Witness Statement Ref. No. 014/2

NAME OF CHILD: Adam Strain

Name: Mary O'Connor

Title: Dr

Present position and institution: Consultant Paediatric Nephrologist, Royal Belfast Hospital for Sick Children, 180 Falls Road, Belfast, BT12 6BE

Previous position(s) and institution(s):
[Since your Witness Statement of 19th July 2005] No change since 2005

Membership of Advisory Panels and Committees:
[Identify by date and title all of those since your Witness Statement of 19th July 2005]

Member of Regional Nephrology Expert Group re Erythropoiesis Stimulating Agents 2007-2009.

Member of Fluid Therapy Group RBHSC 2007 re NPSA guidance implementation.

Member of Kidney Advisory Group, NHS Blood and Transfusion, Organ Donation Directorate (Group first formed October 2008 - meets in Bristol).

Nephrology Networks Group - a sub-group of British Association of Paediatric Nephrology and Royal College of Paediatrics advising about networks of care (Group formed 2010 - meets in Royal College of Paediatrics, London).

Other Statements, Depositions and Reports:
[Identify by date and title all those since your Witness Statement of 19th July 2005]

Statement to PSNI 12.04.06.

OFFICIAL USE:
List of previous statements, depositions and reports attached :

Ref:	Date:	
014/1	19.07.2005	Witness Statement to the Inquiry on Hyponatraemia
093-020	12.04.2006	Statement to PSNI

IMPORTANT INSTRUCTIONS FOR ANSWERING:
Please identify clearly any document to which you refer or rely upon for your answer. If the document has an Inquiry reference number, e.g. Ref: 049-001-001 which is 'Chart No.1 Old Notes', then please provide that number. If the document does not have such a number then please provide a copy of the document.

I QUERIES ARISING OUT OF YOUR INITIAL WITNESS STATEMENT

With reference to your Witness Statement dated 19th July 2005, please provide clarification and/or further information in respect of the following:

(1) Answer to Question 1 at p.2

"At the time of Adam's renal transplant on 27th November 1995 I was employed as a Consultant Paediatric Nephrologist in the Royal Belfast Hospital for Sick Children. I had taken up this post on 1st November 1995"

(a) Describe your work commitments to the Royal Belfast Hospital for Sick Children (RBHSC) from the date of your appointment as a Consultant and particularly over the period 26th November to 28th November 1995

I was appointed as Consultant Paediatric Nephrologist in the Royal Belfast Hospital for Sick Children on 1st November 1995. I was responsible for the care of children with renal problems and also provided on-call cover for general paediatric patients on a rota basis. My renal work commitments involved being present in RBHSC from 9am to 5pm Monday to Friday and being available for telephone consultation or to come back to the Hospital for emergencies on alternate nights and weekends (this commitment was shared with Dr Savage and hence we also covered each other's holidays). The weekend commitment was to do a daily ward round on weekends on-call and to come back to the Hospital as necessary for emergencies. My commitment to general paediatrics was approximately on a one in five nights and weekends basis.

On the day of 26th November I was on a day off as this was Sunday. I arrived at work at approximately 9am on Monday, 27th November and was due to be in the Hospital from 9am to 5pm. My planned commitments during that day would have involved the care of ward patients and day attenders. On my arrival to the Hospital at 9am I discovered that a renal transplant was in progress. A Nephrologist would be responsible pre-operatively for planning the immunosuppressive drug regime to be given intra-operatively (this had already been planned by Dr Savage). I was expecting to provide post-operative care for Adam with regard to supervision of fluid and drug management as Dr Savage planned to go to the University later that morning. I called into theatre on several occasions during the morning to ascertain progress of the transplant. My handwritten notes tell me that I was involved with Adam's care from 12.05pm in the Intensive Care Unit (058-035-135). I have written in Adam Strain's notes at 1 pm and 17.10hrs (058-035-137 and 139) on 27th November 1995. I do not recall who was on call as Nephrologist overnight on 27/11/95 but observe that Dr Savage has written in the notes at 8.30 pm and 11 pm 27/11/95. On the day of 28th November I would have been committed to being in the Hospital from 9am to 5pm for the care of ward patients and day attenders in the morning and to do an outpatient clinic in the afternoon. The notes recorded by Dr Webb on 28/11/95 at

9.10 am (058-035-142) state that I examined Adam's brain stem death criteria with him.

(2) Answer to Question 1 at p.2

"Dr Savage and I had named responsibility for individual patients but provided cross cover for each other at nights, weekends and at times of holiday and emergency when the named Consultant was not available"

(a) State whether you had previously provided *"cover"* for Dr Savage in respect of Adam and/or otherwise been involved in his care, if so please:

- give the dates
- describe and explain the circumstances and what if any action you took
- describe the fluid management regime employed on each occasion
- lessons that you learned from your prior treatment of Adam

I commenced my Consultant post on 1st November 1995 and am unsure if I met Adam between then and his transplant. I have written a detailed note in his chart dated 9th November 1995. This note is incomplete as it is not finished or signed. This note represents a summary for my own benefit of a patient whom I did not know well but would need to get to know for my on-call duties. Adam attended a ward based clinic on 9th November and was seen by Dr Savage (as evidenced by a clinic letter 058-017-047) and I do not recall if I met him on that day (it is possible that I sat in on the consultation). I have no other recollection of being involved in his care as a Consultant prior to 27th November 1995. I was employed as a Senior Registrar in Musgrave Ward from 1/12/92 to 30/7/93 and I met Adam during that time when he was admitted to the ward with a temperature on 10/2/93 (055-053-108) and 22/4/93 (055-053-119).

(3) Answer to Question 1 at p.2

"Adam was a patient of Dr Savage's and preparation for his renal transplant had been made the previous evening on 26th November 1995. The renal transplant surgery was in progress when I arrived in the Hospital on the morning of 27th November 2005."

(a) Describe and explain how you came to be providing *"cover"* for Dr. Savage during Adam's transplant surgery, including:

- when you were first informed about the offer of a possible donor kidney for Adam
- when you were first informed that the transplant surgery was proceeding
- what Dr. Savage asked you to do and what he told you about the information that he had provided to the Anaesthetic and Surgical teams
- what you were informed about the fluid management plan for Adam's surgery, when and by whom

I first knew about the kidney offer and subsequent transplant when I arrived at work on Monday, 27th November 1995 at approximately 9am. The patient had been in theatre for several hours at that time. Dr Savage informed me that the transplant was in progress and requested that I supervise post-operative care and fluid management for Adam as he intended to go to the University later that morning. I was not involved in planning the pre-operative fluid management. I went into theatre on more than one occasion to ask how the operation was

progressing and try to anticipate when I would be needed to supervise the post-operative care. I used some of the time available to begin my note in the chart about the transplant by first summarising the information which was available on the forms from UK Transplant accompanying the donor kidney(058-009-025, 058-009-027) . The fluid management plan peri-operatively was supervised by the anaesthetist. I recall noting at some point in theatre that the CVP was 30mmHg and was informed that the initial reading at the beginning of theatre was 17mmHg, which was unlikely in a child who had just had dialysis, and for this reason the measurements were presumed by the anaesthetic team to be inaccurate.

(b) State what role, if any, you had in the care of Adam prior to his admission to RBHSC, including your knowledge of his condition, medication and previous treatment

I wrote a note in Adam's chart on 9th November 1995 (058-035-143) summarising details about his condition, drug treatment and dialysis for my own benefit. I do not recall if I met him on that day.

(c) State what role, if any, you had in the care of Adam prior to the beginning of his renal transplant surgery at 7am on 27th November 1995 including your knowledge of the results of pre-transplant checks and tests

I arrived in the hospital at approximately 9am on 27th November 1995 so I was not involved in Adam's care prior to his transplant surgery.

(d) Describe and explain what you considered to be your role in relation to and responsibilities towards Adam between 8pm on 26th November 1995 and 11.30pm on 28th November 1995, and in particular:

- **from Adam's admission to RBHSC until his arrival in theatre**
- **while Adam was in theatre until his admission to PICU**
- **from admission to PICU until his death**

From Adam's admission until his arrival in theatre I was not in the Hospital or on duty and had no role in his care. I arrived at work at approximately 9 am on 27/11/95. While Adam was in theatre I considered that my role was to liaise with the Anaesthetist to ensure that immunosuppression was given in an appropriate dose and at the appropriate time. I considered it my responsibility to supervise closely his post-operative fluid and drug management as the first 6hrs following a renal transplant are crucial with regard to fluid management, CVP and blood pressure. However at the end of theatre Adam was obviously seriously ill as he had fixed dilated pupils and absence of breathing suggesting very significant brain dysfunction. My role was to seek a cause for this, administer any appropriate treatment and liaise with neuroradiology, neurologists and intensive care anaesthetists for further investigation and clinical opinion. As Adam was not my patient I recalled Dr Savage to the Hospital immediately on discovering his fixed dilated pupils as he knew the family well and was best placed to speak with them

(4) Answer to Question 1 at p.2

"At some time during the morning of 27th November 1995 Dr Savage had commitments in the School of Medicine, Queen's University in his role as Senior Lecturer and hence I made myself available to

attend to Adam's post operative care."

(a) State the time at which *"Dr. Savage had commitments in the School of Medicine"* and explain what you were told those *"commitments"* were

I do not recall at what precise time Dr Savage went to the School of Medicine. His job contract was such that 50% of his day time work was there. I did not enquire what his commitments were but as Senior Lecturer he was responsible for medical student education.

(b) Explain what you mean by *"I made myself available."*

I was on site in the Hospital ready to supervise the post-operative transplant care. I went into theatre on several occasions as I was keen to know how quickly the operation was progressing and when I would be needed in Intensive Care at the end of surgery.

(c) State the time you arrived at the RBHSC on the morning of 27th November 1995 and the time at which Dr Savage left the RBHSC to attend to his *"commitments in the School of Medicine"*

I would have arrived at approximately 9am on 27th November 1995. I do not recall what time Dr Savage left RBHSC.

(d) State the time on 27th November 1995 you made yourself available to attend to *"Adam's post operative care"* and describe what *"attending to [his] post operative care"* means and what it would entail

I was available from 9am in the Hospital. The post-operative care that I expected to provide would have been that which is routine for any kidney transplant patient. It involves meticulous observation of parameters such as blood pressure, central venous pressure and urine output and the prescription of appropriate fluids on an hourly basis. It also involves prescription of immunosuppressive therapy and other drugs. It would also entail ensuring that appropriate haematological, biochemical and radiological investigations are performed and that results are acted upon in a timely fashion.

(e) Describe the discussions you had with Dr. Savage regarding Adam's condition and care before he left to attend to his *"commitments in the School of Medicine"*

I do not recall the precise discussions that I had with Dr Savage but I would expect that it involved a handover of clinical details about Adam.

(f) State the times at which you were gowned and present in the operating theatre and explain your reasons for being there

I do not recall the times at which I was in present in theatre. I would have worn theatre 'blues' but would not have needed to be gowned as I am not a surgeon. My purpose for being there was to ensure that the immunosuppressives had been prescribed for peri-operative use (which they had been) and to ascertain when the surgery would be over so that I could be immediately available to supervise post operative care.

