Witness Statement Ref. No. 120/1

NAME OF CHILD: Adam Strain

Name: John Alexander

Title: Doctor

Present position and institution:

Retired

Previous position(s) and institution(s):

Consultant Anaesthetist and Physician in charge of the Intensive Care Unit, Belfast City Hospital

Membership of Advisory Panels and Committees:

Previous Statements, Depositions and Reports:

N/A

OFFICIAL USE:
List of reports attached:

Ref:	Date:	

Particular areas of interest:

I have little to add to the very comprehensive report written by Dr Summes. I noticed, looking through the very thorough notes, that on several occasions when Adam was brought to theatre, a central vein catheter was recorded as being in situ. We don't know for what purpose, or for how long, these catheters were in position. Our own experience in adult intensive care was that re-insertion of central vein catheters at a later date could prove to be extremely difficult, and in Adams case, the triple-lumen catheter was in an incorrect position and the CVP measurements incorrect. In the past 15 years ultrasound-guided location of central veins has been popularised, and indeed recommended.

Dr Taylor maintained that Adam could pass an unlimited amount of fluid in his urine, but no allowance was made for the possible effects of anaesthesia and controlled ventilation. I dont understand a distinction between 'dilutional hyponatraemia' and 'hyponatraemia'.

The recognition of the possibility of hyponatraemia has become more common in the past 15 years. A paper 'Management of Hyponatraemia' was published recently in the British Journal of Hospital Medicine under the Modernising Medical Careers Supplement which is aimed at doctors working in the Foundation Years (copy attached).

Finally, to interview a doctor who has striven to provide a service under very difficult circumstances under caution in relation to "manslaughter by gross negligence" is beyond my comprehension.

THIS STATEMENT IS TRUE TO THE BEST OF MY KNOWLEDGE AND BELIEF

Signed: J Alexander Dated: 10 04 11

Management of hyponatraemia

Introduction

Hyponatraemia (serum sodium level <135 mmol/litre) is the most common electrolyte abnormality among hospitalized patients. A prevalence rate as high as 15–30% has been reported among patients admitted to acute and intensive care units (Hoorn et al, 2004; Jaber et al, 2006). Evidence suggests an increase in mortality associated with even a mild degree of hyponatraemia (Waikar et al, 2009). Besides its significance as a potential cause of morbidity and mortality, hyponatraemia could also serve as a useful indicator for undiagnosed underlying pathology such as endocrine disorders or malignancy.

A systematic approach towards the clinical assessment and interpretation of biochemical abnormalities is vital to facilitate the diagnosis and management of hyponatraemia. The optimal treatment of hyponatraemia should take into account its severity, duration and mode of clinical presentation. Overzealous correction could result in irreversible neurological complications.

Pathophysiology

Understanding the normal physiological mechanisms for maintaining eunatraemia (serum sodium 135–145 mmol/litre) is crucial for correctly identifying the cause of hyponatraemia. Serum sodium level and osmolality are both normally tightly regulated by three major mechanisms: thirst, antidiuretic hormone and the angiotensin–renin system. An increase in serum osmolality stimulates the hypothalamic osmoreceptors resulting in the thirst sensation as well as promoting secretion of antidiuretic hormone from the neurohypophysis. Antidiuretic hormone release can also occur in response to volume depletion through activation of the baroreceptors in the carotid sinus. Activation of the renin–angiotensin system in response to volume depletion promotes release of aldosterone which regulates serum sodium level by increasing its reabsorption in exchange for potassium in the distal tubule. Most causes of spontaneous (non-iatrogenic) hyponatraemia can be traced back to a defect in one or more of the above mechanisms.

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Causes of hyponatraemia

The most important step in diagnosing the cause of hyponatraemia is assessing the volume status of the patient. The causes of hyponatraemia can be classified into three categories based on the volume status of the patient: hypovolaemic, euvolaemic and hypervolaemic (*Figure 1*). Hypovolaemic hyponatraemia is characterized by depletion of both total body sodium and water while euvolaemic hyponatraemia is characterized by normal total body sodium and normal or increased total body water. Hypervolaemic hyponatraemia involves both increased total body water and sodium levels.

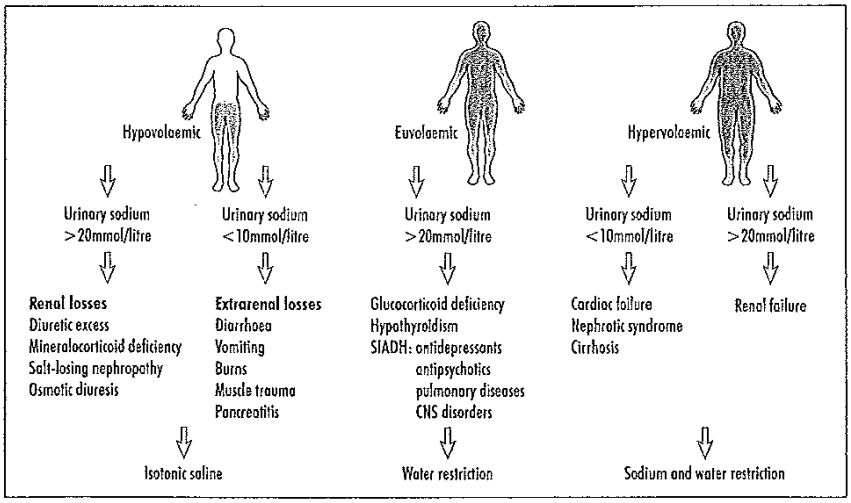
Hypovolaemic hyponatraemia is caused by excess loss of sodium from the kidneys (diuretics, osmotic diuresis and aldosterone deficiency) or by non-renal losses such as from the gastrointestinal tract (vomiting and diarrhoea), the skin (excessive sweating or burns) or third space loss (surgical cases such as small bowel obstruction and pancreatitis). The distinction between renal and non-renal loss of sodium could be obvious from the history but measurement of urinary sodium excretion may further simplify differentiation (>20 mmol/litre in renal causes). The fractional excretion of sodium (FENa), defined as the percentage of sodium filtered by the kidney which is excreted in the urine, is a more reliable measure to differentiate renal from non-renal sodium loss than urinary sodium concentration alone. It is calculated by the formula:

$$\text{FENa} = 100 \times \frac{\text{urinary sodium} \times \text{serum creatinine}}{\text{serum sodium} \times \text{urinary creatinine}}$$

A low FENa (<1%) indicates the presence of a hypovolaemic stimulus to the kidneys to conserve sodium (non-renal loss) while confirming satisfactory renal tubular resorptive function. A high FENa (>3%) indicates sodium wasting secondary to an intrinsic renal pathology or cerebral salt wasting. The use of diuretics increases the FENa and this should be ruled out to avoid misdiagnosis of the cause of hyponatraemia.

Euvolaemic hyponatraemia is by and large secondary to the syndrome of inappropriate antidiuretic hormone secretion (SIADH) but other causes such as psychogenic polydipsia, glucocorticoid deficiency and hypothyroidism should also be considered. The causes and criteria for diagnosis of SIADH are discussed separately.

Figure 1. Classification of causes of hyponatraemia. SIADH = syndrome of inappropriate antidiuretic hormone secretion.



Hypervolaemic hyponatraemia is encountered in volume overload states such as congestive cardiac failure, nephrotic syndrome, chronic renal failure and cirrhosis.

Symptoms of hyponatraemia

The nature and severity of symptoms of hyponatraemia depends on the level of hyponatraemia and the rate at which the serum sodium level dropped below the normal range. Common symptoms reported with mild to moderate hyponatraemia (serum sodium <135 mmol/litre and <130 mmol/litre respectively) include headache, lethargy, anorexia, nausea, dysguesia, muscle cramps, drowsiness and confusion. Severe hyponatraemia can result in hallucination, seizures, coma, respiratory arrest and death (Adrogue and Madias, 2000).

Patients with acute hyponatraemia are more prone to develop profound symptoms and cerebral oedema. In contrast, patients with chronic hyponatraemia have less pronounced symptoms because of adaptive mechanisms in the brain that mitigate the degree of cerebral oedema. The normal brain tissue adapts to hyponatraemia via extrusion of sodium and organic osmolytes to the extracellular compartment to prevent cellular oedema. However, this protective adaptive mechanism usually takes several hours to begin which accounts for the severity of symptoms of acute hyponatraemia and the associated risk of cerebral oedema (Strange, 1992; Adrogue and Madias, 2000).

Clinical assessment

A meticulous clinical assessment is necessary to identify the severity, duration and cause of hyponatraemia. The history should focus on the nature and duration of hyponatraemic symptoms, symptoms of extra-renal loss of body fluid such as vomiting and diarrhoea, the presence of underlying pathology such as endocrinopathy, infection and malignancy as well as drug history such as the use of diuretics or medications which could cause SIADH.

A thorough physical examination to assess the volume status of the patient including assessing skin turgidity, tachycardia and postural hypotension helps to identify the cause of hyponatraemia as well as dictating further investigation and management. The presence of oedema, which is often caused by both salt and water retention, indicates a hypervolaemic state. SIADH is not associated with oedema since water retention is not accompanied by salt reabsorption and there is no significant increase in extracellular fluid volume because of the hyposmolality-mediated shift of water into the intracellular compartment.

Searching for stigmata of endocrine disorders such as Addison's disease, hypothyroidism and hypopituitarism could prove useful as hyponatraemia can be the initial manifestation of these treatable but potentially life-threatening conditions. Both early and advanced malignancy can also present with hyponatraemia secondary to SIADH.

