress of Paediatric Anaesthesia
Amsterdam
June 1992
: Transplantation: Are children different?

Munich
July 1992
: Neonatal anaesthesia

Royal College of Anaesthetists
Fellowship Course
1992

315

: Respiratory support in paediatrics

1992 : Knights of Malta Lecturer, University of Bologna (Italy)

1994-99 : Invited speaker, Munich, Mannheim, Amsterdam, Brussels,
Bergamo, Florence, Jerusalem, Brussels, Roumania, Paris, Estonia, Wiesbaden

1996 Invited speaker - World Congress of Anaesthesiology, Sydney, Australia.

1999 Invited speaker - ASEAN Congress, Kuala Lumpur
Invited speaker - Association of Anaesthetist, London
Invited speaker - Japanese Society Annual Meeting, Mito
Jackson Rees Lecturer, Erasmus University, Rotterdam October
2000 Invited speaker, European Congress, Vienna
Biannual Paediatric Anaesthesia Weekend, Coimbra, Portugal
World Paediatric Anaesthesia Meeting, Halifax, Nova Scotia
2001 Invited Speaker European Congress of Paediatric Anaesthesia
Helsinki
2002 Delivered the first Prof Appukutty Memorial Oration Chennai, India Jan 19th
"Neonatal Anaesthesia: then and now".

PUBLICATIONS:

PAPERS:

Quinsy tonsillectomy: A safe procedure. Sumner E (1973) *Anaesthesia* 28: 558.

Porphyria in relation to surgery and anaesthesia. Sumner E (1975) *Annals of the Royal College* of *Surgeons, England,* 56: 81.

The use of tolazoline in congenital diaphragmatic hernia. Sumner E and Frank DJ (1981) *Archives of Disease in Childhood* 56:350.

Congenital diaphragmatic hernia: improved prognosis. An experience of 62 cases over 2 years. Marshall A and Sumner E (1982) *Journal of the Royal Society of Medicine* 75: 607.

Late perforation by central venous cannulae. Henderson A and Sumner E (1984) *Archives* of *Disease in Childhood* 59: 776.

Tracheal perforation in newborns. Macleod B and Sumner E (1987) *Anaesthesia* 41, 67.

Prune Belly Syndrome - anaesthetic hazards. Vallis C, Henderson A and Sumner E (1987) *Anaesthesia* 42: 54.

Fatal intraoperative tumor embolus in a child with hepatoblastoma. Dormon F, Sumner E and Spitz L (1985) *Anesthesiology* 63: 692.

Halothane hepatitis in a baby. Whitburn R and Sumner E (1986) Anaesthesia 41: 611.

The use of opioids in neonates. A retrospective study of 933 cases. Purcell-Jones G, Dorman F and Sumner E (1987) *Anaesthesia,* 42: 1316.

The use of opioids in neonates. A survey. Dorman F, Purcell-Jones G and Sumner E (1988) Pain
Pain
Macleod B and Sumner E (1987) Neonatal tracheal perforation. *Anaesthesia* 41: 67-70.

Braude N, Ridley SA, Sumner E. Parents and paediatric Anaesthesia. A prospective survey of parental attitudes to their presence at induction. *Ann R Coll Surg Eng* 1990; 72: 41-4

Creagh-Barry P and Sumner E (1992) Neuroblastoma and anaesthesia. *Paediatric Anaesthesia* 2: 147-153.

Sumner E (1993) Gas exchange in children. *Paediatric Anaesthesia* 3: 1-3.

Sumner E (1994) Paediatric Anaesthesia. *Paediatric Anaesthesia* 4: 1-2.

GR Lauder, E Sumner (1995) Larsen's Syndrome: anaesthetic implications. *Paediatric Anaesthesia* 5, 133-138.

J de Lima, AR Lloyd- Thomas, RF Howard, E Sumner (1996) Infant and Neonatal pain: anaesthetists' perceptions and prescribing patterns. *BMJ* 313 787-8.

J Challens, E Facer, E Sumner. Epidural anaesthesia and Dysautonomia (Riley Day Syndrome) *Paed Anaesth* 1999

CHAPTERS:

Anaesthesia for the older child. Kaufman L and Sumner E (1980) In General Anaesthesia, Ed Gray TC, Nunn JF and Utting JE. 4th Edition. London, Butterworth.

The paediatric patient. Sumner E and Patrick EK (1980) In Preparation for Anaesthesia. Ed Stevens AJ. Tunbridge Wells, Pitman Medical.

Paediatric anaesthesia and intensive care. Sumner E. In Anaesthesia Reviews. Ed Kaufman L. London, Churchill Livingstone.