(g) Describe what happened whilst you were in theatre, including:

- **when you arrived in theatre and when you left**
- **what consultation took place, with whom and to what end, including in respect of Adam's fluid management during the course of his surgery**
- **what time the surgery ended (as in when was the last skin suture made)**

I do not recall when I arrived or left theatre. I do not recall what consultation took place with regard to fluid management other than discussing with Dr Taylor the CVP readings which he felt to be inaccurate. I do not know at what time the surgery ended but my clinical note tells me that the child returned to Intensive Care which was immediately adjacent to theatre at 12.05hrs.

(h) State who was present in theatre whilst you were present, including whether there was a designated anaesthetic nurse present during the procedure, and if so, who that was

I do not recall everybody who was present in theatre. I know that Dr Taylor, Mr Keane and Mr Brown were present. There would have been a scrub nurse and a team of theatre nurses. I do not know if there was a designated anaesthetic nurse.

(i) Describe any discussions you had with Adam's mother about his transplant surgery, including:

- **when and how often you spoke to her**
- **the reason for speaking to her, what you told her about Adam's condition and the progress of the surgery**

I do not recall what discussions I had with Adam's mother. I was new to my Consultant post in November 1995 and since that time I have developed a habit of being present intermittently in theatre during renal transplants (if other clinical commitments allow) and of leaving theatre once the vascular anastomosis of the kidney has been made to speak to parents and inform them about progress of the transplant. I am unsure if I did this in Adam's case or if I only developed this habit as my clinical practice continued.

(j) Describe any discussions you had with Dr. Savage in relation to Adam's transplant surgery, including:

- **when and how often you spoke to him**
- **the reason for speaking to him, what you informed him about the progress of the surgery and Adam's condition thereafter**

My clinical notes tell me that I informed Dr Savage about Adam's condition shortly after discovering that his pupils were fixed and dilated at 12.05pm. I would have informed him about my concerns of serious brain dysfunction.

(5) Answer to Question 1 at p.2

"I was present in theatre towards the end of the operation (I do not recall precise timings). I was aware that at the end of surgery Dr Taylor discovered Adam to have fixed dilated pupils"

(a) State when and where Dr Taylor discovered Adam had fixed dilated pupils

At the end of the anaesthetic when trying to wake Adam up Dr Taylor discovered that he had

fixed dilated pupils. I think this was about 12 midday as he was moved immediately to intensive care and I recorded a note there at 12.05 pm (058-035-135).

(b) Explain how you were *"aware that at the end of surgery Dr Taylor discovered Adam to have fixed dilated pupils"* and describe what was done when Dr Taylor made that discovery and by whom

Dr Taylor told me that Adam had fixed dilated pupils. Adam was immediately transferred to Intensive Care for hyperventilation and I prescribed mannitol in an attempt to reduce cerebral oedema(058-035-137).

(c) Explain what *"present in theatre "* and *"the end of the operation"* mean

I do not recall whether I was waiting in theatre for the operation to finish or whether I was called to theatre. The surgical procedure would have finished before Dr Taylor tried to wake Adam up and it was at this point that his fixed dilated pupils were noted.

(6) Answer to Question 1 at p.2

"I also contacted Dr Savage to inform him of the situation and he returned immediately from the University"

(a) Describe your conversation with Dr Savage when you informed him of the situation, including:

- **when you spoke to him**
- **what was discussed**

My note tells me that I spoke to Dr Savage sometime between 12.05 and 1pm. I would have discussed Adam's neurological state as he had fixed dilated pupils.

(b) State at what time Dr Savage returned to the RBHSC

I do not recall at what precise time Dr Savage returned to RBHSC but his first note in the chart was made sometime between 1pm and 1.20pm.

(7) Answer to Question 2 at p.2

"My notes confirm that I examined Adam at 12:05pm on 27th November 1995 (Ref: 058-035-135 to Ref: 058-035-137)"

(a) State if you made any of the entries in the medical notes at Ref: 058-035-134

I made the notes on the first half of page 058-035-134. They were made prior to 12.05 pm. They were intended to be the beginning of a summary for my post -operative note and were not signed as I had intended to continue writing on this page.

If so, state the source(s) of the information contained in these notes, including the source(s) of the information that was recorded in Adam's notes in respect of the retrieval of the donor kidney,

time and date thereof

The source of this information was the UK TSSA kidney donor information form which accompanied the kidney (058-009-025, 058-009-027).

(b) State if you made any notes or completed any of the transplant form at Ref: 057-007-008. If so, state:

- **which notes you made**
- **when these notes were made**
- **the source(s) of information for those notes**

I made the notes on the first 3 lines of this transplant investigation summary. The photocopy of this on the Inquiry website is incomplete as it does not include entries on the left side of the page and a full copy has been enclosed (1). These notes were probably made after 4.30 pm on 27/11/95 after the third recorded blood result was available. The first line of results (11 pm 26/11/95) were sourced from the handwritten note in the chart (058-035-144). On reviewing these notes now, for the purpose of answering this question, I think the value for sodium written by Dr O'Neill may have been 139 rather than 134 as I recorded. The second line of results were probably obtained urgently by telephone call from the laboratory to the intensive care unit and were entered into the clinical notes by me sometime after 1.20 pm on 27/11/95 (058-035-138).The third line of results was probably telephoned urgently.

(c) State if you recorded *"vascular anastomosis ~ 10.30am 27/11/95"* in Adam's notes (Ref: 059-006-012)

I made this note in the margin of page 058-035-134 as this information is routinely recorded in order to calculate kidney ischaemic times. This information was written in the margin of the page to remind me of this time when writing formal notes later.

- **or, in the alternative, the individual who made the entries**

(d) If so, state when was the *"vascular anastomosis"* entry made

This entry was made sometime between 10.30 am and 12.05 pm on 27/11/95.

(8) Answer to Question 2 at p.2

"Peri-operative CVP had risen to 30mmHg. My note written at the time states that the CVP was known to be 17mmHg at the start of procedure and in view of this high initial CVP the accuracy of recordings was uncertain"

(a) Describe and explain who considered that the *"accuracy of [the high initial CVP] recordings was uncertain"*, including:

- **any discussions about the *"high initial CVP"* and**
- **who participated in those discussions**

I recall discussing the CVP with Dr Taylor as I had noted a high reading of 30 mmHg peri-operatively. Dr Taylor informed me that the reading had been 17 mmHg at the time of insertion of the line. As this was clinically unlikely in a child who had received overnight dialysis he had presumed the reading to be inaccurate as the position of the line was not certain. Adam's previous multiple line placements had made insertion of the CVP line more

difficult. My note on page 058-035-138 made after viewing the post operative chest X-ray states that I observed the line to be going up the neck vessels. This note is not clearly seen on the Inquiry website as these words in the notes have been highlighted by someone in blue highlighter. A clearer copy is enclosed (2).

(b) Describe and explain the basis of *"the CVP was known to be 17mmHg at the start of procedure and in view of this high initial CVP the accuracy of recordings was uncertain"*

The CVP would be expected to be between 4 and 8 mmHg in a normally hydrated child. As Adam did not appear to Dr Taylor to be clinically fluid overloaded and had not received his full prescribed quota of fluid pre-operatively, he had presumed the initial reading to be incorrect. The reading is affected by the position of the catheter tip. As the initial reading was presumed to be incorrect subsequent readings were also presumed to be incorrect.

(9) Answer to Question 2 at p.2

"I had been informed that there had been difficulty inserting a central venous line at the start of the procedure and I made a presumption that this difficulty was due to previous multiple venous access. I assumed that he may have had one of his external jugular veins tied off as this was common practice at this time of insertion of central lines in RBHSC in 1995. I had not read previous notes at this time to see if this was confirmed. However I felt it likely that the CVP measurement may have been unreliable"

(a) State who told you that there had been a *"difficulty inserting a central venous line at the start of the procedure"* and state when they informed you of such a *"difficulty"*

Dr Taylor informed me that the CVP readings had been around 17 mmHg at the start of the procedure and he thought them to be unreliable. As he had not achieved a reliable CVP line I surmised that line placement had been difficult. I was not present at the time of line placement. Adam had had previous central lines which often makes line placement difficult.

(b) Explain the basis of your assumption that the *"difficulty was due to previous multiple venous access"*

I have often known it to be difficult for a central line to be placed by an anaesthetist if the vessels have been manipulated before as there is scar tissue present and there may be occluded or collateral vessels.

(c) Describe and explain what steps were taken, including by you, to determine whether Adam *"had one of his external jugular veins tied off"*

At that time I took no steps to determine this. I did know from other patients that this was common surgical practice in RBHSC at that time.

(d) State how long it had been *"common practice"* in the RBHSC to tie off an external jugular vein and explain the reason for the practice

I am not competent to answer questions about surgical practice but I was aware of other patients having external jugular veins tied off at the time of line insertion.

(e) Explain why you *"felt it likely that the CVP measurement may have been unreliable"*

The CVP would be expected to be between 4 and 8 mmHg in a normally hydrated child. As Adam did not appear to Dr Taylor to be clinically fluid overloaded and had not received his full prescribed quota of fluid pre-operatively, he had presumed the initial reading to be incorrect. The reading is affected by the position of the catheter tip. As the initial reading was presumed to be incorrect subsequent readings were also presumed to be incorrect.

(10) Answer to Question 2 at p.3

"I have summarised the fluid balance as recorded in the anaesthetic chart as I would do in all cases of renal transplantation. I have recorded that there was 911ml of blood in the suction bottle during theatre presumed to be blood loss and that the total input during theatre consisted of 1 litre of HPPF, 500ml of Hartmann's solution, 1500ml of 0.18% saline 4% dextrose solution and 250ml of packed red cells."

(a) Identify and describe each of the paediatric renal transplant operations in which you had been involved prior to 27th November 1995, including:

- **date and hospital**
- **anaesthetist involved and the fluid management regime employed**
- **surgeon involved**
- **the outcome**

Prior to 26/11/85 I was involved in the acute peri-operative care of 13 children undergoing transplants, 12 in Bristol and 1 in Belfast.

Date	Hospital	Age at Transplant (yrs)
22/07/1994	Southmead Hospital , Bristol	14
26/06/1994	Southmead Hospital , Bristol	10
03/12/1994	Southmead Hospital , Bristol	17
05/12/1994	Southmead Hospital , Bristol	7
18/12/1994	Southmead Hospital , Bristol	2
06/02/1995	Southmead Hospital , Bristol	12
01/04/1995	Southmead Hospital , Bristol	11
16/05/1995	Southmead Hospital , Bristol	10
03/07/1995	Southmead Hospital , Bristol	6
09/07/1995	Southmead Hospital , Bristol	14
03/07/1995	Southmead Hospital , Bristol	6
05/10/1995	Southmead Hospital , Bristol	13
17/11/1995	RBHSC, Belfast	3

I was a Specialist Registrar in Paediatric Nephrology in Southmead Hospital, Bristol from 1st July 1994 to 31st October 1995. I do not have a record of the surgeon or anaesthetist involved in the transplants for the 12 children in whose peri-operative care I was involved. My duties, as a

nephrologist, did not entail being present in theatre but I did attend theatre on one occasion as I was interested to observe the surgery. I have enclosed (3) a copy of the protocol I used in Bristol with regard to drug and fluid management.
The outcome for these 12 Bristol children was 10 successful transplants, 2 transplant nephrectomies due to rejection and thrombosis and no patient deaths.