Investigations

A basic biochemical profile including electrolytes, urea, calcium, glucose, renal and liver function is a useful starting point. Euvolaemic hyponatraemic patients should also have measurement of paired serum and urine osmolalities, urine sodium level, thyroid function and a 9.00am cortisol level which are all part of the necessary criteria to diagnose SIADH. Further investigations may be needed such as short synacthen and/or full pituitary function profile in the cortisol-deficient patient, chest X-ray and computed tomography scan of the thorax and abdomen in patients with SIADH for which no apparent cause has been identified, or computed tomography scan or magnetic resonance imaging scan of the brain in those suspected to have an underlying intracranial pathology.

Pseudohyponatraemia

Pseudohyponatraemia should be considered in the differential diagnosis of euvolaemic hyponatraemia. It is often caused by a disproportionate increase in the non-aqueous components of plasma such as lipids (hyperlipidaemia) and proteins (multiple myeloma). This results in a decrease in the aqueous component of plasma without any change in the physiologically important concentration of sodium per unit volume of water. Pseudohyponatraemia should be suspected in patients with euvolaemic normosmolar hyponatraemia and history of multiple myeloma, recent immunoglobulin therapy or marked hyperlipidaemia. This can be differentiated from true hyponatraemia by direct ion-selective photometry which specifically measures the physiologically relevant aqueous component of plasma.

Another common cause of spurious hyponatraemia is inappropriate blood sample collection from a venous site in the immediate vicinity of an intravenous infusion. The unexpected finding of an acutely low serum sodium level which does not correlate with the patient's clinical status should be verified with repeat measurement of the serum sodium level before it triggers unnecessary investigation and treatment.

Hyperglycaemia-associated hyponatraemia

Hyperglycaemia has a variable effect on plasma sodium level. It causes an increase in serum osmolality which promotes a shift of water from cells thereby causing dilutional hyponatraemia. The plasma sodium level is estimated to fall by 1 mmol/litre for every 3.5 mmol/litre rise in plasma glucose level above the normal range. Hyperglycaemia also causes osmotic diuresis which often involves a greater rate of urinary water loss than urinary sodium loss. This excess water loss from osmotic diuresis could raise plasma sodium level and osmolality thereby counteracting the direct dilutional effect of hyperglycaemia on plasma sodium.

Treatment of hyponatraemia

The treatment of hyponatraemia should take into account different factors such as its cause, severity and duration, the clinical status of the patient and the presence of comorbidities such as heart failure which could dictate the rate of fluid replacement. The temptation to rapidly correct hyponatraemia should be resisted to prevent the potentially catastrophic, often irreversible neurological complication of central pontine myelinolysis. This is particularly true for cases of chronic hyponatraemia where the brain tissue has already taken adaptive measures to mitigate cerebral oedema. The aim of treatment should be to achieve a slow and steady increase in serum sodium level at a rate not exceeding 0.5 mmol/hr (Greenberg et al, 2007). The volume status of the patient, hourly measurement of input-output and serial measurement of serum sodium level are all useful parameters to monitor response to treatment.

The treatment of hypovolaemic hyponatraemia is fluid replacement with isotonic saline guided by physiological parameters such as heart rate, blood pressure and urine output. The euvolaemic patient with mild

to moderate hyponatraemia can be treated by addressing the underlying cause (e.g. withdrawing offending drugs, treating infections) as well as fluid restriction. The actual volume of fluid restriction is determined by the desired rate of improvement in serum sodium level and the expected compliance of the patient but in most cases fluid restriction of 50–60% of the normal fluid intake or <800 ml/24 hr is sufficient to create a negative water balance and restore eunatraemia (Gross, 2001; Greenberg et al, 2007). The level of fluid restriction can be further escalated if necessary depending on the biochemical and clinical response of the patient.

The use of antidiuretic hormone antagonist drugs such as demeclocycline is recommended in mild to moderate hyponatraemia refractory to fluid restriction or in cases of severe hyponatraemia alongside fluid restriction. Vasopressin-2 receptor antagonists such as tolvaptan are also effective in treating both euvolaemic and hypervolaemic hyponatraemia although their relatively high cost currently precludes their routine use (Czerwiec et al, 2006; Berl et al, 2010). Hypertonic (3%) saline is usually reserved for use in acute severe hyponatraemia with neurological manifestations such as seizure, drowsiness and coma. This treatment should ideally be supervised by a specialist in an intensive care setup.

Central pontine myelinolysis (osmotic demyelination syndrome)

Acute hyponatraemia (<48 hours duration) can be complicated by cerebral oedema which can manifest with confusion, seizures and coma. This is the result of osmotic gradient-mediated movement of water into the brain cells. The brain can adapt to cellular oedema over 2–3 days by promoting extrusion of sodium, potassium and organic osmolytes to the extracellular space (Chan et al, 1991; Strange, 1992).

Rapid correction of hyponatraemia once the brain's adaptive mechanism against cerebral oedema has taken place could cause shrinkage of brain cells and the syndrome of osmotic demyelination also known as cerebral pontine myelinolysis, although the condition can involve other areas of the brain besides the pons (Chan et al, 1991; Laureno and Karp, 1993; Cappuccio et al, 1994). The condition manifests with symptoms of dysarthria, dysphagia, paraparesis, quadriparesis, seizures and coma. The demyelination lesions can be detected on computed tomography and magnetic resonance imaging brain scans but the absence of radiological signs does not rule out the diagnosis as it can take up to 4 weeks for signs to evolve (Brunner et al, 1990).

This neurological complication is often irreversible but some manifestations can improve with time and supportive therapy. Reports from animal models and case histories have indicated some response to relowering of serum sodium with desmopressin (Grieff et al, 2008). The best approach remains prevention of this debilitating neurological complication through careful monitoring of the rate of correction of hyponatraemia. The recommended rate for the correction of hyponatraemia is 0.5 mmol/litre/hour or not more than 12 mmol/litre in the first 24 hours.

Table 1. Causes of syndrome of inappropriate antidiuretic hormone secretion

Malignancy	Carcinoma	Lung
		Oropharynx
		Gastrointestinal tract
		Genitourinary tract
	Lymphoma	
Pulmonary disorders	Infections	Pneumonia
		Tuberculosis
	Positive pressure ventilation	
	Asthma	
Drugs	Psychotropic	Selective serotonin-reuptake inhibitors
		Tricyclics
		Antipsychotics
	Carbamazepine	
	Clofibrate	
	MDMA (Ecstasy)	
	Narcotics	
	Non-steroidal anti-inflammatory drugs	
	Cyclophosphamide	
CNS disorders	Infections	Meningitis
		Encephalitis
		Acquired immune deficiency syndrome
	Intracranial bleeding	
	Cerebrovascular accident	
	Brain tumour	
	Head trauma	

Syndrome of inappropriate antidiuretic hormone secretion

SIADH is the most common cause of euvolaemic hyponatraemia. It is caused by non-osmotic dependent release of antidiuretic hormone which results in inappropriate water retention in the face of low serum osmolality and sodium level. Several clinical entities cause SIADH. The most commonly encountered causes include pulmonary and CNS diseases (infections, neoplasms, haemorrhage), other neoplasms, drugs with psychotropic effects such as antidepressants and antipsychotics, narcotics and chemotherapeutic agents (*Table 1*).

Diagnosis of SIADH requires the presence of hyponatraemia and inappropriately concentrated urine in the face of low effective serum osmolality (*Table 2*).

There are important caveats to the biochemical criteria for the diagnosis of SIADH. Glucocorticoid deficiency and hypothyroidism should be ruled out since both conditions can result in a biochemical profile indistinguishable from SIADH. Clinical euvolaemia is also an essential criterion to fulfil before diagnosing SIADH since volume depletion is also a potent stimulus for the release of antidiuretic hormone (Berl and Ellison, 2007). This is particularly true in cerebral salt wasting which is difficult to distinguish from SIADH on biochemical criteria alone. Erroneous diagnosis of SIADH in the volume-depleted hyponatraemic patient could result in inappropriate treatment with fluid

Table 2. Criteria for diagnosis of syndrome of inappropriate antidiuretic hormone secretion

Essential features of syndrome of inappropriate antidiuretic hormone secretion
Decreased effective osmolarity: <275 mOsm/kg of water
Urinary osmolarity >100 mOsm/kg of water
Urinary sodium >40 mmol/litre with normal dietary salt intake
Normal thyroid and adrenal function
Clinical euvolaemia
No recent use of diuretic agents

restriction causing further worsening of clinical status (Cerdà-Esteve et al, 2008).

Cerebral salt wasting syndrome

Cerebral salt wasting syndrome is characterized by hyponatraemia and extracellular volume depletion. It is often confused with SIADH because it has essentially similar biochemical features. This is particularly true when the clinical signs of extracellular volume depletion are subtle. However, the distinction from SIADH is crucial as fluid restriction exacerbates the volume depletion and the clinical status of the patient.

Cerebral salt wasting syndrome is often encountered in patients with subarachnoid haemorrhage but it can also occur in association with CNS tumours, infections and following neurosurgical procedures. There are two major proposed mechanisms to explain the renal loss of sodium in cerebral salt wasting syndrome. Impaired sympathetic input at the level of the proximal tubule leading to sodium and urate loss as well as diminished renin release is one potential mechanism which could explain salt wasting. The other is the release of brain natriuretic protein from injured brain tissue which could also result in impaired sodium reabsorption and renin release (Cerdà-Esteve et al, 2008).

The biochemical profile encountered in cerebral salt wasting syndrome is difficult to distinguish from SIADH (Palmer, 2000, 2003; Cerdà-Esteve et al, 2008). Useful clues include urine sodium level >40 mmol/litre, urine osmolality >300 mosm/kg and low serum uric acid level owing to increased renal loss. The diagnosis, however, relies on accurate detection of signs of volume depletion such as decreased skin turgor, dry mucus membranes, tachycardia and hypotension. The treatment of cerebral salt wasting syndrome is fluid replacement with isotonic saline while closely monitoring volume status and biochemical profile.