One 1982
Two 1983
Four 1987

Paediatric Anaesthesia. Sumner E (1984) In Practice of Anaesthesia. Ed Wylie D and Churchill-Davidson CD. London, Lloyd-Luke.

Artificial ventilation of children. Sumner E. In Diagnosis and Management of Paediatric Respiratory Disease. Ed Dinwiddie R. London, Churchill Livingstone. 1989.

Respiratory care in paediatrics. Sumner E (1984) In Anaesthesia and Patient Care. Ed Anis and Salim, Pakistan.

Paediatric Anaesthesia. Sumner E (1988) In Operative Surgery-Paediatric Surgery. Ed Spitz L. London, Butterworth.

Preparation for Anaesthesia: the paediatric patient. Sumner E and Facer EK (1986) Ed Stevens J. Preparation for Anaesthesia. Clinics in Anaesthesiology. London, Saunders. Vol 4.

Unusual Paediatric Conditions. Sumner E and Facer EK (1986) Ed Stevens J. Preparation for Anaesthesia. Clinics in Anaesthesiology. Vol 4.

Postoperative care in surgery for congenital heart disease. Eds Stark and de Leval. Philadelphia, Saunders. 1994.

Congenital Heart Disease. Sumner E and Cullen S. In: Medicine for Anaesthetists. Ed. Prof Vickers. Oxford, Blackwell. 1999

Anaesthesia for children with congenital heart disease for noncardiac surgery. In: Anaesthesia for the New Millenium. Ed Wong A ISBN 983-808-072-1 1999

BOOKS:

Medical Problems and the Anaesthetist. Kaufman L and Sumner E (1980) London, Arnold.

Neonatal Anaesthesia. Hatch DJ and Sumner E (1981) London, Arnold.

Paediatric Anaesthesia. Ed Sumner E and Hatch DJ Clinics in Anaesthesiology ( )1985 vol 3)

Neonatal Anaesthesia and Perioperative Care. Hatch DJ and Sumner E (1986) 2nd Edition. London, Arnold.

A Textbook of Paediatric Anaesthetic Practice. Sumner E and Hatch DJ. London, Bailliere-Tindall.

The Surgical Neonate: Anaesthesia and Intensive Care. London, Arnold. Hatch DJ, Sumner E and Hellman J (1995)

Paediatric Anaesthesia. Sumner E and Hatch DJ 1999 London, Arnold

In preparation:

The Respiratory System. Sumner E. In Clinical Paediatric Anatomy. Ed Dickson JSR. Oxford, Blackwells. In press.

Other Publications:

"Blood". An Essay. The Blood Show. Five Mile Gallery, Underwood Street, London N1

EDITORIAL DUTIES:

Editor-in-Chief: Paediatric Anaesthesia. Blackwell Science. An International Journal.

LEARNED SOCIETIES:

Association of Anaesthetists of the United Kingdom.

Association of Paediatric Anaesthetists of the United Kingdom.

European Neonatal and Paediatric Intensive Care Society.

European Society of Anaesthesiology.

Society for Pediatric Anesthesia

RESEARCH INTERESTS:

I started the first Paediatric Acute Pain Service in the UK in 1990.

Popularised - epidural analgesia in infants and children.

- axillary artery cannulation in infants.

Projects have included - the pulmonary circulation

- gastro oesophageal reflux.

MEDICO-LEGAL WORK:

On the Panel of the Association for Victims of Medical Accidents.

Expert for the Bristol Royal Infirmary Public Inquiry 1999/2000

HER MAJESTY'S CORONER

DISTRICT OF GREATER BELFAST

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Dr E Sumner MA, BM, BCh, FRCA
Consultant Paediatric Anaesthetist

[redacted]

10th February 2003

Dear Ed,

RAYCHEL FERGUSON, DECEASED

I am writing to advise you that the inquest has now concluded. Once again I am most grateful to you for all your help and assistance. Your report was invaluable and without it I really would have been at a loss.

I am writing to the Chief Medical Officer of Northern Ireland, Dr Henrietta Campbell, suggesting that she draws the protocol to the attention of the Chief Medical Officers of England and Wales, Scotland and the Republic of Ireland. Dr Fulton and Dr Nesbitt from Altnagelvin Hospital expressed the view that the protocol was not prescriptive enough and that it should in fact apply to all patients rather than just children. It may be that the Working Party will be re-convened to consider these views.

Once again my very sincere thanks. Also, I am sorry that we were not able to have dinner on Wednesday evening. I know you appreciated the fact that as the inquest was ongoing it might have been regarded as inappropriate for you and me to have dined together. Hopefully, we will have another opportunity.

[redacted]

With my very best wishes.

Yours sincerely

RF - PSNI

J L LECKEY
HM CORONER FOR GREATER BELFAST

098-043e-180 - 416