After taking up my consultant post in RBHSC I cared for a 3 year old child who was transplanted on 17/11/95.The surgeons were Mr Keane and Mr Boston and I have no recollection of which anaesthetist was involved. The outcome was excellent as he is now aged 18 and the transplant is still functioning. I used the fluid management protocol from Bristol with which I was most familiar. I was responsible for the pre-operative and post-operative fluid management and prescription of immunosuppression.

To cite these figures within the whole UK perspective, the 94/95 United Kingdom Transplant Support Service Authority activity report shows that from Jan-Dec 1994 in all of the UK there were a total of 1750 cadaveric kidney transplants and 35 of these recipients were aged 0-5yrs and 40 were aged 6-11 yrs. (extract enclosed (4)).

(b)Describe and explain how Adam's blood loss during his surgery was measured

Blood loss during theatre is measured in a calibrated suction bottle and by weighing of swabs. All fluid from the operation site will be sucked into this bottle so possibly there will also be irrigation fluid contained in the bottle. I have recorded on page 058-035-136 that 911ml of blood was in the suction bottle. I obtained this figure from swab count sheet 058-007-021 where I now observe that the 911mls was recorded as a combination of 500mls in the suction bottle and 411mls in weighed swabs.

(c) Describe any concerns expressed by Dr Taylor during Adam's transplant surgery, including in relation to:

- **blood loss**
- **fluid balance**
- **the action to be taken**

I recall having a discussion with Dr Taylor about the unreliability of the CVP reading. I do not now 15 years later recall if we had any specific conversations about fluid balance during the operation.

(d)Describe and explain how Adam's *"total input during theatre"* was measured
Total input during theatre would have been measured by the anaesthetist recording the volume of all fluids given.

(11)Answer to Question 2 at p.3

"I observed [Adam] to be puffy and recorded that the CVP measurement at the time of my examination was now 11[mmHg]"

(a) Describe and explain what you meant by *"puffy"*, when you made that observation and exactly what you observed as being *"puffy"*

I have recorded in my note at 12.05pm on 27th November 1995 (058-035- 136) that Adam was puffy. This comment has been obscured on the Inquiry website as it has been highlighted by someone in blue highlighter. A clearer copy is enclosed (5). I would have used this word to refer to the appearance of oedema or subcutaneous fluid. I have not recorded where I observed this but the most obvious place to look is usually around the eyes.

(b) Explain the significance of the CVP measurement in the light of your view that *"the CVP measurement may have been unreliable"*

I have recorded on page 058-035-136 that the CVP was now 11. I am not an expert in the measurement of CVP but the patient had been moved from theatre to Intensive Care at this time. Changes of position can affect CVP readings. I subsequently observed on page 058-035-138 that the chest x-ray showed that the CVP line was not in a correct position to measure CVP as it was going up the neck vessels.

(12) Answer to Question 2 at p.3

"I recorded that there had been 49ml of output from his native kidneys from the time of transplantation and that there had been no recorded output to date from the transplanted kidney. It is possible to differentiate the outputs as there was a feeding tube inserted into the transplant ureter and an ordinary bladder catheter inserted separately into his bladder"

(a) Explain how you measured the output from the native kidneys from the time of transplantation

The operation note on page 058-035-135 states that the Surgeon inserted a size 14 Malecot catheter (which is a type of supra-pubic catheter) into the bladder. This would have been connected to a drainage bag and urine from the native kidneys should have drained from the kidneys into the bladder, out the catheter and into the bag. I have recorded on page 058-035-137 that 49ml of urine from the native kidneys had been drained by the time of my note at 12.05pm on 27th November 1995.

(b) Explain how you measured the output from the transplanted kidney

The operation note on page 058-035-135 states that the Surgeon inserted a size 8 feeding tube into the ureter. This would have been done in the later part of the operation and this tube would have been connected to a drainage bag. I would have expected any urine from the transplant kidney to drain through this tube.

(c) State who inserted the bladder catheter, and:

- **when this was inserted**
- **who was responsible for measuring the contents of the catheter**

The operation note by Mr Keane on page 058-035-135 recorded that a Malecot (a type of supra-pubic catheter) was inserted. This does not necessarily mean that Mr Keane did it as two Surgeons were present (also Mr Brown). The catheter would have been inserted in the latter

part of the surgery. When the surgical drapes were removed the catheter would have been connected to a drainage bag and it is the responsibility of nursing staff to measure the contents of this bag on an hourly basis.

(d) State who inserted the donor ureteric catheter, and:

- **when this was inserted**
- **who was responsible for measuring the contents of the catheter**

The operation note by Mr Keane on page 058-035-135 recorded that the donor ureteric catheter was inserted. A size 8 feeding tube was used for this and placed supra-pubically as was common practice. This does not necessarily mean that Mr Keane did it as two Surgeons were present (also Mr Brown). The catheter would have been inserted in the latter part of the surgery. When the surgical drapes were removed the catheter would have been connected to a drainage bag and it is the responsibility of nursing staff to measure the contents of this bag on an hourly basis.

(13) Answer to Question 2 at p.3

"I questioned in my notes whether he had 'coned' due to cerebral oedema and noted that he had high fluid intake and possible abnormal venous drainage. However, it is normal for there to be a very positive fluid balance at the end of a renal transplantation as a high central venous pressure is required in order to perfuse the transplanted kidney adequately"

(a) Explain what you mean by *"he had high fluid intake"*

I have recorded in my note (058-035-136) that the total input of fluid was 3000ml of crystalloid and plasma and 250ml of blood. There is no standard amount of fluid that is given during a kidney transplant as this is individualised for each patient and depends on the size of patient, CVP and blood pressure measurements and the observed perfusion of the transplant kidney. Any fluid deficit present before theatre requires correction before a transplanted kidney is anastomosed. My comment about the high fluid intake was because his intake was 2292mls greater than his measured loss of 911ml plus his estimated insensible losses of 47 mls for 5 hrs (300ml/m^2/day). I did not have any recording of a measurement of his urine output during the operation which would have been on top of the measured and estimated other outputs.He usually passed 1000-2000 mls per day as recorded in my note of 9/11/95- 058-035-143.This note has been obscured on the Inquiry website as it has been highlighted by someone in blue. A cleaner copy has been enclosed (6). It is not routine to measure urine output during a transplant as any pre-operatively inserted bladder catheter is usually kept clamped in order to allow the bladder to fill.

(b) Explain what you mean by *"a very positive fluid balance"* in the context of what you regard as *"normal"* at the *"end of a renal transplantation"*

Any child who has a renal transplant will have a positive balance of fluid at the end of the surgery. This is because extra fluid is required to increase the CVP prior to the anastomosis of the transplant kidney and to perfuse the kidney. Many children who are on peritoneal dialysis are discovered to be fluid deficient after induction of anaesthesia and insertion of a CVP line, and deficits need to be replaced before perfusion of the transplant kidney. It can be very difficult to accurately clinically assess fluid balance in small children on dialysis.

(14) Answer to Question 2 at p.3

"My subsequent notes of approximately 1hr 15 mins later (Inquiry Reference number 058-035-138) state that a chest x-ray had now been obtained showing that the central venous line was seen going up through his neck vessels rather than downward toward the heart and I queried this may have caused some obstruction of venous return"

(a) State with whom you *"queried this may have caused some obstruction of venous return"* and what happened as a result of your query

This query was made in the notes as a record of my thoughts after seeing the chest x-ray and just before Adam went for an emergency CT scan. As a result of my query I was aware that the CVP reading from this line could be considered unreliable.

(b) Describe and explain the basis upon which you thought the central venous line going up through Adam's neck vessels *"may have caused some obstruction of venous return"*

I am not an expert in measuring CVP or in venous anatomy. I surmised that a CVP line going into a narrow bore neck vessel would occupy a bigger percentage of the vessel lumen than it would if it was placed into a larger vessel nearer to the heart.

(c) State, in relation to your note at Ref: 058-035-138, what you meant by your marginal entry *"?dilutional"* and when you made it

My note on page 058-035-138 was made sometime shortly after 1.20pm on 27th November 1995. My habit is often to record blood test results in the margin of a page as they can be clearly seen. The comment "?dilutional" was made as I was trying to find an explanation for the sodium level to have fallen from a value of 134mmol/L (see Q 7(b) as I now believe I misread this handwritten number from page 058-035-144 and that it reads 139) pre-operatively to a value of 119mmol/L post-operatively. Sodium values in the blood depend on the relative amounts of sodium and water present and one possible explanation for low sodium is an excess of water in relation to sodium which I would have described as dilutional.

(15) Answer to Question 2 at p.3

"I have also recorded that the post operative serum sodium was 119mmol/L and I queried that this was due to haemodilution"

(a) State with whom you *"queried that this was due to haemodilution"* and what happened as a result of your query

I queried this myself and I expect that I discussed it with my clinical colleagues including Dr Taylor and Dr Savage. The CT scan and clinical condition at this stage were consistent with irreversible brain damage. Mannitol had already been given to try to decrease cerebral oedema and hyperventilation was in place to reduce carbon dioxide levels which would be beneficial with regards to cerebral oedema. Further IV fluids were restricted to 10ml per hour which mostly would have been made up by Cyclosporin and Dopamine infusions as well as small infusions to keep central and arterial lines open.

(b) Explain what you meant by *"haemodilution"* and how you came to that conclusion

By haemodilution I meant the presence of excess water compared to sodium content in the blood. I surmised this to be possible as the intake during theatre was 2292ml greater than the estimated output (not including peri-operative urine).

(16) Answer to Question 2 at p.3

"I assumed that his normal polyuric state complicated his fluid management and that his possible abnormal cerebral venous drainage may have made him more susceptible to cerebral oedema"

(a) Describe and explain the basis of your assumption that Adam's *"normal polyuric state complicated his fluid management"*, including the way his fluid management would have been rendered more complicated

The most frequent situation when a child goes for a renal transplant is that they produce very little urine from their native kidneys and hence this does not need to be taken into account during the fluid balance peri-operatively. In Adam's case he was known to pass a lot of weakly concentrated urine as his normal fluid intake at home to maintain a steady weight was 2100ml (100ml/kg) and his urine output was estimated at 1-2 litres per day (as in my clinical summary on page 058-035-143).

(b) Describe and explain the mechanism by which *"possible abnormal cerebral venous drainage may have made [Adam] more susceptible to cerebral oedema"*

I am not an expert on cerebral venous drainage and this comment was simply my conjecture. I do not know if possible abnormal cerebral venous drainage could have been a contributing factor in Adam's cerebral oedema. I know it was common practice by some Surgeons in RBHSC at that time to tie off external jugular veins when broviac central lines were inserted.