Exercise-associated hyponatraemia

This is defined as hyponatraemia occurring during the first 24 hours following prolonged physical activity such as participation in endurance events. This has become increasingly common because of a trend towards non-thirst-driven copious fluid intake. An incidence as high as 13% has been reported among participants in endurance events (Almond et al, 2005). The mechanism of exercise-associated hyponatraemia involves exercise-induced antidiuretic hormone release coupled with high fluid intake and loss of sodium through excessive sweating (Almond et al, 2005; Clement et al, 2007; Ayus et al, 2008).

The presenting symptoms can be mild such as headache, nausea and dizziness but severe symptoms including drowsiness, collapse and seizures can also occur. Fatal cases of exercise-associated hyponatraemia have been reported (Clement et al, 2007). The mainstay of treatment in mild to moderate exercise-associated hyponatraemia is fluid restriction to allow spontaneous aquaresis and restoration of eunatraemia but severe hyponatraemia and presentation with neurological symptoms warrants treatment with hypertonic (3%) saline. The best approach to prevent this potentially serious cause of morbidity is avoiding excessive fluid intake during period of prolonged physical activity and using the physiological stimulus of thirst as a guide to fluid intake.

Conclusions

Hyponatraemia is a common electrolyte disorder among hospitalized patients. It is associated with significant morbidity even when the level of hyponatraemia is mild. A systematic approach towards its diagnosis and investigation allows appropriate management with optimal ou tcome. Correction should be undertaken with caution to avert neurological complications. BJHM

Conflict of interest: none.

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KEY POINTS

- Hyponatraemia is the most common electrolyte disorder among hospitalized patients, and even mild hyponatraemia can cause significant morbidity.
- The presence of hyponatraemia can be a valuable clue to an undiagnosed underlying pathology.
- Accurate assessment of volume status facilitates correct diagnosis and treatment of the cause of hyponatraemia.
- The mode of clinical presentation of hyponatraemia depends on both its severity and duration.
- Management of chronic hyponatraemia requires close monitoring to avoid rapid correction which could result in irreversible neurological sequelae.

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Ultrasound-assisted vascular access in children

Revalidation
FOR ANAESTHETISTS
RCA Revalidation matrix
Matrix reference 2D03

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Key points

Ultrasound is widely available, decreases the complications of paediatric vascular access (if used appropriately), and is a useful training tool.

Scrupulous attention to ultrasound technique and knowledge of normal (and common variations) anatomy is essential to avoid complications.

Ultrasound is particularly useful for assisting access to the internal jugular and femoral veins in all age groups and the subclavian vein in infants.

A small footprint hockey-stick probe of frequency 7–10 MHz is adequate for most children, but higher frequency probes are useful for smaller veins and difficult cases.

Developments in ultrasound technology including three- and four-dimensional probes are likely to improve vessel resolution and successful cannulation in children.

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Vascular access in children can be challenging. There is a considerable body of evidence supporting the use of ultrasound to aid central venous access in adults, but less so in children. Benefits for experienced operators may be small, but there is evidence of benefit for those acquiring skills and for less frequent operators.[1] Special considerations to bear in mind in paediatrics are the smaller size of the patient, the greater mobility of some of the vascular structures, the smaller and more superficially positioned vessels, greater variability in the anatomy, potentially less cooperative patients, and the smaller size of the equipment. The success and complication rates will depend on factors which include site of cannulation, ultrasound technique, the size and condition of the child, operator experience, and the presence of vascular anomalies, coagulation abnormalities, or previous cannulations.

Advantages of ultrasound techniques over landmark insertion methods

(i) Clearly demonstrates vein presence, diameter, patency, direction, and relation to surrounding structures.
(ii) Puncture site and angle of approach of needle to target can be optimized to minimize the risk of complications.
(iii) Using real-time ultrasound can guide needle tip into vessel or can observe vessel compression so a transfixion technique can be used (Fig. 1).
(iv) Guidewire placement within the correct vessel can be confirmed (Fig. 2).
(v) Some immediate complications can be diagnosed or excluded (haematoma/carotid puncture/pneumothorax/haemothorax/pericardial effusion) with appropriate training.

Disadvantages of ultrasound techniques over landmark insertion methods

(i) More complex technique and need for good hand-eye coordination. Poor technique may lead to failure to visualize the needle tip. If the shaft of the needle is mistaken for the tip or if tissue deformation only is seen, then a false impression of the needle position may be given.
(ii) Limitations of ultrasound physics can cause errors, for example, reverberation artefact (Fig. 2) may give a false impression that the needle tip is deeper than it actually is.
(iii) In small infants, the physical size of the probe may be limiting.
(iv) Cost and maintenance of ultrasound equipment.

Ultrasound machines and probes

Over the last few years, there has been considerable advancement in the quality of ultrasound imaging available at the bedside. Initial ambivalence on the part of the paediatric anaesthetic community may in part be related to early experience with inappropriate technology. Although several machines are currently on the market, none offers a clear advantage. The optimal machine should incorporate a lightweight linear array probe with a small footprint, clear screen, high-quality two-dimensional imaging and availability of colour and spectral Doppler. Probes operating at 7–10 MHz are suitable for internal jugular puncture in most children and infants. Higher frequency imaging (13 MHz) will give higher definition and is of value for cannulation of smaller vessels such as antecubital fossa veins, long saphenous veins, and femoral vessels in infants. Lower tissue penetration with high-frequency probes leads to poorer visualization of deeper structures. However, this is of less practical importance in

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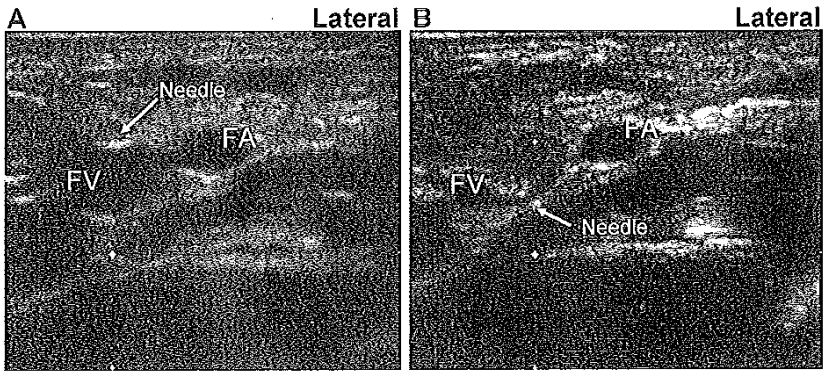

Fig 1 (A and B) Ultrasound images of the left femoral vessels in newborn child. In (A), the artery (FA) and vein (FV) are clearly seen with the needle on the anterior wall of the vein. In (B), the needle has been advanced further causing the vein to be compressed. When cannulating small veins, it is common for the vein to be compressed in this way. Overly tentative advancement of the needle will often lead to failure in cannulation. Assuming no vulnerable structure lies deep to the vessel, it is often advisable to aim to transfix the vessel (with a careful, visualized, short thrusting movement) then withdraw the needle without imaging (while aspirating) until free flow of blood is seen.

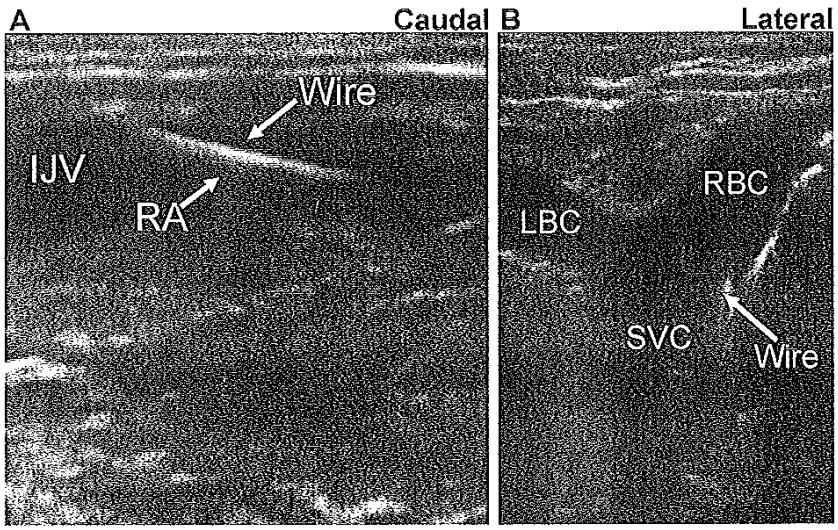

Fig 2 (A and B) In image (A), the IJV is imaged in long axis. The typical appearance of the vessel in long axis is a thick dark line extended across the screen. The guidewire is seen to lie freely within the lumen as a bright echogenic line. Deep to the line, a series of fainter parallel lines are seen, this is reverberation artifact (RA). In this view, it is easy to differentiate this artifact from the true image of the wire; however, when imaging a needle 'out of plane', this artifact can create a false impression of the depth of the needle. Image (B) is an alternative long-axis view of the jugular and brachiocephalic veins (right, RBC; left, LBC). The image is achieved by following the vein caudally in short axis until the probe is against the clavicle. The probe is then angled down acutely to point towards the heart. In this way, the patency of the vessels and the correct position of the wire can be confirmed as far as the superior vena cava (SVC) or right atrium.

children. A robust design, ease of use, ability to run on battery, and fast start up are further considerations.

Principles of ultrasound vascular imaging

Positioning

The machine, operator, and patient should be orientated in a straight line (Fig. 3).

The patient should be positioned to give easy access to the area of interest while avoiding compression or distortion of the anatomy. Aseptic precautions are mandatory.

Initial examination

Establish correct orientation; the probe is orientated so that the left side of the screen (as seen by the operator) corresponds to the left side of the patient.

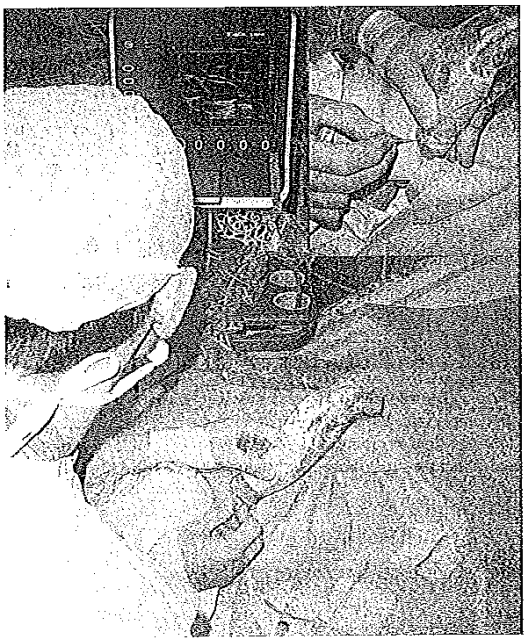

Fig 3 The operator, the child, and the ultrasound machine are positioned such that they form a single line of sight. This allows the operator to look easily from the screen to the insertion site. It also leads to a more logical and intuitive orientation of the ultrasound image, such that objects on the left of the field appear on the left (as seen by the operator) of the screen. The inset shows the detail of how the probe and needle are held. The needle is held in the dominant hand. The position should be comfortable. The ulnar border of the scanning hand should rest gently on the patient (to steady it) and the needle and probe are held in a pencil grip. This allows for fine movements of the probe and needle. The needle is held at a steep angle and the probe angled towards the needle to maximize visibility. There is no need to aspirate on the syringe while advancing the needle, if the tip is continually visualized.

Vessels are echolucent (black) and compressible. Anterior and posterior vessel walls are bright, whereas lateral walls are less distinct.
Doppler mode can demonstrate vascular flow.
Veins are more compressible and less pulsatile than arteries.
Venous flow is towards the heart.
Examine structures around and deep to target vessel to avoid arteries, nerves, and pleura.

Cannulate

Hold needle in dominant hand and probe in non-dominant hand.
Commonly, vessel is visualized in transverse (short axis) plane. Vessel will appear as round or elliptical. The needle is introduced above the probe (out of plane) and appears as a small, bright dot (Fig. 1).
Alternatively, vessel viewed in longitudinal (long axis) plane. Vessel appears as black bar across the screen (Fig. 2). The needle is introduced from the side of the probe (in plane) and appears as bright, white line across the screen.

Using either approach aim to visualize the needle tip, rather than the shaft continuously during advancement.

Peripheral access

Ultrasound may be a useful adjunct when cannulating veins in the antecubital fossa and above the elbow. Light touch is required to avoid compression of the vessel and transfixion is common during cannulation. Ultrasound assistance may help avoid the brachial artery and median nerve. This site may also be useful for peripherally inserted central catheters in neonates, infants, and children. The long saphenous vein can also be visualized with ultrasound.

External jugular vein

The external jugular vein (EJV) is consistently found in the superficial fascia of the neck crossing the sternocleidomastoid muscle obliquely (the vein may be duplicated). In case of difficulty, it is easily visualized on ultrasound examination more laterally and superficially than the internal jugular. It may be increased in size by head-down positioning, Valsalva manoeuvre, and distal occlusion (above the clavicle). It can be used in an emergency if there is no delay (otherwise intraosseous access may be more appropriate), although it is not technically easy to perform EJV cannulation in the awake child. Efforts to pass lines into the deeper veins are inconsistent (50–90%) as wires often fail to traverse the vein as it penetrates the deep fascia above the clavicle.

Internal jugular vein

The internal jugular vein (IJV) is a large and accessible vein from which a line can usually be easily passed into the superior vena cava (especially on the right). Compared with adults, cannulation of the right IJV (RIJV) may be more difficult due to short neck, shallow mobile compressible vein, variable anatomy, and overall reduced space to place the ultrasound probe. The overall and first pass success rates are therefore decreased, even with ultrasound; however, complication rates are not increased.[2] Difficulty may also occur if there have been previous cannulations.

Anatomy

The vein is most commonly in the anterolateral position in relation to the carotid artery (CA) (Fig. 4). Variations in anatomy occur in up to 18% of children under 6 yr of age [absent (<1%)/small/medial/extremely lateral].[3] The relationship to the CA also changes with distance from the subclavian junction, with greater venous overlapping lower in the neck. Location, size, relationship to the CA, and thrombus can all be observed on ultrasound examination, as can complications such as pneumothorax and pericardial effusion (with suitable training). Very low in the neck, the subclavian or vertebral arteries may also be seen posterior to the vein.

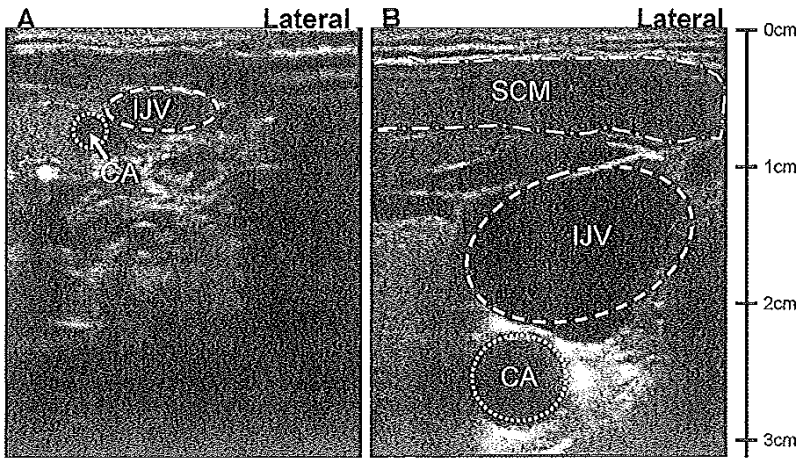

Fig 4 Ultrasound images of the RIJV taken in a 2-month-old infant (A) and adult (B) in the same scale (maximum depth of 3.2 cm). In absolute size, the adult vessels are much larger and deeper than in the infant. In practice, when cannulating an infant's vessel, the image would be magnified to place the vein at half to two-thirds of the depth of the screen (or the maximum resolution of the machine/probe), thus allowing greater resolution of the superficial structures. The anatomy is variable in both adults and children. In this adult, the CA is completely overlapped by the vein and lies deeper than is common. Mean diameter of the vein has been reported as 0.47 cm in infants and 1.94 cm in adults (without associated manoeuvres).

Vein size, head position, and associated manoeuvres

Optimal positioning for cannulation is debatable. Most practitioners will extend the neck and rotate the head away from the site of cannulation. This will improve access to the neck in infants, but will also increase overlap of the vein and artery. Therefore, a neutral position is advocated. The use of ultrasound would detect excessive overlap and allow adjustment of position and site of cannulation. Head-down position, Valsalva, and liver compression will increase the vein diameter in children, but is of limited value in infants.[4] Small diameter veins may cause difficulty with J-wires (the diameter of which is often greater than that of the vessel). Alternatively, straight wires may fail to pass sharp turns in the direction of the vein and may increase the risk of perforation. It is wise to have alternative wires of both kinds available.

Length and size of line

Correct positioning of central catheters is essential to avoid potentially life-threatening complications. Most are not checked until a postoperative chest X-ray (CXR) is obtained. Formulae for insertion length based on patients' height or weight have been reported, but do not take into account variation in puncture site and head position. An alternative is the use of surface landmarks to determine length.[5] In practice, lines are more secure if placed up to their full length. Five-centimetre lines are suitable for most newborns, whereas 8 cm lines should be used in patients >10 kg. On CXR, the carina is a useful landmark, generally being above the pericardial reflection. The diameter of line should be selected according to the intended application. Lines should be no larger than required with no more lumens than necessary. Larger diameter lines carry an increased risk of complication (thrombosis or stenosis of the vessel); however, recommendations made by manufacturers may be unnecessarily conservative. Even in small neonates, 4 Fr double lumen or 5 Fr triple lumen lines are frequently placed during cardiac surgery when multiple infusions and large volume transfusion are anticipated. Alternatively, a 3 Fr or 20 G single lumen catheter will be acceptable for many applications.

Femoral vein

The femoral vein (FV) has traditionally been favoured in small children (to avoid intrathoracic complications). Care must be taken to avoid inadvertent puncture of the femoral artery (FA). Drawbacks are increased risk of infection, kinking, less accurate filling pressures, and increased incidence of thrombosis compared with the RIJV (range reported 4–35%). General anaesthesia is usually required, although insertion with local or regional anaesthesia is possible.

Anatomy

The FV lies superficially in the femoral triangle immediately medial and deep to the FA. The most common anatomical arrangement is shown in Figure 5. The common mnemonic NAVEL (Nerve Artery Vein Empty space Lacunar ligament) from lateral to medial can be used to remember the anatomy; however, the external landmarks do not always predict the internal anatomy.