(17) Answer to Question 2 at p.3

"My main role in the care of Adam was between 12:05pm and approximately 1pm on 27/11/95 when Dr Savage took over his management"

(a) Describe what was entailed in the *"main role"* that you had in the care of Adam *"between 12:05pm and approximately 1pm on 27th November 1995"*

A nephrologist is generally responsible for the immediate post-operative care of a transplant patient. When a kidney functions well there is usually a very large urine output in the first 6-24hrs and it is very important that the fluid management is meticulous at this time. The complicated drug regime which is required for immunosuppression post-transplantation is also the responsibility of the nephrologist. I would have expected in a normal post-operative period for these issues to have been my main concerns. In Adam's case it was obvious as soon as Dr Taylor attempted to wake him up from the anaesthetic that he had fixed dilated pupils. This suggested very severe brain dysfunction and my priority in his care at this time was to investigate this with the help of neuro-radiology and to attempt to treat cerebral oedema by giving mannitol. I ceased to be the main consultant responsible for Adam's care when, following my phone call to him, Dr Savage returned from the University. As he was Adam's named consultant and had known the family since Adam's birth I felt it was more appropriate

for him to speak with them rather than me.

(b) **Describe and explain by what means Adam's care came under your management *"between 12.05pm and approximately 1pm on 27/11/95"***

The statement means that as the operation was now over I considered that Adam's medical care should be co-ordinated and delivered by the nephrology team with the help of the Intensive Care team. I was the only consultant nephrologist present between 12.05pm and 1pm on 27th November 1995 until Adam's named consultant returned from the university.

(c) **Describe and explain any role that you had in Adam's care and management (as opposed to a *"main role"*) before 12.05pm on 27th November 1995 and/or after 1.00pm on 27th November 1995**

I arrived at work at approximately 9am on 27th November 1995 and was informed by Dr Savage that Adam was in theatre having a transplant. I liaised with Dr Taylor regarding Adam's immunosuppression during theatre (which had been already planned by Dr Savage) and went into theatre several times as I was curious about the progression of the transplant. In my training as a paediatric nephrologist my role had always been to be immediately available in the recovery ward to prescribe post-operative fluids and drugs and I considered this to be my responsibility.

After 1pm on 27th November 1995 I was present in the Intensive Care Unit along with Dr Savage. I made notes on pages 058-035-137-9 with regard to the management of Adam's drugs and blood pressure. I am unsure which nephrologist was on-call overnight on 27th November 1995 but note that Dr Savage wrote in the notes at 8.30pm and 11pm so I surmise that he was involved in Adam's care overnight. The notes on page 058-035-142 on 28th November 1995 at 9.10am made by Dr Webb state that brain stem death criteria were examined by Dr Webb and myself.

II QUERIES ARISING OUT OF YOUR PSNI STATEMENT

With reference to your PSNI Statement dated 12th April 2006, please provide clarification and/or further information in respect of the following:

(18) ***"After the operation I discussed the CVP figures with Dr. Taylor and noted the initial reading of 17 mmHg was high and the later reading of 30 mmHg was very high but the conclusion was that these readings may have been unreliable"***

(a) **State when and where that discussion took place**

I think that this conversation took place in theatre rather than the intensive care unit but do not recall precise timings. I recorded details about the CVP readings at 12.05pm on 27th November 1995 on page 058-035-135

(b) **Describe what was discussed and the process by which the conclusion was reached *"that these readings may have been unreliable"***

I do not recall the precise details of the discussion. However the CVP would be expected to be between 4 and 8 mmHg in a normally hydrated child. As Adam did not appear to Dr Taylor to be clinically fluid overloaded and had not received his full prescribed quota of fluid pre-operatively, he had presumed the initial reading to be incorrect. The reading is affected by the position of the catheter tip. As the initial reading was presumed to be incorrect subsequent readings were also presumed to be incorrect.

(19) *"I have recorded that the kidney was 'bluish' at the end of theatre. This would have made me anxious to observe the urine output over the next few days, however, in my experience kidneys which have appeared bluish at the end of theatre have later proved to be satisfactory after sometimes as much as 3 weeks, in that the recipient did not need to go back on dialysis"*

(a) State whether the bluish colour of the donor kidney at the end of theatre was discussed in theatre, including, the identity of those who discussed it and the result of those discussions

I do not recall whether I was told that the kidney was bluish or whether I observed this. I note that the operation note by Mr Keane on page 058-035-135 states that the kidney was perfused "reasonably" at the end of the procedure. In a successful kidney transplant the Surgeon usually describes the kidney as being well perfused. I do not recall the detail of any discussions.

III ADDITIONAL INFORMATION

(20) **Describe in detail the education and training that you received in fluid management (in particular hyponatraemia) and record keeping through the following, providing dates and names of the institutions/bodies:**

(a) Undergraduate education

I obtained the degree of MB, BCh, B.A.O. with commendation in Medicine and Surgery from Queen's University in Belfast in 1984 and won the finals Gold Medal prize in Paediatrics in 1984. I do not recall the details of my training with particular reference to fluid management or hyponatraemia but these subjects would have been addressed during my lectures and clinical placements. The issue of record keeping would have been addressed in my lectures, particularly in the undergraduate course in forensic medicine in 1983.

(b) Postgraduate education and training

Junior House Officer 1984-1985.
Royal Victoria Hospital including three months in Children's Hospital. I was responsible for the prescription of fluids and the recording of notes for all my patients.

Senior House Officer 1985-1987.
In these years I spent one year in Adult medicine and two years in Paediatrics during which time my routine responsibilities would have included the prescribing of fluids and recording of records. I was provided in 1985 with the RBHSC in-house manual "Paediatric Prescriber" which contained guidance about fluid and electrolyte replacement for children on pages 71-74 (copy

enclosed (7)). I attended all post-graduate lectures in the Hospital and during this time obtained the qualification MRCP. In my studies for this exam I would have studied fluid and electrolyte management and would have been guided by the standard of text books of the day including "A Paediatric Vade-Mecum, 11th Edition,1986, Insley, Jack." which had guidance about fluid and electrolyte therapy on pages 53-67(copy enclosed (8)).

Registrar and Senior Registrar in Paediatrics 1988-1992.
My responsibilities in these jobs would continue to have involved note keeping and prescription of fluids in a variety of clinical situations. I would have attended regular post-graduate meetings.

Senior Registrar in Paediatric Nephrology 1992-1994.
During this time I was involved in the acute care of 12 renal transplant children in the pre-operative and immediately post-operative phases. I was involved in specialist consults in Southmead Hospital, Bristol with regard to electrolyte disturbances. I attended post-graduate meetings including the Great Ormond Street post-graduate renal week and the British Paediatric Association College meeting. I attended the meeting of the European Society For Paediatric Nephrology in Amsterdam.
I successfully completed the APLS (Advanced Paediatric Life Support) Course in Bristol November 1994. The Manual for APLS, published by the BMA 1993, reprinted 1994 deals with fluid and electrolyte balance, including hyponatraemia on pages 221-232 (copy enclosed (9)).

(c) Hospital induction programmes

During my post-graduate career Hospital induction programmes were not available in any of the Hospitals in which I worked. I have since been involved in delivering part of the Hospital induction programme in RBHSC.

(d) Continuous professional development

I have written several protocols for the management of pre and post-operative care for Paediatric renal patients between 1996 and 2011.

I have read extensive literature in Nephrology and Paediatric journals relating to hyponatraemia.

I have been involved in a working group in RBHSC with regard to designing a wall chart and giving advice about fluid prescription and a new fluid balance chart for ward use.

I have completed the British Medical Association learning module on hyponatraemia in children.

I have attended the European Society for Paediatric Nephrology meeting on alternate years since 1995.

I have attended the British Association for Paediatric Nephrology and Royal College of Paediatric Meetings regularly.

I have attended the Renal Association Advanced Nephrology Course in 2010 including the

sessions on electrolyte imbalance, hyponatraemia and fluid management.

(21) Prior to 26th November 1995, describe in detail your experience of children:

(a) With hyponatraemia, including:

- **the estimated total number of such cases, together with the dates and where they took place**
- **the number of the children who were aged less than 6 years old**
- **the nature of your involvement**
- **the outcome for the children**

In my role as a Paediatric and Renal Trainee from August 1986 until October 1995 I regularly prescribed fluids for children of all ages. I would have encountered many cases of moderate hyponatraemia and some cases of severe hyponatraemia (particularly in infants with post obstructive diuresis and sepsis). I do not know the numbers of those children and do not recall any adverse outcomes due to hyponatraemia..

(b)Undergoing renal transplants, including:

- **the number of all such transplants, together with the date of each and where they took place**
- **the number of the children who were aged less than 6 years old**
- **the nature of your involvement**

Prior to 26/11/85 I was involved in the acute peri-operative care of 13 children undergoing transplants, 12 in Bristol and 1 in Belfast. Two of these children were aged less than 6 years old.

I was a Specialist Registrar in Paediatric Nephrology in Southmead Hospital, Bristol from 1st July 1994 to 31st October 1995. The nature of my involvement was to organise the pre-operative care and investigation of transplant patients under supervision of my consultants and to monitor post-operative clinical parameters for these patients and prescribe appropriate fluids and drugs. My duties did not entail being present in theatre but I did attend theatre on one occasion as I was interested to observe the surgery.

	Date	Where	Age at Transplant (yrs)
1	22/07/1994	Southmead Hospital , Bristol	14
2	26/06/1994	Southmead Hospital , Bristol	10
3	03/12/1994	Southmead Hospital , Bristol	17
4	05/12/1994	Southmead Hospital , Bristol	7
5	18/12/1994	Southmead Hospital , Bristol	2
6	06/02/1995	Southmead Hospital , Bristol	12
7	01/04/1995	Southmead Hospital , Bristol	11
8	16/05/1995	Southmead Hospital , Bristol	10
9	03/07/1995	Southmead Hospital , Bristol	6
10	09/07/1995	Southmead Hospital , Bristol	14
11	03/07/1995	Southmead Hospital , Bristol	6
12	05/10/1995	Southmead Hospital , Bristol	13
13	17/11/1995	RBHSC, Belfast	3

From the time of my appointment as a consultant on 1/11/95 until 27/11/95 I was responsible for the supervision of pre-operative and post-operative care of one 3 year old child who was transplanted on 17/11/95 in RBHSC.

To cite these figures within the whole UK perspective, the 94/95 United Kingdom Transplant Support Service Authority activity report shows that from Jan-Dec 1994 in all of the UK there were a total of 1750 cadaveric kidney transplants and 35 of these recipients were aged 0-5yrs and 40 were aged 6-11 yrs (enclosure (4)).

(22) Since 27 November 1995, describe in detail your experience of children:

(a) With hyponatraemia, including:

- **the estimated total number of such cases, together with the dates and where they took place**
- **the number of the children who were aged less than 6 years old**
- **the nature of your involvement**
- **the outcome for the children**

Since 27th November 1995 I have often been consulted about fluid management for patients in RBHSC with hyponatraemia and have advised about their treatment. I do not recall the numbers or ages of these children and do not recall any adverse outcomes.

(b)Undergoing renal transplants, including:

- **the number of all such transplants, together with the date of each and where they took place**
- **the number of the children who were aged less than 6 years old**
- **the nature of your involvement**

Since 27th November 1995 there have been 62 patients from RBHSC transplanted. Ten of these transplants took place in Belfast City Hospital (older children) and 2 took place in Great Ormond Street Hospital, London (due to complex extra renal problems). This means that 50 transplants took place in RBHSC. I was involved in the immediate peri-operative care of 36 of these patients. I would also have been involved in the care in the immediate few days post-transplant in most of the others. The ages and dates of transplant of these 62 patients are listed below. Ages range from 2 years to 17 years and 10 children were aged less than 6 years old. Of the 10 children aged less than 6 years old, 9 were transplanted in RBHSC and 1 in Great Ormond Street Hospital.

The outcome for these 62 children was: 57 successful transplants; 3 early transplant nephrectomies due to graft thrombosis (two in BCH, patients aged 15yrs and 17yrs and one in RBHSC, patient aged 14yrs); 1 kidney removed at time of transplant due to poor perfusion (RBHSC, patient aged 15yrs); and 1 death at 6 weeks post-transplantation in a 16 year old (with complex medical problems) due to gut problems and haemolytic- uraemic syndrome causing bleeding (RBHSC).

The nature of my involvement in these children's care would have been to supervise and

organise the pre-transplant investigations, the peri-transplant immunosuppression and the post-operative care for these children. In most cases since 27th November 1995 I was present in theatre during the operation to observe and discuss fluid management with the anaesthetist, if my other clinical commitments allowed.

NO	DATE TX	HOSPITAL	AGE AT TRANSPLANT (YRS)
1	27/02/1996	RBHSC	4.0
2	07/04/1996	BCH	10.1
3	16/06/1996	RBHSC	7.2
4	21/06/1996	BCH	16.8
5	04/08/1996	BCH	15.5
6	14/08/1996	BCH	17.2
7	28/08/1996	RBHSC	11.0
8	24/07/1996	RBHSC	6.0
9	09/11/1996	RBHSC	9.4
10	26/05/1997	BCH	14.0
11	29/07/1997	RBHSC	3.9
12	01/08/1997	RBHSC	12.2
13	27/12/1997	RBHSC	5.7
14	22/02/1998	RBHSC	13.5
15	22/04/1998	RBHSC	14.7
16	09/02/1999	BCH	12.9
17	19/03/1999	RBHSC	10.1
18	26/04/1999	RBHSC	11.2
19	15/09/1999	RBHSC	2.1
20	31/03/2000	RBHSC	11.3
21	17/7/ 2000	RBHSC	14.2
22	10/11/2000	RBHSC	16.6
23	19/02/2001	BCH	15.8
24	03/03/2001	RBHSC	15.9
25	04/02/2002	RBHSC	4.7
26	21/03/2002	RBHSC	4.7
27	20/10/2002	RBHSC	16.4
28	20/11/2002	RBHSC	11.5
29	21/04/2003	RBHSC	13.1
30	25/10/2003	RBHSC	15.8
31	01/07/2004	RBHSC	7.2
32	31/07/2004	RBHSC	13.1
33	12/01/2005	RBHSC	14.6
34	19/01/2005	RBHSC	5.4
35	23/02/2005	RBHSC	12.3
36	26/09/2005	RBHSC	16.9
37	03/11/2006	RBHSC	12.6
38	10/04/2007	BCH	17.2
39	08/08/2007	RBHSC	14.2
40	23/12/2007	RBHSC	6.0
41	14/04/2008	RBHSC	11.1
42	11/05/2008	RBHSC	12.0

NO	DATE TX	HOSPITAL	AGE AT TRANSPLANT (YRS)
43	11/11/2008	BCH	17.6
44	01/11/2008	GOSH	17.6
45	20/12/2008	BCH	16.8
46	06/02/2009	RBHSC	15.9
47	15/06/2009	RBHSC	12.6
48	31/08/2009	RBHSC	15.1
49	22/12/2009	RBHSC	14.3
50	04/01/2010	RBHSC	6.9
51	04/02/2010	RBHSC	13.7
52	15/02/2010	RBHSC	5.0
53	22/03/2010	RBHSC	6.1
54	02/05/2010	RBHSC	12.6
55	18/05/2010	GOSH	4.4
56	07/07/2010	RBHSC	12.8
57	06/09/2010	RBHSC	14.2
58	22/09/2010	RBHSC	14.9
59	13/10/2010	RBHSC	13.3
60	17/11/2010	RBHSC	14.8
61	03/12/2010	RBHSC	3.8
62	11/03/2011	RBHSC	7.1

(23) Identify any 'Protocols' and/or 'Guidelines' which governed Adam's renal transplant surgery

Adam's surgery was governed by the guideline "Renal Transplantation in Small Children" written by Dr Savage in September 1990. His immunosuppression was influenced by the protocol I had recently (1/11/95) brought from Bristol (enclosed (3)).

(24) The 'RBHSC Renal Transplant Guidelines' *"updated Sept 96"* bears your name along with that of Dr. Savage, describe and explain your role in the development of those *"updated"* Guidelines and how (if at all) they were influenced by lessons learned from Adam's case, together with the function of the 'Renal Transplant Guidelines' at the RBHSC

Dr Savage and I produced these 1996 guidelines after consulting together. In producing these guidelines I also referred to the Southmead Hospital Bristol guidelines which I was previously familiar with and also read several guidelines from other regional Paediatric centres in the UK. I also researched up to date relevant literature regarding immunosuppression and management of paediatric transplantation.

These 1996 guidelines were influenced by lessons learned by Adam's case with regard to hyponatraemia. These guidelines are very prescriptive about frequent checking of serum electrolytes, both immediately pre-operatively and two hourly during surgery. Peri-operative fluid is advised to consist only of 0.9% saline, HPPF (which is isotonic) or rarely blood.

The function of these guidelines is to provide clinicians with a convenient summary of the pre-operative investigations and management needed for a transplant child and to document the appropriate doses of drugs for use in the peri-operative period and beyond. The guidelines give

very prescriptive guidance about the use of fluids and their type in the peri-transplant period. Each child has a different clinical history and may require individual adjustment of these guidelines according to their individual need.

(25) Describe the differences between the 'RBHSC Renal Transplant Guidelines' and their predecessor and explain the reasons for them, including the basis upon which the provision *"D/W Consultant if Na<133. Repeat U/E at time of going to Theatre"* was included in the *"updated"* Guidelines of September 1996

The guideline of 1996 is much longer and more detailed than the 1990 guideline (nine pages instead of four). There is a longer section on pre-operative planning including instructions about pre-operative fluids and the necessity to check electrolytes before theatre if fluids have been given. The instruction to *"D/W Consultant if Na<133. Repeat U/E at time of going to Theatre"* was included in the protocol as a direct consequence of Adam's hyponatraemia and tragic death. This advice was to ensure that the consultant was aware of any hyponatraemia prior to transplant.

The drugs used for immunosuppression are different in the 1996 protocol. In 1996 methylprednisolone is given intraoperatively and azathioprine and cyclosporin pre-operatively whereas in 1990 the instruction was for hydrocortisone and azathioprine to be given intraoperatively.

The 1996 protocol gives instructions to check arterial blood gases and electrolyte 2 hourly in theatre and also to use only normal saline, plasma or blood to raise the CVP to 8-10mmHg prior to removal of vascular clamps. The 1990 protocol instructs the use of blood, PPF or N/2 saline to ensure a good intravascular volume as determined by reference to BP and CVP levels.

The 1996 protocol recommends the use of 0.45% saline, 2.5% Dextrose as urine and insensible loss replacement (300ml/m^2/day) and makes no mention of the use of 0.18% saline, 4% Dextrose.

The 1996 protocol gives more detailed instructions regarding post-transplant immunosuppression, assessment and treatment of rejection than the 1990 protocol.

The 1996 protocol also includes a pre-operative theatre checklist which requests pre-operative blood results and calculates peri-operative drug doses and a nursing checklist which records some pre-operative measurements and administration of drugs.

(26) Explain the process of and mechanism for the revision of such protocols at the RBHSC

The revision of a transplant protocol involves researching any up to date literature about new drugs or management and a discussion between the team of Consultants. In a small speciality like Paediatric Nephrology we also obtain protocols from other units in the UK to compare with our own practice before updating our own protocol. The RBHSC protocol was updated again in 2004 and 2011 (enclosed (10)).

(27) Identify and describe any standards or guidelines governing dialysis that were operating at the time of Adam's dialysis on 26th November 1995 and those currently in operation

The standards or guidelines relating to dialysis prior to 26th November 1995 would have related to the old fashioned PacX machine which is no longer in use and hence I do not have a copy of protocols related to this machine. The current guidelines regarding peritoneal dialysis, dialysis prescription sheets and instructions for beginning and ending dialysis are enclosed. We use The British Association for Paediatric Nephrology guidance for peritoneal dialysis which was produced in 2007 enclosed (11). This is also available on the British Association for Paediatric Nephrology website << http://www.bapn.org/clinical_standards.html>>.

(28) Identify precisely on Adam's medical notes and records the entries that you made or which were made on your direction and state below:

(a) When each of the identified entries was made

(b) The source of the information recorded in the entry

Entry 055-053-108
This entry was made on 10th February 1993. At that time I was the Senior Registrar working in Musgrave Ward. The note recorded my examination of Adam when he attended the Ward with a pyrexic illness.

Entry 055-053-112
On 12th February 1993 (date erroneously written as 12.02.92) I recorded that Adam was clinically well on his antibiotics and his urine culture showed no growth. This information would have been from the laboratory results. On 14th February 1993 I recorded that his temperature was normal and that he had had five days of antibiotics and was going home for the day.

Entry 055-053-115
On 29th March 1993 and 30th March 1993 I have recorded a note about Adam's clinical examination and urine results and have recorded that Mr Boston was requested to see Adam.

Entry 055-053-119
On 22nd April 1993 I have recorded a clinical examination and urine culture result for Adam. Urine culture result would have been from the laboratory.

Entry 055-053-120
On 20th April 1993 I have recorded a clinical note and a plan about a potential gastrostomy tube insertion. I have also recorded a list of blood tests to be performed.

Entry 055-053-121
On 23rd April 1993 to 25th April 1993 I have recorded clinical examination notes and the result of a pyelogram x-ray which would have been obtained from the x-ray department and the result of the urine culture which would have been obtained from the lab.

Entry 058-035-143 and 058-035-144
These pages are numbered out of sequence on the Inquiry website and should be between 058-035-130 and 131. On 9th November 1995 I wrote a summary of Adam's condition for my own benefit. This was shortly after I was appointed as Consultant and was getting to know a lot of new patients. The source

of the information would have been review of his notes. I am unsure whether I was present at the clinic appointment later that day when he was seen by Dr Savage. It is quite likely that I was in an effort to get to know him. A clinic letter from that day has been written by Dr Savage (058-017-047).

Entry 058-135-134
This entry was made on 27th November 1995 sometime between 9am and 10.30am. The information is a summary of information I would have obtained from the UK Transplant Support Service Authority Form which would have accompanied the donor organ. This form is referenced at 058-009-025 and 058-009-027. I made a note in the margin of page 058-035-134 to say that the vascular anastomosis had been performed at 10.30am. I would have been given this information in theatre and I do not remember if I was actually present at that time or not.