Ultrasound studies show that the FV was completely or partially overlapped by the FA in 12% of children <9 yr old.[6] An adult study indicated a higher frequency of overlap. The

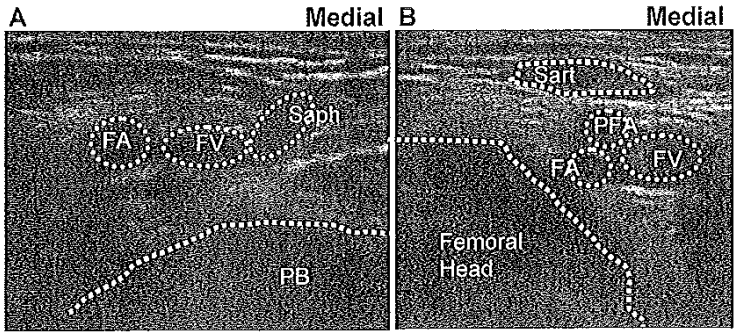

Fig 5 (A and B) The anatomy of the femoral vessels is demonstrated in a 2-yr-old child. (A) is just below the inguinal ligament. In this case, the vein (FV) lies completely medial to the artery (FA) with no overlap. The sapheno-femoral junction is demonstrated. The saphenous vein (Saph) should not be mistaken for the FV. At this level, its course is transverse across the inguinal triangle and cannulation is unlikely to succeed. Deep to the vessels, the pelvic brim is seen (PB). (B) is lower in the leg and two arteries are seen, the FA and profunda femoral artery (PFA). As the vessels are followed distally the PFA lies increasingly anterior and overlapping the vein, and the sartorius muscle (Sart) comes to overlie them. The artery should generally be cannulated above its bifurcation.

percentage of overlap increases more distal to the inguinal ligament. The position of the sapheno-femoral junction is also variable and a potential cause of confusion. The vein is generally cannulated close to the inguinal ligament, but should not be punctured above the ligament as external compression may not be possible. Location, size, relationship to the FA, and thrombus can all be observed on ultrasound examination.

Vein size, position, and associated manoeuvres

Ultrasound studies demonstrate that reverse Trendelenburg positioning, Valsalva manoeuvre, or compression (1–2 cm above the inguinal ligament) can increase the size of the vein. There is no summative increase with multiple manoeuvres and compression may be the simplest and most effective of these techniques.[7] Most practitioners put a bolster or sandbag under the ipsilateral hip and advocate a small amount of external rotation.

Length and size of line

Advice on size is similar to RIJV. As the inferior vena cava is longer than the superior vena cava, length is less critical. In obese children, short multilumen lines can cause problems of subcutaneous extravasation from proximal side holes.

Subclavian vein

The subclavian vein (SCV) is a common site for central venous access (especially longer term), due to reduced infection rates, reduced mechanical problems, and patient comfort. The vein is less mobile and less likely to collapse than the IJV. There is, however, a significant risk of complication with landmark techniques (3–34%) related to age, side of insertion (R>L, as there are more technical problems), and indication. The success rates in landmark studies in infants are consistently only around 80%, with the worst results in those <6 months. Ultrasound techniques offer the promise of improved results, although there is limited evidence even in adults.

Anatomy, techniques, and position

The SCV is formed as a continuation of the axillary vein as it passes the lateral border of the first rib and runs medially in the space between the clavicle and the first rib to the medial border of scalenus anterior where it joins the IJV to form the braciocephalic vein. It can be visualized and cannulated above or below the clavicle with either long- or short-axis views, depending on the preferred technique. An interesting technique that has been very successful in a small study in infants and children involves obtaining a supraclavicular long-axis view of the SCV, combined with an infraclavicular in plane puncture. The needle is seen passing through the anterior wall of the SCV which can be catheterized under direct vision in the direction of its confluence with the IJV. The ipsilateral IJV can also be scanned to exclude or adjust wire malposition. This study of 25 patients had 100% success and no complications.[8]

SCV diameter is maximal with Trendelenburg positioning, no shoulder roll, and neutral head position. Some head rotation and a small roll may be required to create space in infants.

Arterial lines

A small pilot study appeared to demonstrate a significant increased success rate when ultrasound was used for radial cannulation in infants.[9] Ultrasound does allow for cannulation of vessels in atypical sites such as the mid-forearm, where pulses are less readily palpable. It is the authors' practice to use ultrasound when cannulating FAs in infants and newborns. Ultrasound may also allow cannulation in situations where palpation is more difficult, such as low cardiac output conditions.

Training in the use of ultrasound

There are currently no national standards for training anaesthetists in ultrasound-guided vascular access in adults or children.[10] The British Medical Ultrasound Society and The Royal College of Radiologists have attempted to define levels of competency in ultrasound use for non-radiologists (www.rcr.ac.uk/docs/radiology/pdf/ultrasound.pdf), where vascular access is within the critical care block. They suggest a minimum of 25 supervised line insertions on top of theory for basic accreditation in adults. There is no guidance in children.

A survey of paediatric anaesthetists in 2007 suggested that 74% of consultants had received some training in ultrasound use. Of these, 78% of the training was delivered in the workplace and 22% had attended external courses (usually 1 day).[11] It is very unlikely that many would have fulfilled the above requirements.

Training should ideally include:

Theoretical knowledge

Ultrasound physics and manipulation of image.

Relevant topographical anatomy, sonoanatomy, and common anatomical variants.

Practical supervision

Classroom and clinical situations.

'Phantoms', models, or computer simulators.

Named supervisor

Difficult with rotating trainees.

Logbook

With regular review and audit.

Formalized assessment accreditation and revalidation

Not yet available in anaesthesia.

Future developments

Machines are already getting smaller, faster, and have greater resolution than just a few years ago. This trend is likely to continue. The miniaturization of probes is especially welcomed in paediatric practice. Software that automatically steers the ultrasound beam more perpendicular to the needle, to enhance visibility, has already been developed. Manufacturers continue to try and improve needle-tip echogenicity. The development of three- and four-dimensional probes that allow tracking of needles without the need to move probes is increasingly reported in the literature[12] and low footprint (hockey stick) three-dimensional probes are commercially available with potential application in paediatrics. It is also hoped that the gaps in training and accreditation can be addressed to ensure the best use of the available technology.

Conflict of interest

None declared.

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Please see multiple choice questions 8–12.

NOTE FOR PROFESSOR JOHN ALEXANDER
RE: ADAM STRAIN

Background

1. Adam Strain, Claire Roberts, Raychel Ferguson and Conor Mitchell are four children who are the subject of a public Inquiry established under Article 54 of the Health and Personal Social Services (Northern Ireland) Order 1972 and being conducted in Northern Ireland by John O'Hara QC. The current terms of reference of the Inquiry are:

> To hold an Inquiry into the events surrounding and following the deaths of Adam Strain and Raychel Ferguson, with particular reference to:
>
> 1. The care and treatment of Adam Strain and Raychel Ferguson, especially in relation to the management of fluid balance and the choice and administration of intravenous fluids in each case.
>
> 2. The actions of the statutory authorities, other organisations and responsible individuals concerned in the procedures, investigations and events which followed the deaths of Adam Strain and Raychel Ferguson.
>
> 3. The communications with and explanations given to the respective families and others by the relevant authorities.
>
> In addition, Mr O'Hara will:
>
> (a) Report by 1 June 2005 or such date as may be agreed with the Department, on the areas specifically identified above and, at his discretion, examine and report on any other matters which arise in connection with the Inquiry.
>
> (b) Make such recommendations to the Department of Health, Social services and Public Safety and report on any other relevant matters which arise in connection with the Inquiry.
>
> (b) Make such recommendations to the Department of Health, Social Services and Public Safety as he considers necessary and appropriate.

The cases of Claire Roberts and Conor Mitchell have been added to the Inquiry's work by the Chairman under his discretionary power to examine and report on any other matters which arise in connection with the Inquiry

2. Adam Strain was born on 4th August 1991 with cystic, dysplastic kidneys with associated problems with the drainage of his kidneys related to obstruction and vesico ureteric reflux. He was referred to the Royal from the Ulster Hospital in Dundonald. He died on 28th November 1995 in the

Royal following kidney transplant surgery on 27th November 1995 from which he never recovered consciousness.

3. The Inquest into his death was conducted on 18th and 21st June 1996 by John Leckey the Coroner for Greater Belfast, who engaged you as an expert along with (i) Dr. Edward Sumner Consultant Paediatric Anaesthetist at Great Ormond Street Hospital for Sick Children ("Great Ormond Street"); and (ii) Professor Peter Berry of the Department of Paediatric Pathology in St. Michael's Hospital, Bristol. The Inquest Verdict identified Cerebral Oedema as the cause of his death with Dilutional Hyponatraemia as a contributory factor.

4. An investigation was subsequently carried out into the death of Adam Strain and the other children (save for Conor Mitchell) by the Police Service of Northern Ireland ("PSNI"). The PSNI engaged a number of Experts to assist them with their investigation into Adam's death. In addition to you, they also engaged Dr. Edward Sumner Consultant Paediatric Anaesthetist, Mr. Geoff Koffman Consultant Surgeon at Guy's & St. Thomas Hospital and Great Ormond Street and Professor R.A Risdon Consultant Paediatric Pathologist at Great Ormond Street.

5. All of the Experts engaged by the Coroner and the PSNI produced Reports.

The Inquiry

6. The Inquiry has appointed a Panel of Advisers[1] to assist it in its investigations in respect of the children. It has also engaged Experts to deal with a number of discrete issues that are child-specific. The work of all the Inquiry's Advisers is peer reviewed by a team of international Experts.[2]

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1 Dr. Peter Booker (Paediatric Anaesthesia), Dr. Harvey Marcovitch (Paediatrics), Ms. Carol Williams (Paediatric Intensive Care Nursing), and Gren Kershaw (Health Service Management and Patient Safety)

2 Professor Allen Arieff at the University of California Medical School in San Francisco (Internal Medicine & Nephrology), Dr. Desmond Bohn of the Critical Care Unit at the Hospital for Sick Children in Toronto (Paediatric Anaesthesia), Ms. Sharon Kinney at the Intensive Care Unit and Clinical Quality and Safety Unit at the Royal Children's Hospital in Melbourne (Paediatric and Intensive Care Nursing)

Background to Adam

7. Adam Strain was born with cystic, dysplastic kidneys with associated problems with the drainage of his kidneys related to obstruction and vesico ureteric reflux. He was referred to the Royal from the Ulster Hospital in Dundonald and came under the care of Dr. Maurice Savage (Consultant Paediatric Nephrologist)[3] and Mr. Stephen Brown (Consultant Paediatric Surgeon).