Entry 058-035-135, 136, 137.
On these pages at 12.05pm on 27th November 1995 this entry records the fact that Adam did not breathe post-operatively and had fixed dilated pupils. This information would have been given to me by Dr Taylor. I then record a summary of blood pressure and CVP readings and make a note about the epidural and the drugs given in theatre. On pages 058-035-136 and 058-035-137, I recorded fluid balance in theatre. I would have obtained the input information either from the anaesthetic summary chart or from conversation with Dr Taylor. I obtained the loss of 911 mls from the swab count sheet 058-007-021 (where I now observe that the 911mls was recorded as a combination of 500mls in the suction bottle and 411mls in weighed swabs).
I also recorded that the kidney looked bluish at the end of theatre. I think this is my own observation but I cannot recall now whether I observed this directly or was informed of this by a colleague in theatre.
I then record my clinical examination. I record the post-operative urine output which would have been observed by me by looking at the catheter bags. My management plan is recorded.
A note in the margin shows a blood count result which would have been obtained from the laboratory by telephone.

Entry 058-035-138, 139
On this page on 27th November 1995 I have recorded at 1.20 pm that diazepam was given in case Adam's high blood pressure represented seizure activity. I record that the chest x-ray showed the CVP line was going up the neck vessels. This information would have been obtained by looking at the x-ray which was performed in the Intensive Care Unit. I have also recorded in the margin of this page blood results stating that the serum sodium was 119 and that I surmised that this might have been due to haemodilution. This is my own query based on the blood results. I have recorded on that page that Dr Webb, Neurologist would be available to see Adam at 7pm. This information would have been obtained by a phone call. My recollection is that Dr Webb was doing a clinic in Altnagelvin during the day time. On page 058-035-139 at 17.10hrs on 27th November 1995 I have recorded a clinical examination, blood pressure readings for Adam and the source of this was the results of my examination of him at that time.

(29) Provide any further points and comments that you wish to make, together with any documents, in relation to:

(a) The care and treatment of Adam from his admission for the renal transplant surgery on 26th November 1995 to his death on 28th November 1995

(b) Record keeping

(c) Communications with Adam's family about his care and treatment in respect of the renal transplant surgery

(d) Lessons learned from Adam's death and how that has affected your practice

Adam's tragic death is deeply regretted by the clinical team at RBHSC and we have tried to learn from it. Following Adam's death Dr Savage and I revised the RBHSC guidelines for Paediatric Renal Transplantation. These guidelines are very prescriptive about frequent checking of serum electrolytes, both immediately pre-operatively and two hourly during surgery. Peri-operative fluid is advised to consist only of 0.9% saline, HPPF (which is isotonic) or rarely blood. Although it is not standard practice for a Nephrologist to be present in theatre for a renal transplant in most of the UK units, I now make it my practice to be present in theatre as much as possible and to have frequent discussions with the anaesthetist with regard to CVP, fluid replacement and electrolyte results. In recent years the use of 2.7% saline for the emergency treatment of symptomatic hyponatraemia has become standard practice in the UK. This treatment is advised on our RBHSC wall charts for fluid management in children and we have ensured that 2.7% saline is readily available in all wards. I have continued to read up to date paediatric literature regarding fluid management in children. In cases where the pre-operative CVP was thought by the anaesthetist to be unreliable I would request that a pre-operative x-ray be done to check line position.

(e) Current 'protocols' and procedures

Most recent revision of Transplant Protocol 2011 enclosed (10).
BAPN guidelines for peritoneal dialysis enclosed (11).
Prescription and training sheets for peritoneal dialysis enclosed (12).

(f) Any other relevant matter

List of All Enclosures with Statement 014/2 from Mary O'Connor

(1) Transplant form at Ref: 057-007-008 – clearer copy as referenced in Q 7(b).

(2) Clearer copy of page 058-035-138 as referenced in Q 8 (a).

(3) Southmead Hospital Bristol Transplant Protocol as referenced in Q 10 (a).

(4) 94/95 United Kingdom Transplant Support Service Authority activity report page 32 as referenced in Q 10 (a) and 21 (b).

(5) Clearer copy of page 058-035- 136 as referenced in Q 11 (a).

(6) Clearer copy of page 058-035-143 as referenced in Q 13 (a).

(7) Paediatric Prescriber 1985, pages 71-74, as referenced in Q 20 (b).

(8) A Paediatric Vade-Mecum, 11th Edition,1986, Insley, Jack, (ISBN 0-85324-206-2) pages 53-67 as referenced in Q 20(b).

(9) Advanced Paediatric Life Support Manual, published by the BMA 1993, reprinted 1994 (ISBN 0- 7279-0792-1) pages 221-232 as referenced in Q 20 (b).

(10) Most recent revision of RBHSC Transplant Protocol 2011 .

(11) The British Association for Paediatric Nephrology 2007 guidance for peritoneal dialysis as referenced in Q 27 .This is also available on the British Association for Paediatric Nephrology website <<http://www.bapn.org/clinical_standards.html>>.

(12) Prescription and training sheets for peritoneal dialysis.

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

Signed: Mary G O'Connor **Dated:** 11/4/11

Seán T Craig, James W Shaw, Christopher S Ritchie, Amanda Wylie

Your ref Unknown
Our ref 11/AVW/M935-703945 (AVW)

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Date
12 March 2012

Ms Anne Dillon
Solicitor to the Inquiry
The Inquiry into Hyponatraemia-related Deaths
Arthur House
41 Arthur Street
Belfast
BT1 4GB

Dear Madam

HYPONATRAEMIA INQUIRY - DR MARY O'CONNOR
INVESTIGATION INTO DEATH OF ADAM STRAIN

We write with reference to Dr O'Connor's witness statement (document WS-014/2).

At page 24 of the witness statement, Dr O'Connor has provided a response to the following question:

(28) Identify precisely on Adam's medical notes and records the entries that you made or which were made at your direction and state below:

(a) when each of the identified entries was made

(b) the source of the information recorded in each entry.

Having reviewed the medical notes and records again in detail, it has now come to Dr O'Connor's attention that there are two further entries which were made by her. Accordingly, the following two entries should have been included into her response:

Kennedys

"Entry 055-053-122

On 5 May 1993 I have recorded a clinical note on a ward round stating that Adam was having a micturating cystogram X Ray (MCU) that day, was to have three doses of antibiotic after it (ceftaz), and was to go home that day with renal clinic review in three weeks.

Entry 055-015-021

On the evening of 27 November 1995 I have written a prescription for dialysis to be performed for Adam in the intensive care unit. This is dated 27/11/95."

We apologise for any inconvenience caused by this oversight and trust that you will amend your records accordingly.

Yours faithfully

Kennedys

Kennedys

TRANSPLANT No.

(8)

Date			Ht	Weight Kgs.	Max. B/P		Urine Studies						Blood Chemistry (mmol/l)										Haematology						XR's scan IVP etc.	CyA levels	Immuno-suppressive therapy				Other Medication and Comments
Day	Month	Year			Systolic	Diastolic		Sodium	Protein mg/hr/m^2	Creatinine ml/min/1.73m^2	Pred. GFR	M.S.U.	Creatinine	Urea	Na	K	HCO_3	Cl	Ca	PO_4	Alk P		hb.	PCV	wbc.	Differential N.	Differential L.	Platelets				CyA	ALG	Azathioprine	Steroids	
26	11	95	11pm	PS CP									702	16.8	134	3.6			2.54	1.28			10.5		9.3			336					✓	✓	Theatre 200mg MP 250mg AZA
27	11	95	1pm	PS CP									477	11.5	119	4.8	21.4						10.6	29	8.7			140	CTbrain =cerebral oedema						CyA 3mg/kg/hr
28	11	95	4.30pm										467	12.4	124	5.0																		
27	11	95	10pm											14	120	6.0																		
28	11	95	4am										539	15	121	6.4							14.4	40	9.75			252						
28	11	95	8am										545	16.4	125	6.3																		

DATE	CLINICAL HISTORY, EXAMINATION AND PROGRESS
12 pm	Response needs anti hypertensive Rx — BP now 170/100 S/L nifedipine 5mg. stat.
12 pm	diazemuls 6mg IV. given in case ↑ BP = seizure — no effect
	CXR — CVP line going up neck vessel ? obstructing venous return.
Na 149	27/11/95
K 4.8	CT Rankin
Co2 21.4	
Urea 11.5	There is marked generalized cerebral swelling with compression
TP 41	of the lateral ventricles and obliteration of the third & fourth
Creat 477	ventricles, basal cisterns and cortical sulci. No focal
? dehydrated	abnormality seen.
	Dr Chinn
	Neuroradiology Registrar.
27/11/95	Postion & prognosis fully explained to mother + relatives. Explained Cerebral oedema 'brain swelling' with pressure on vital control centres. Have explained we will try to reduce swelling but that hope of recovery is remote. I have said we will continually reassess situation + make further decisions after 24 hrs
	Dr Webb — Neurology will come this pm ~ 7pm re

Enclosure (3)

GUIDELINES FOR MANAGEMENT OF CHILDREN (IE ALL PATIENTS FROM JOHN MILTON/ VICTOR NEALE) UNDERGOING RENAL TRANSPLANTATION

UK Transplant or the transplant co-ordinator will phone with the offer of a kidney. Often the Consultant Paediatrician is consulted directly. If the call comes to a junior medical member of staff then it is important to talk to Dr McGraw, Dr Chambers, Dr Tizard or the Senior Registrar immediately as usually only half an hour is available to accept the kidney.

ONCE KIDNEY IS ACCEPTED

Inform transplant co-ordinator if he/she has not already been informed.

Other individuals to be informed are as follows. The transplant co-ordinator may do this although it is important for the member of the junior medical staff to establish exactly who is to make which phone calls.

i Phone UK Transplant and arrange for kidney to be sent to Bristol if harvested from elsewhere.

ii Phone the patient and arrange for them to arrive as soon as possible.

iii Check with tissue typing if a recent serum is available for cross-matching. If there is a very recent specimen and the patient has not been transfused since, this specimen may be used for the direct cross-match which may then commence as soon as the kidney arrives. If a fresh sample is needed from the patient it is vital to take this immediately he/she arrives as the cross-match takes eight hours and delay postpones the operation.

iv Inform also:

a Ward and nursing staff

b Transplant surgeon

c Theatre and make provisional booking

d First on-call anaesthetist and ask him/her to contact Senior Registrar and/or Consultant on-call anaesthetist

ONCE PATIENT ARRIVES

1 Take blood samples before anything else. It is important that the 5 mls of clotted blood for tissue typing (and any other specimens the tissue typing laboratory require) are sent immediately on arrival of the patient. #20w citrate

In addition bloods need to be taken for full blood count, coagulation screen, renal SMAC (with U&E, creatinine, calcium and albumin to be available at the time of transplantation. Other results may be available later), serum for virology (baseline CMV titres) and cross-match 4 units of blood (2 packed cells 2 whole blood).

2.....

2 Complete paediatric check list pre-transplant. All aspects must be filled in. Note in particular the need to record target weight, blood pressure and normal urine output and body surface area. This check list must go with patient to theatre.

3 Set up intravenous infusion at maintenance fluids appropriate for size of patient and replacing any deficit on assessing difference between current and target weight.

4 Institute other investigations including chest X-ray, ECG, PD culture, urine culture.

5 If history of hyperkalaemia or potassium of greater than 5 mmol/l

a Fast cycle if on PD

b Give calcium resonium 1 gm/Kg PR if on haemodialysis and discuss with consultant if haemodialysis needed in view of chemistry

6 Talk to parents and obtain consent.

7 Write up immunosuppression to be given in the theatre. For all patients this will be Methylprednisolone 10 mg/kg i.v. 5-10 minutes before vascular clamps are released.

8 Write up instructions for intra-operative blood transfusion on checklist once haemoglobin is known. This will be the number of mls of packed cells required to bring the patients haemoglobin up to 10 gms/dl.

9 Write up appropriate Mannitol dose on checklist (0.5 g/Kg 20% Mannitol ie 2.5 ml/Kg).

10. Write up induction dose of antibiotics iv augmentin 30 mg/kg (max 1.2g).

IN THEATRE

1. Insert CVP and arterial line.

2. Use normal saline, plasma or blood, (as appropriate) to raise CVP to + 8-10 cm H20 (6-8 mm Hg) before the vascular clamps are removed from the donor kidney. Aim to keep CVP at this level for the remainder of the operation. Please always take into account actual and target weights and overall hydration state.

3. Give intra-operative blood transfusion as per checklist instructions.

4. Give intra-operative immunosuppression as per checklist 10 minutes prior to the vascular anastimosis together with Mannitol see checklist for dose).

5. Start dopamine 2-3 ug/kg/min via central line

3.....

IMMEDIATE POST-OPERATIVE CARE

1 Allow nurses to make patient comfortable and connect up the monitoring equipment and lines, which will be as follows:

i Weigh bed

ii Rectal and toe temperature probes

iii Hewlett-Packard ECG, BP and CVP monitor or equivalent connected to CVP and arterial lines

iv Peripheral intravenous infusion(s)

v Urethral catheter

vi Ureteric catheter (in some cases only)

vii PD catheter spigotted and empty (if appropriate)

2 Write up the following drugs:

a Morphine 0.01-0.02 mg/kg/hour (half the patients weight in kg = mg in 50 mls at 2 mls/hour) or

Papaveretum 0.02 to 0.04 mg/kg/hour or fentanyl as per selection protocol

b Methylprednisolone 10 mg/metre^2/day as b.d. dosage. First dose to be given 12 hours post-operatively

c Cyclosporin 6 mg/kg/24 hours written up as continuous infusion 3 mg/kg/12 hourly. This will be converted to Cyclosporin 14 mg/kg/day orally given as b.d. dosage when the patient is tolerating oral fluids

d Ranitidine 2 to 4 mg/kg/day orally (as bd dose)

e Dopamine 2 micrograms/kg/minute - see drug infusion protocol

f Anti-thymocyte globulin (Fresenius) for those on high risk protocol only (ie previous graft or high cytoxic antibodies) 4 mg/kg/day iv. Need to give test dose intravenously first.

g Other drugs such as Hydralazine or Frusemide may be written up when required

h Further antibiotic therapy is discretionary and would be used only if there was a history of recent infectin in either the patient or the donor, or the recipient is known to have vesicoureteric reflux into their native kidneys. This should be discussed with the consultant.

4.....

i Acyclovir Post Renal Transplant

Check CMV status donor and recipient. If donor and recipient negative, no Acyclovir given. If donor positive course given for 12 weeks regardless of recipient status. If donor negative and recipient positive, course given for six weeks.

Dosage * 800 mg po 2-6 hours pre op
* 800 mg po 24 hours post op
Thereafter according to GFR:

> 25 ml/m	6 hourly	* 800 mg po
10-25 ml/m	8 hourly	* 800 mg po
< 10 ml/m	daily	* 800 mo po
dialysis dependent	12 hourly	* 800 mg po

* half dose in under 2 years old

OBSERVATIONS

Pulse, blood pressure, core and peripheral temperature, CVP and urine output should be measured hourly. One should aim to maintain:

i CVP between 8 and 12 cm H20 (6-9 mm Hg)

ii Temperature gap less than or equal to 2 degrees centigrade

iii Blood pressure to be decided on an individual basis within that patients normal target blood pressure guidelines

iv The patient should be weighed daily. It is important to note that weigh bed weights may differ markedly from pre-operative weights and that whilst changes in weigh bed weights are important no significance can be attached to the difference between weights made on different scales

v Optimal urine output to be decided on an individual basis. In polyuric patients this will be around 4 ml/Kg/hour initially falling to 2 ml/Kg/hour when stable. In previously anuric patients far lower urine outputs may be acceptable if A.T.N. has occurred Check transplant troubleshooter for management guidelines on response to falling urine outputs. Otherwise aim for at least 2ml/Kg/hour

FLUIDS

Arterial line 0.9% Heparinised saline at 3 mls/hour with a pressurised bag or 1-3 mls/hour heparinised 0.45% saline with a syringe pump. Take care not to confuse the 3 ml/hr flush device used with a pressurised bag with the 30 ml/hr device used with a syringe pump.

Central line half normal saline/2.5% dextrose plus or minus KCL at rate to replace all losses (sodium and potassium content to be adjusted according to urine electrolytes)

Peripheral line at insensible loss and containing drug infusions

5.....

INVESTIGATIONS

U & E (urine and plasma, glucose and calcium) (6 hourly for 24 hours, 12 hourly for next 24 hours and thereafter 24 hourly)

Full blood count, renal SMAC daily and chest X-ray daily for first two to three days

Urine cytology daily

Cyclosporin levels, urine culture, magnesium, 24 hour urine, creatinine and protein clearance twice weekly. Urine and saliva for CMV once weekly

A doppler ultrasound renal scan should be performed early if there are doubts about initial function and thereafter if there are concerns about deterioration in renal function. The decision for a scan should be made by the Paediatric Consultant and timing discussed with Dr Howarth.

Comments/corrections in writing to

Dr ME McGraw - Consultant Paediatrician and Nephrologist
or
DR D E Holland - Consultant Anaesthetist/Intensive Care

Updated January 1995 ref: A/:glines

Organ Use and Transplant Activity

Kidney Transplantation

Figure 20 shows the recipient age at transplant for the 1750 cadaveric kidney grafts carried out in the UK and Republic of Ireland in 1994.

In 31% of cases the donor and recipient were in the same age group, but 26% of adult recipients (16 years or over) received a kidney from a donor of a higher age band.

79 (75%) of the 106 paediatric recipients grafted in 1994 received a kidney from a paediatric donor (both under 16 years) (Table 13); 68 of these grafts (86%) were achieved through kidney exchange. 101 kidneys from donors under 16 years were used for recipients aged 16 years or over; 65 at the local centre.

14% of recipients transplanted in 1994 had previously received a kidney. This re-graft level was in line with that seen in 1992, following a decline to 13% in 1993.

21 kidney recipients in 1994 were reported as simultaneously receiving a pancreas transplant. In 1993, 17 simultaneous kidney and pancreas transplants were reported to UKTSSA.

Donor and recipient blood groups for the 1750 cadaveric grafts are documented in **Tables 14 and 15.** As in 1993, 7% of group O kidneys were used for non-O group recipients during 1994. In 96% of cases the donor and recipient were of identical blood group. **Table 15** shows that the use of O group kidneys for non-O recipients was mainly a local practice, rather than resulting from kidney exchange (9% of O kidneys used locally, 3% exchanged).

Figure 20

Recipient age at transplant for 1750 cadaveric kidney transplants performed in the UK and Republic of Ireland reported to UKTSSA, 1 January - 31 December 1994

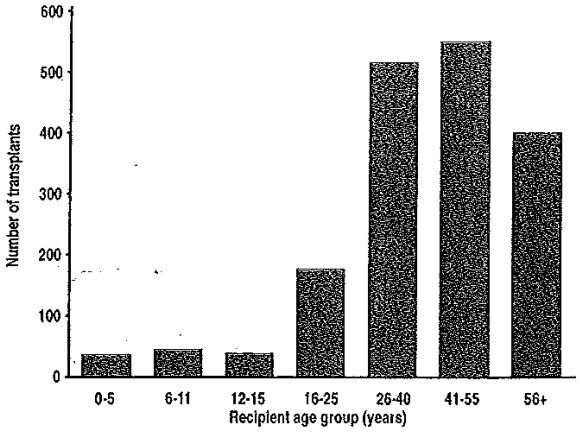


Table 13

Donor and recipient age at transplant in 1750 cadaveric kidney transplants performed in the UK and Republic of Ireland reported to UKTSSA, 1 January - 31 December 1994

Donor Age (years)	Recipient Age (years)						
	0-5	6-11	12-15	16+	**TOTAL**	%	%1993
0-5	2	7	2	10	**21**	1	2
6-11	16	16	9	39	**80**	5	4
12-15	9	7	11	52	**79**	5	4
16+	8	10	9	1543	**1570**	90	90
TOTAL	**35**	**40**	**31**	**1644**	**1750**		
%	2	2	2	94		100	
%1993	1	3	3	93			100

(1993 percentage provided for comparison)

Table 14

Donor and recipient blood group in 1750 cadaveric kidney transplants performed in the UK and Republic of Ireland reported to UKTSSA, 1 January - 31 December 1994

Donor Blood Group	Recipient Blood Group						
	O	A	B	AB	**TOTAL**	%	%1993
O	761	19	34	2	**816**	47	51
A	2	728	0	5	**735**	42	37
B	0	0	136	5	**141**	8	9
AB	0	0	0	56	**56**	3	3
TOTAL	**763**	**747**	**170**	**68**	**1748 ***		
%	44	43	10	4		100	
%1993	47	38	10	4			100

* Recipient blood was not reported for 2 cases

(1993 percentage provided for comparison)

Table 15

Use of blood group O kidneys in cadaveric kidney transplants performed in the UK and Republic of Ireland reported to UKTSSA, 1 January - 31 December 1994

Source	Recipient Blood Group						
	O	A	B	AB	**TOTAL**	%	%1993
Local	501	15	32	1	**549**	67	71
Exchange	260	4	2	1	**267**	33	29
TOTAL	**761**	**19**	**34**	**2**	**816 ***		
%	93	2	4	<1		100	
%1993	93	5	2	<1			100

*Recipient blood group was not reported for 1 case

(1993 percentage provided for comparison)

Contents

Part I

Part II

(136) Enclosure (5)

DATE	CLINICAL HISTORY, EXAMINATION AND PROGRESS

Fluids in theatre:

losses — 911 blood (in suction bottle)

input — 3000 (HPPF 1000, Hartmans 500, N/5 Wacq 1500.
+250 packed cells

KIDNEY — looked 'blush' at end of theatre.