8. Adam had multiple operations to his urinary tract, during which he was largely under the care of Mr. Stephen Brown. He had re-implantation of his ureters on 2 occasions and had nephrostomies performed during the early months of his life. On several occasions, he was critically ill and required care in PICU and a brief period of dialysis due to acute renal failure. In addition a fundoplication procedure was carried out in 1992 when Adam was less than a year old, to prevent gastro-oesophageal reflux. Eventually he required all his nutrition through a tube and in 1993 he had a cystoscopy and percutaneous gastrostomy. In October 1995 there was a change of his gastrostomy.[4]

9. Adam was subject to recurrent urinary tract infections and his renal function deteriorated to the point where he required dialysis for uraemia. His mother was trained in the home peritoneal dialysis technique so that he could be dialysed at home. Adam was polyuric and, when he was a few months old, the sodium content of his urine ranged between 29 and 52 mmol/L.[5]

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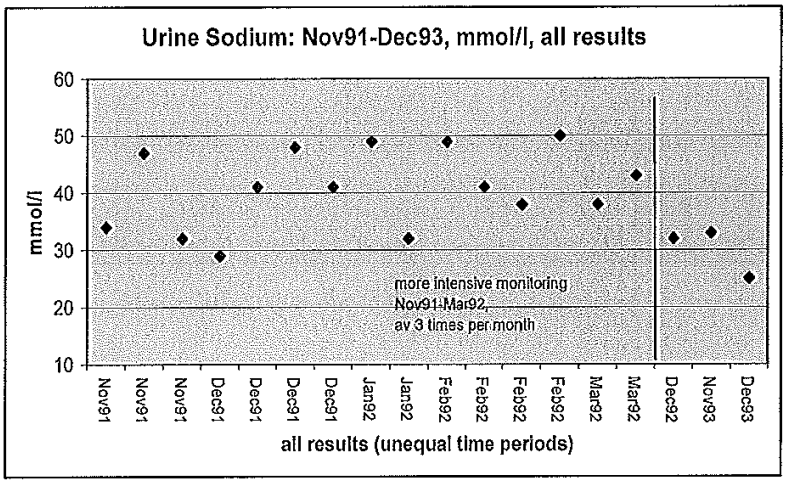
[3] Now Professor Maurice Savage

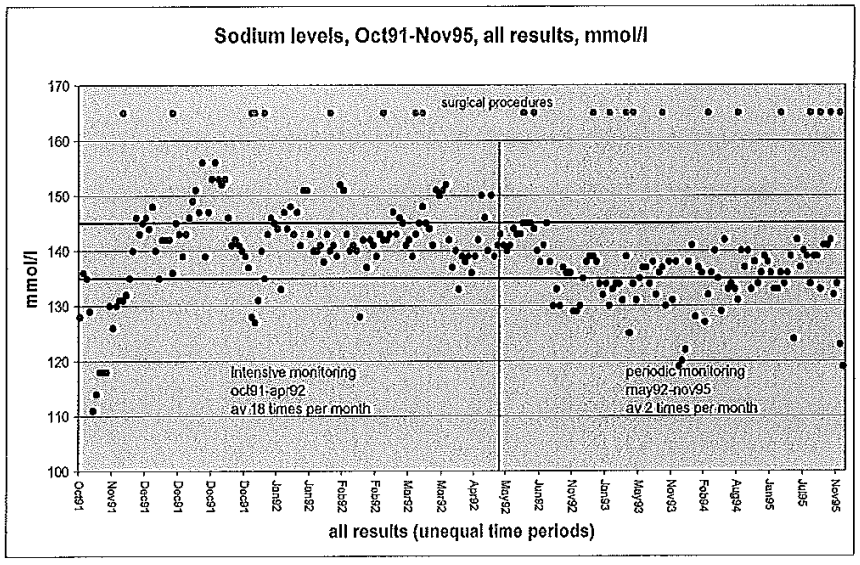
[4] A Schedule is attached-Tab 1 of the accompanying core files- showing all Adam's surgical procedures and their dates together with the surgeons and anaesthetists involved

[5] See biochemistry results in 1995 at: ref: 058-041-187-224 and 050-018-055 - Tab 2

10. A graph of all Adam's recorded urine sodium results is shown below:[6]



11. According to Dr. Maurice Savage,[7] Adam had a potential for generating low serum sodium concentrations and so he received extra sodium in his feeds as 100 mls/day of normal saline and 50 mls/day of 8.4% sodium bicarbonate. A graph of all of his recorded serum sodium concentrations is shown below:



6 The 2 graphs and accompanying tables are found at Tab 3 of the accompanying core file For ease of reference the parallel red lines indicate the normal range of 135-145 mmol/L

7 See ref. 011-015-113. See also letter dated 17th January 1996 from Adam's Mother to the Coroner referring to the fact that it was commonly known that Adam had an ongoing problem with his sodium for which he had been treated the previous 4 years - ref: 011-041-174 -Tab 4

12. The management of his sodium levels appears to have been largely carried out under the care of Messrs. Victor Boston and Stephen Brown, both Consultant Paediatric Surgeons. Adam's recorded serum sodium concentrations for 1995, the year of his transplant surgery, show one very low result of 124 mmol/L and a number below the normal range of 135-145 mmol/L.

13. Adam was put on the waiting list for a kidney transplant once he was placed on dialysis. His total daily gastrostomy feeds in the months prior to the transplantation surgery are documented either as 1700 mls or 2100 mls (1200 or 1500 mls overnight) and he passed an undocumented, so unknown amount of urine, but probably in excess of 1000 ml each day.[8]

14. Adam received the offer of a reasonably matched kidney on 26th November 1995. The donor kidney had been removed from a heart-beating 16-year-old donor with normal renal function at 1.42am on 26th 1995.[9] Transplant surgery was scheduled for 6.00am on 27th November 1995.

15. At 11.00pm on 26th November 1995, Adam's serum sodium was recorded as either 139 or 134 mmol/L (no printed report available) and Hb 10.5. As part of the preparation for his surgery, his feeds were changed although there remains an issue as to exactly what they were changed to. According to his charts, he was given 952 ml of 'clear fluid' to stop 2 hours before going into theatre.[10] The nursing records do not state the nature of the 'clear fluids' given. Some witnesses have claimed that fluid was Dioralyte (containing 60 mmol of sodium /L). However, Dr. Maurice Savage corrected his Deposition to delete 'Dioralyte' and substitute 'N/S Saline Dextrose'.[11] In any event, it is thought that he received just over 1 litre of fluids. It was planned between Dr. Maurice Savage and Dr. Robert Taylor (Consultant Paediatric Anaesthetist) that Adam should receive intravenous fluid (75 ml/h)[12] after the tube feeds were discontinued and have his blood chemistry checked before going to theatre. In fact, IV fluids did not start until anaesthesia was induced and electrolytes were not re-measured preoperatively after 11pm. Suggested reasons were difficulty in achieving

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8 See ref: 059-006-121 – Tab 5

9 See Report of Mr. Geoff Koffman Consultant Surgeon for the PSNI dated 5th July 2006, ref: 094-007-027 – Tab 6

10 See ref: 057-010-013 Tab 7

11 See ref: 011-015-109 – Tab 4

12 See ref: 059-006-022 – Tab 8

venous access[13] and estimated delay in receiving results back from the laboratory ("1-3 hrs").[14]

16. The main events surrounding Adam's transplant surgery are summarised in the following table:

Date	Event		Reference
26.11.1995	1.42am	Donor kidney removed by Mr. Casey at Southern General Hospital, Glasgow	058-009-025 (Kidney Donor Information Form)
	9.00pm	Adam admitted to Musgrave Ward at the RBHSC for possible renal transplant	011-009-001 (Deposition of Ms. Strain 18th June 1996)
	9.30pm	Pre-op investigations for possible renal transplant carried out by Dr. Cartmill (Surgical SHO); Nursing admission details taken by SN Murphy	058-035-144 (Extract from Medical Notes and Records) 049-036-245 (Royal's Chronology of Care)
	10.00pm	Evaluation Nursing Report taken by SN Murphy	049-036-245 (Royal's Chronology of Care)
	11.00pm	i.v. fluids commenced prescribed by Dr. Larkin (Community SHO); Results of investigations recorded by Dr. O'Neill (SHO) as haemoglobin 10.5g/dl, sodium 139 (or 134) mmol/l and urea 16.8; Dioralyte (or some form of dextrose saline solution) instead of Nutrison gastrostomy feeds on Dr. Taylor's (Consultant Paediatric Anaesthetist) advice	• 049-036-245 (Royal's Chronology of Care) • 011-014 & 015 (Depositions of Drs. Savage and Taylor 21st June 1996) • 057-007-008 (biochemistry result list) • 058-035-144 (Manuscript Extract from Medical Notes and Records
	11.30pm	Medical history and clinical examination taken by Dr. O'Neill (Senior House Officer): (i) temp. 36.4; (ii) pulse 97; (iii) blood pressure 108/56; (iv) weight 20.2kg	059-006-009 (Extract from Medical Notes and Records)
27.11.1995	1.30am	SN Murphy recorded i.v. infusion tissued and informed	049-036-245 (Royal's Chronology of Care)

[13] See ref: 011-014-099 and ref: 093-006-017 for the explanation of the difficulty in achieving venous access. Tab 9 See also: 093-035-105 for the other basis as to the time taken to receive results back from the laboratory Tab 10

[14] See ref: 093-035-105 for the other basis as to the time taken to achieve results back from the laboratory Tab 10

NOTE

		Dr. O'Neill	
	5.00am	Unsuccessful attempts at i.v. cannulation ; between 11.00 pm and 5.00 am 952 ml of 'clear fluids' given via gastrostomy, peritoneal dialysis as usual (750 ml 1.36% Dextrose solution – 8 cycles given before theatre)	• 049-036-246 (Royal's Chronology of Care) • 011-015 (Deposition of Dr. Savage 21st June 1996)
	7.00am	General anaesthesia induced in the presence of his mother. 0.18% NaCl in 4%glucose started i.v. by Dr. Taylor – 500 ml given by 7.30am and 685 ml by 7.55 am. Lumbar epidural catheter inserted.	• 058-003-005 (Anaesthetic Record) • 011-014 (Deposition of Dr. Taylor 21st June 1996
	7.30am	Catheter inserted into right subclavian vein. Initial CVP reading taken at 0730 (as per Dr Taylor) or 0755 (as per trend monitor) was 17 mm Hg (normal 2-7 mm Hg); transplant surgery started by Mr. Keane (Consultant Urologist); further 500 ml of Dextrose Saline given up to 8.45am	• 011-014-105 (Transcript of Dr. Taylor 21st June 1996) • 058-003-005 (Anaesthetic Record)
	8.30am	Donor kidney removed from ice; 400 colloid fluids (HPPF) given	• 058-009-027 (Kidney Donor Information Form) • 058-003-005 (Anaesthetic Record)
	8.45am	Rate of Dextrose saline fluids drastically slowed (500ml of given up to 11.00am) and 500ml Hartmann's solution commenced	• 058-003-005 (Anaesthetic Record) • 059-004-007 (Dr. Taylor's note to Mr. Brangam, Solicitor)
	9.15am	400 colloid fluids (HPPF) given	• 058-003-005 (Anaesthetic Record)
	9.32am	Results of blood gases and electrolytes received, showing sodium at 123 mmol/L (normal 135-145 mmol/L) and haematocrit at 18% (normal 35-40%); 250 ml packed red blood cells given	• 058-003-003 (BGE Report) • 058-003-005 (Anaesthetic Record)
	10.45am	200 ml colloid fluids (HPPF) and 250 ml packed red blood cells given	058-003-005 (Anaesthetic Record)
	11.00am	Skin closure; neostigmine and glycopyrrolate administered by Dr. Taylor to reverse the	• Ref:011-014 (Transcript of Dr. Taylor 21st June 1996);

		neuromuscular blockade; blood loss recorded from swabs (328 ml), suction (500 ml) and other (300 ml)	• 058-003-005 (Anaesthetic Record)
	11.55 noon	Adam failed to wake, did not breathe and pupils fixed and dilated	011-014 (Deposition of Dr. Taylor 21st June 1996)
	12.05pm	Adam transferred to PICU for ventilation of his lungs and assessment; puffy appearance with CVP reading of approx 30 mm Hg dropping to 11 mm Hg; Mannitol 50 ml prescribed and reduction in fluids	• 058-005-013 (Drug record sheet) • 058-005-014 (Extract from Medical Notes and Records recorded by Dr. O'Connor) • 094-006-022 (Theatre log)
	12.15pm	Adam's appearance 'bloated'	011- 009 (Deposition of Ms. Strain 18th June 1996) 093-003 & 093-005 (PSNI witness statements of Adam's mother) Photographs.
	7.35pm	First brain stem test carried out by Dr. Webb (Consultant Paediatric Neurologist)	058-004-009 (Brain Death Form)
28.11. 1995	9.10am	Second brain stem test carried out by Dr. Webb (Consultant Paediatric Neurologist)	058-004-009 (Brain Death Form)
	9.15am	Life pronounced extinct	011-010-011 (Report of Autopsy 29th November 1995)
	11.30am	Ventilatory support withdrawn from Adam in the presence of his Mother	011-015-110 (Deposition of Dr. Savage 21st June 1996)

Issues

17. Dr. Edward Sumner (Consultant Paediatric Anaesthetist) concluded in his Report to the Coroner dated 22nd January 1996:[15]

> I believe that on a balance of probabilities **Adam's gross cerebral oedema was caused by the acute onset of hyponatraemia** (see reference) from the excess administration of fluids containing only very small amounts of sodium (dextrose-saline and plasma). This state was exacerbated by the blood loss and possibly by the overnight dialysis.
>
> A further exacerbating cause may have been the obstruction to the venous drainage of the head. If drugs such as antibiotics were administered through a venous line in a partially obstructed neck vein then it is possible that they could cause some cerebral damage as well.

[15] See ref: 011-011-053 - Tab 11

(emphasis added)

18. Dr. Sumner also gave evidence at Adam's Inquest and his Deposition of 18th June 1996[16] records him as having expressed the following views:

> All the fluids given after dialysis may have been given to increase central venous pressure. It may have had the effect of causing the dilution of the sodium in the body. **Fluid balance in paediatrics is a more controversial area with a variety of views. With kidney transplants one gives more fluids than in other operations** *["it is usual to be generous with fluids to maintain a CVP of 10-12 to optimise perfusion of the new kidney and to establish its urine-producing function"[17]]*. When the new kidney is perfused it is vital that sufficient fluids are available. I got the impression that Dr. Taylor was not believing the CVP readings he was getting. I believe they were probably correct but high. I think I would have believed them. A high CVP can mean too much fluid has been administered[18] ... The **low sodium was indicative of the hyponatraemia. Below 128 is a hyponatraemic state**.
>
> (Emphasis and parenthesis added)

19. Dr. Robert Taylor (Consultant Paediatric Anaesthetist) gave evidence at the Inquest. His Deposition of 21st June 1996[19] shows that he disagreed with Dr. Sumner's principal finding:

> **I cannot understand why a fluid regime employed successfully with Adam previously**, led on this occasion to dilutional hyponatraemia ... I believe that **the underlying cause of the cerebral oedema was hyponatraemia (not dilutional)** during renal transplant operation.
> ...
> Adam was the only child with polyuric renal failure I have anaesthetised for renal transplant. He needed a greater amount of fluid because of the nature of the operation *["All the more important in this case is the need to avoid dehydration that will deprive the donor kidney of sufficient fluid to produce urine"[20]]*. I believe the fluids given were neither restrictive or excessive. **The new kidney did not work leading to a re-assessment of the fluids given**. This made us think we have underestimated fluid and we gave a fluid bolus at 9.32.
>
> (Emphasis added)

20. Dr. Robert Taylor commented on preparing Adam for the surgery:

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16 See ref: 011-011-042 – Tab 11

17 See Dr. Sumner's Report of 22nd January 1996 at ref:011-011-059 – Tab 11

18 Dr. Sumner prepared his Report on the basis that Adam received 900mls of Diorolyte. See at ref: 011-011-055. That figure was corrected in correspondence between the Coroner and Dr. Armour but it is not clear that the correspondence from Adam's mother referring to the lower figure was passed to Dr. Sumner. Dr. Armour thought that the difference between the two figures made no difference to her opinion on the cause of Adam's death: *"It is not just the volume of fluid he received but the type."* See at ref: 011-079-214 – Tab 12

19 See ref: 011-014-108 – Tab 9

20 See Deposition at ref:011-014-100 – Tab 9

"with paediatric anaesthesia there is a compromise to be made ... we knew from many times on dialysis that his blood chemistry and his water content of his blood were ... fixed so we could assumptions [sic], do we hurt him with needles or do we assume that this management of dialysis was the same as before."[21]

21. He further commented on the amount of fluids that Adam could tolerate:

"I agree with Drs. Sumner and Alexander that any other child would not have been given that quantity of fluid. Adam was very exceptional and I don't feel that those two individuals really understood Adam. Dr. Taylor confirmed that the 300mls given by Dr. Loan was given over one hour. The knowledge that Dr. Taylor had was that Adam could tolerate very high quantities of this fluid without any loss from his body and recover safely".

The reference to Dr. Loan's previous anaesthetic regime for Adam was relied upon by Dr. Taylor as establishing that: "Adam was not a normal child because a normal child could not cope with 300mls in one hour".[22]

22. Dr Taylor also commented on 'dilutional hyponatraemia' and its applicability to Adam:

"It was impossible for Adam to suffer from dilutional hyponatraemia contrary to the view of the Coroner and the experts because he could not concentrate urine. Therefore Adam could not fit Dr. Sumner's theory ... He stressed that dilutional hyponatraemia was only a theory, that cases had been described but only in children with intact kidneys."[23]

See also: "For the dilutional hyponatraemia theory to work intact kidneys were required which in periods of dehydration shut down and retain water. This was the mechanism in the deaths of Raychel Ferguson and Lucy Crawford who has passed small volumes of concentrated urine, retaining free water while losing sodium and hence suffered dilutional hyponatraemia. It was impossible for Adam to suffer from dilutional hyponatraemia contrary to the view of the Coroner and the experts because he could not concentrate urine. Therefore Adam could not fit Dr. Sumner's theory".[24]

See further: "Dr. Taylor then contended that it was possible that if Adam was given 500 ml he could pass 500 ml in urine. No-one knew what his maximum output was, only that his minimum output was 200 ml. Dr. Taylor's knowledge of the disease was such that he believed Adam could pass an unlimited amount of fluid. No one had established a maximum output for Adam."[25]

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21 See ref: 093-035-094. Dr. Robert Taylor was interviewed by the PSNI under caution in relation to 'manslaughter by gross negligence' in respect of Adam's death – ref: 093-035-089 – Tab 10

22 See ref: 093-035-096

23 See ref: 093-035-102 **Tab 10**

24 See ref: 093-035-102

25 See ref: 093-035-103.

See also: "the theory of dilutional hyponatraemia was improperly applied to Adam and involved making the diagnosis for a known disease. He stated there was no evidence that a child like Adam could get dilutional hyponatraemia."[26]

Finally: "Dr. Taylor had many patients in intensive care whose sodium is low at the time of death, whether that was the cause of death or the result of a dying process is debatable. He acknowledged that hyponatraemia was present but not that it caused his death. Police put to Dr. Taylor that he had said in a letter to a solicitor that 0.18% saline was isotonic, when in effect, its effect once infused is hypotonic. Dr. Taylor stated that depended on the metabolism of the patient, depending on how quickly he burned the glucose. Dr. Taylor explained that Adam did not need too much glucose as the body burns less under anaesthetic. This enhances the ability of the fluid to remain isotonic. This was another reason for the theory of dilutional hyponatraemia to be inapplicable – none of Arieff's patients had died on the table they had all died post-operatively."[27]

23. Mr. Geoff Koffman (Consultant Surgeon at Guy's & St. Thomas Hospital and Great Ormond Street), was retained by the Police Service of Northern Ireland (PSNI)[28] to assist with their investigation into the circumstances of all of the children's deaths. He states in his Report of 5th July 2006 that:

"The sodium and potassium should have been repeated prior to start of surgery. The polyuric patient with poor renal function would pass large quantities of dilute urine and may have difficulty controlling the concentration of sodium and potassium in the blood".[29]

See also "[it being] commonly agreed that he [Adam] was polyuric and could cope with an oral intake of in excess of 2 litres a day"30 and, in respect of the "minority of patients that are polyuric", that "the bladder may be left of free drainage in these patients. It would not be particularly important to monitor the urine output in these patients."[31]

Queries

24. You made a Deposition at the Inquest on 18th June 1996, in which you provided your written report[32] and oral answers to Counsel on cross-examination[33].

25. The Inquiry has appointed Dr. Peter Gross as an Expert Witness on hyponatreamia to assist its investigation in respect of the fluid management issues.

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26 See ref: 093-035-103 – Tab 10

27 See ref: 093-035-110

28 The PSNI conducted an investigation into the deaths of all of the children over a period of about 2 years before deciding not to prosecute anyone in connection with their deaths

29 See ref: 094-007-032 – Tab 6

30 See ref: 094-007-029

31 See ref: 094-007-035

32 See ref: 011-012-079 and ref: 011-012-084 – Tab 13

33 See ref: 011-012-083

NOTE

26. Issues have arisen out of your Deposition that require clarification, expansion and/or further explanation:

(i) *"There was a fluid deficit between 5am and 7am. That would have been a normal precaution for any child coming to surgery."* (Ref: 011-012-083)

- Quantify the "fluid deficit" that you state would have existed and explain why that would have been "a normal precaution".

(ii) *"During surgery it would have been impossible for the anaesthetist to measure urinary output."* (Ref: 011-012-083)

- Explain the reasons why "it would have been impossible for the anaesthetist to measure urinary output" during Adam's transplant surgery.

(iii) *"I am not convinced that tying off the internal jugular vein effected drainage from the vein."* (Ref: 011-012-083)

- Explain the reasons why you are "not convinced that tying off the internal jugular vein effected drainage from the vein".

(iv) *"I would not entirely concur with Dr. Sumner's view that a compromised renal function is not a factor in the onset of hyponatraemia."* (Ref: 011-012-083)

- Explain the basis of your difference of view with Dr. Sumner in respect of compromised renal function beign a factor in te onset of hyponatraemia.

(v) *"With the benefit of hindsight sodium levels in children with a compromised renal function should be monitored"* (Ref: 011-012-083)

- State the reasons why "sodium levels in children with a compromised renal function should be monitored"
- State how frequently "sodium levels in children with a compromised renal function should be monitored"

(vi) *"I agree that a reading of 123 suggests that something should be done but I would not have been particularly alarmed"* (Ref: 011-012-083)

- Describe exactly what you consider "should be done" when there is "a reading of 123"
- State the reasons why you "would not have been particularly alarmed" by a reading of 123 and explain whether your statement is a general one or in relation to the particular circumstances of Adam's case.

(vii) *"If I thought a transducer was giving a faulty reading I would get another one. I think it was unlikely that a 1000ml infusion of saline would raise the venous pressure to 17mm. I do not know what volume would achieve that. I do not believe that the problem could be recognised until after the operation"* (Ref: 011-012-083)

- Specify what you mean when you refer to "the problem"
- Specify what you mean by "recognised"
- Explain the reasons for not believing "that the problem could be recognised until after the operation"

(viii) *"I would agree that in Arieff's paper and in Adam's case there was a high infusion of fluids"* (Ref: 011-012-083)

- Explain and quantify what you mean by "a high infusion of fluids"

27. Issues have arisen out of your Report[34] that also require clarification, expansion and/or further explanation:

(i) *"He was 4 years 3 months of age, weighed 21 kg"* (Ref: 011-012-084)

- State the basis upon which you report that Adam weighed 21 kg.

(ii) *"Relevant blood tests that evening were ... sodium 139 millimoles per litre (mmol/l) ... the remainder [of results] were within normal limits"* (Ref: 011-012-084)

- State what you consider as the "normal limits" for blood serum sodium results.

(iii) *"He was given 952ml 'clear fluid', presumably water, overnight into his gastrostomy and this was stopped at 0500 on the 27th"* (Ref: 011-012-084)

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[34] See ref: 011-012-084 – Tab 13

- State the basis upon which you state that the "clear fluid" was "presumably water"

(iv) *"There were no dramatic changes and no evidence of either hypoxia or hypotension, as documented by Dr. Taylor's meticulous records, and confirmed by the computerised print out obtained at the end of the operation"* (Ref: 011-012-085)

- Explain what you mean by "no dramatic changes", describe what measurements you included in that assessment and explain why.
- Identify the "computerised printout" to which you refer and (if you have access to it) provide a copy of it with your response
- Explain the basis for your description of Dr. Taylor's records as "meticulous"

(v) *"Central venous pressure remained very high throughout the procedure; this may have been partly due to a technical problem with the pressure transducer but also partly deliberate, since releasing the clamps on a transplanted near-adult sized kidney in a child can divert most of the cardiac output into the new organ with a dramatic fall in blood pressure; a high venous pressure will encourage a high cardiac output and avoid this problem"* (Ref: 011-012-085)

- Explain how the central venous pressure remaining "very high throughout the procedure" was "partly deliberate"
- Describe the "deliberate" acts which resulted in this "very high" central venous pressure
- Describe and explain what you consider to be an acceptable range for central venous pressure during a paediatric renal transplant

(vi) *"A simple calculation reveals that if 1500ml 1/5 isotonic or 'normal' saline is infused into a child of this size, plasma (or serum) sodium will fall to about 120mmol/l. Since it takes some time for infused fluids to leave the vascular compartment, serum (or plasma) sodium is likely to be even lower than this and the situation may be made worse by increased levels of antidiuretic hormone produced during anaesthesia which will cause water retention by the kidneys"* (Ref: 011-012-085)

- Explain the "simple calculation" which "reveals that if 1500mls 1/5 isotonic or 'normal' saline is infused into a child of this size, plasma (or serum) sodium will fall to about 120mmol/l"
- Explain the length of time it would likely haven taken "for infused fluids to leave the vascular compartment" in Adam

- Assess and explain how much lower than 120mmol/l you consider that Adam's plasma sodium is likely to have been given the time it would have taken "for infused fluids to leave the vascular compartment"
- Describe and explain the levels of plasma sodium (ie the "situation may be made worse") that might have been achieved due to "increased levels of antidiuretic hormone produced during anaesthesia which will cause water retention by the kidneys"
- Explain what conclusions can be drawn from the fact that Adam's plasma sodium levels remained so low for so long (ie 123mmol/L at 9.32am falling to 119mmol/L at 1.00pm following his transfer to PICU and thereafter does not rise above 123mmol/L until 8.00am on 28th November 1995 when it is recorded at 125mmol/L)

(vii) *"The complex metabolic and fluid requirements of this child having major surgery led to the administration of a large volume of hypotonic (0.18%) saline which produced a dilutional hyponatraemia and subsequent cerebral oedema. The operation was difficult and prolonged and the problem could not be recognised until the surgery was completed"* (Ref: 011-012-086)

- Specify "the complex metabolic requirements" of Adam "having major surgery"
- Explain how these "complex metabolic requirements" led to Adam being administered "a large volume of hypotonic (0.18%) saline which produced a dilutional hyponatraemia and subsequent cerebral oedema"
- Explain what alternative type and volume of saline solution could have been administered to address Adam's "complex metabolic requirements" during this surgery
- Explain "the complex ... fluid requirements" of Adam "having major surgery"
- Describe and explain what alternative type and volume of saline solution could have been administered to address Adam's "complex ... fluid requirements" during this surgery
- Describe and explain "the problem" to which you refer
- Explain the reasons why this "problem could not be recognised until the surgery was completed"

NOTE

DECLARATION OF INTEREST FORM

TO Anne Dillon
Solicitor to the Inquiry

FROM

I confirm that I have read the list set out below and have marked on the attached sheet those individuals with whom and (where those individuals represent an organisation, firm or government department) that organisation, firm or government department with which I declare an interest:

I confirm that: (please delete as appropriate)

a) I have disclosed on an attached sheet the existence and particulars of any personal or professional interest that I have had with the following individuals and organisations:

Dr. Maurice Savage
Dr. Mary O'Connor
Dr. Robert Taylor
Dr. Terence Montague
Mr. Patrick Keane
Mr. Stephen Brown
The RBHSC and its administrators and management, including Dr. G. A Murnaghan, Dr. J. Gaston, Dr. S. McKaigue, Dr. P.M. Crean
Belfast Health and Social Services Care Trust formerly the Royal Group of Hospitals and Dental Hospital Heath and Social Services Trust

"Professional interest" includes contact through collaboration on research, other investigations and committee work.

b) I have no such interest to declare

I acknowledge that I am under a continuing duty to declare any personal or professional interest with those listed above that may arise hereafter.

SIGNED:

DATE :