O/E Pupils 7mm, R=L

Kinked

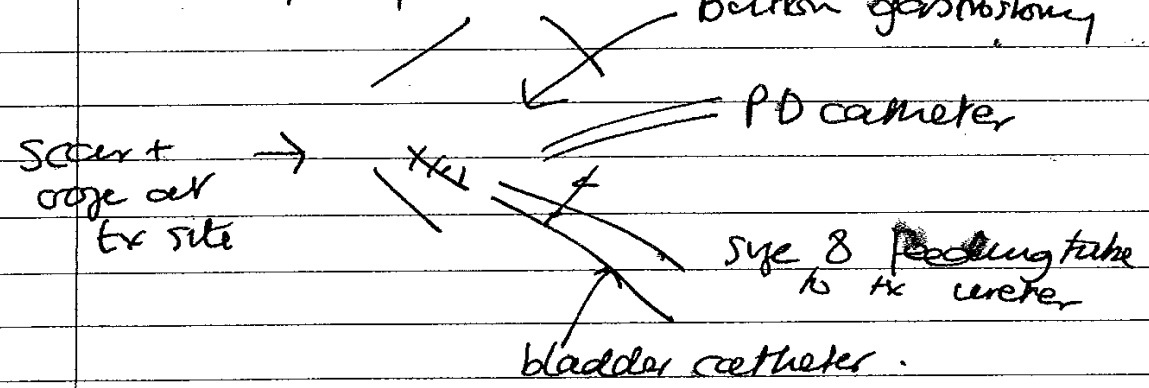
(R) fundus — disc indistinct
haemorrhage @ 12 oc

(L) fundus — disc indistinct.
haemorrhage @ 9 oc, 11 oc

[redacted], CVP = 11,
Peripheral pulses not present (also hand oedema)
Plantars ⊖

Ventilated.

CVS p w7 BP 1°
RS A/E R=L
Abd Soft. BS present.



Enclosure (6). Aadan Strain (143) 102cm 25-50%

DATE	CLINICAL HISTORY, EXAMINATION AND PROGRESS 20.9kg 97%C
9/11/95	Age 4yr 3/12
	PacX dialysis. – since ~~[illegible]~~ Sept 94.
	Reflux nephropathy.
	Bloods ok – ok
	Last pTH Sept – 229 pg/ml
	gastrostomy swab → Pseudomonas, green discharge.
	Drugs: Na bic. 12.5 mls qds (gastrostomy)
	" Ca carbonate 10 mls od (1200mg) (mixed in tube feed) 60mg/kg.
	Reflex 5 ml nocte
	Ferramol 4 ml od
	Id 3 ml od
	Nebivolol ½ daily
	EPO 1000 u x 3 times a week.
	= 100 u/kg/week
	Dialysis: Dry wt 20kg.
	750 ml x 15 cycles. ½ hr dwell.
	13 hrs.
	PM tt ? how much ? 1-2 bags
	feeds gastrostomy – 3x200 boluses
	(Button). 1500 ml O/N.
	? harmony calories.
	1540 kcal = 77 kcal/kg.
	Sucks bread. No food.
	Seen by Geraldine Welford & Adam this week
	Hum seeing alone next week

WNC 762/OS 4043

Enclosure (7)

Paediatric Prescriber

Compiled by: Moira Stewart
Elizabeth Simpson
Dennis Carson

This booklet outlines the first-line drug therapy currently used in The Royal Belfast Hospital for Sick Children. We acknowledge with gratitude the contribution made by the medical staff towards the completion of this booklet and Miss Eileen O'Hare for secretarial assistance.

We wish to thank the following: Dr John Henry, Poisons Unit, New Cross Hospital, London for permission to use the graph "paracetamol plasma levels";
Churchill Livingstone for permission to include the table "rough guide to fluid needs in first 24 hrs of treatment of severe diabetic ketoacidosis" and the "nomogram relating serum salicylate to severity of intoxication following acute ingestion of aspirin", adapted from Dome 1960;
Update Publications Limited for permission to reproduce the graph "plasma paraquat concentration related to time after ingestion" published in Poisoning, Diagnosis and Treatment, 1981, edited by Vale and Meredith.

First Edition 1985

INTRAVENOUS FLUIDS

The initial IV fluid used for normal maintenance requirements is:

(a) 10% dextrose for neonates (5% dextrose if <1000 g).

(b) 0.18% sodium chloride in 4% dextrose for infants and children.

Maintenance fluid volume requirements in 24 hrs:

	In incubator	Under radiant warmers
Day 1	50-80 ml/kg	80-100 ml/kg
Day 2	80-100 ml/kg	100-120 ml/kg
Day 3	100-120 ml/kg	120-140 ml/kg
Day 4	120-150 ml/kg	140-160 ml/kg
Day 5	150 ml/kg	160-180 ml/kg
>5 days	150 ml/kg	200 ml/kg

Maintenance electrolyte requirements in 24 hrs:

Sodium 2-4 mmol/kg (33 ml 0.18% NaCl = 1 mmol)

Potassium 1-3 mmol/kg (1 ml 7.5% KCl = 1 mmol)

Calcium 0.75 mmol/kg (4 ml 10% Calcium-Sandoz = 1 mmol)

VOLUME OF FLUIDS REQUIRED FOR MAINTENANCE AND CORRECTION OF 10% DEHYDRATION IN 24 HRS

Age (yrs)	Expected wt (kg)	Deficit due to 10% dehydration (ml)	Fluid per kg needed for maintenance (ml)	Total volume of fluid for maintenance (ml)	Volume needed for deficit and maintenance (ml)
1	10	1000	120	1200	2200
5	18	1800	100	1800	3600
10	30	3000	75	2250	5250
15	50	5000	50	2500	7500

Additional fluids are required for continuing losses eg vomiting, diarrhoea, polyuria.

Isotonic dehydration is corrected over 24 hrs with 0.18% NaCl in 4% dextrose.

Hypernatraemic dehydration requires careful correction of dehydration over a 24-72 hr period using 0.18% NaCl in 4% dextrose or, in some cases, 0.45% NaCl in 2.5% dextrose.

If the clinical situation allows, rehydration is safer orally compared to the IV route.

PARENTERAL NUTRITION

Parenteral feeding aims to provide children on long-term IV fluids with adequate fluid, calories and protein, as well as vitamins and trace elements. A copy of the regimen used is available on each ward, but responsibility for IV feeding is generally with the TPN (total parenteral nutrition) team.

Protein

This is given as crystalline L-amino acids and the preferred solution is Vamin/Glucose as it contains cystine and histidine (essential in small children).

Carbohydrate

About 60% of calories are provided as glucose, the concentration depending on the site of IV infusion, the patient's requirements and tolerance.

Fat

Intralipid contains a high proportion of essential free fatty acids, and its emulsifier is a naturally occurring phospholipid. 10% solution is used for patients <2 kg or those with jaundice, while 20% solution is used for all others.

Vitamins

Water soluble vitamins are contained in Solivito 0.5 ml/kg to max of 10 ml daily. Fat soluble vitamins are contained in Vitlipid which is put into Intralipid (max of 4 ml daily).

Trace Elements

The currently used solution is Addamel (1 ml/kg/day) which contains Ca, Mg, Fe, Zn, Mn, Cu, F and I.

Electrolytes

Sodium, potassium and phosphate are given in addition to the above; amount added depending on current electrolyte status.

Central Venous Catheters

Broviac catheters are inserted into right atrium via internal jugular vein. They are used for:

- parenteral nutrition
- antibiotics
- chemotherapy

If parenteral nutrition is running, antibiotics and chemotherapy must be given through a separate line.

Great care must be taken of these catheters to protect the patient from infection and air embolism. A detailed schedule is on each ward.

Enclosure (8)

A Paediatric Vade-Mecum

Edited by Jack Insley

M.B., F.R.C.P.E., D.C.H.
Consultant Paediatrician and Clinical Geneticist,
Birmingham Children's Hospital and Birmingham Maternity Hospital

Eleventh Edition

1986

Edward Arnold
A division of Hodder & Stoughton
LONDON MELBOURNE AUCKLAND

IV

Fluid and electrolyte therapy and parenteral nutrition

Fluid and electrolyte therapy

Fluid and electrolyte therapy involves three basic considerations:

1. Provisions of maintenance requirements.
2. Replacement of pre-existing deficits.
3. Correction of continuing losses.

Calculation of maintenance requirements

Maintenance requirements of fluid and electrolytes are proportional to the child's calorie needs. Sick children may require more or less than usual, depending upon their metabolic rates.

TABLE 11 Maintenance I.V. Fluid and Electrolyte Requirements

Body Weight	Kilocalories* /day	Fluid ml/day	Sodium mmol/day	Potassium mmol/day
Less than 10 kg	100/kg	100–120/kg	2.5–3.5/kg	2.5–3.5/kg
10–30 kg	75–100/kg	60–90/kg	2.0–2.5/kg	2.0–2.5/kg
over 30 kg	45–75/kg	40–90/kg	1.5–2.0/kg	1.5–2.0/kg

*1 kcal = 4.2 kJ (kilojoules)

Maintenance fluid and sodium requirements are most conveniently administered as 0.18 per cent sodium chloride and dextrose 4 per cent. The glucose supplied does not satisfy calorie needs but will prevent the development of ketosis if oral feeding has been stopped. *Note that intravenous fluid requirements are less than oral.*

Calculation of pre-existing deficit

In practice, deficits are most easily expressed in terms of body weight, e.g. 5 per cent dehydration in 5 kg baby=250 ml.

Estimation of Dehydration expressed as a percentage of body weight.

mild—5%: decreased skin turgor;
dry mucous membranes.

moderate—10%: increased severity of above signs;
sunken fontanelle;
reduced intra-ocular pressure;
tachycardia, oliguria.

severe—15%: marked increase in severity of above signs; shock,
drowsiness, hypotension.

Types of dehydration Three types are recognised, resulting from variable loss of sodium in relation to water:
hypotonic – serum sodium < 130 mmol/l
isotonic – serum sodium 130–150 mmol/l
hypertonic – serum sodium > 150 mmol/l

The degree of dehydration may be underestimated in hypertonic states, due to increased tissue turgor.

Practical management of dehydration

Hypotonic and isotonic

1. *If the patient is more than 5 per cent dehydrated or is shocked.*
   Give an isotonic solution, such as 0.9 per cent saline, or plasma at 20 ml/kg I.V. over 1–2 hrs, then continue as 2. Monitor B.P. and urine output.
2. *If the patient is less than 5 per cent dehydrated and cannot tolerate oral fluids.*
   Replace the dehydration deficit as calculated as 0.18 per cent saline, or 0.45 per cent saline with dextrose if the serum sodium is 130 mmol/l or less, changing to 0.18 per cent saline in 4 per cent dextrose as the serum electrolytes improve. Add potassium chloride (3 mmol/kg/24 hrs) after urine flow is established and the blood urea is falling towards normal. Once the circulation has been restored it is usually possible to complete rehydration by the oral route (with a rehydration mixture, e.g. Dextrolyte).
   A guide to the volumes needed is found on the next page.
3. Monitor serum electrolytes, and possibly H^+ concentration on admission and after 2, 12, and 24 hours. Record fluid intake and output accurately, and weigh the patient daily.
   I.V. fluids given to infants should always be administered from a graduated chamber to prevent inadvertent sudden fluid overload.

Hypertonic (hypernatraemic) dehydration

Intracellular fluid loss predominates initially so that the classical features of dehydration and circulatory failure develop slowly. The skin is of a doughy consistency and neurological features may be prominent. Most cases result from loss of hypotonic fluid, i.e. greater water than electrolyte losss; however some may result from excess sodium administration. After circulating blood volume has been restored, lower serum sodium slowly over 2–3 days to avoid CNS disturbance. Hypocalcaemia may be present and should be corrected.

TABLE 12 Rough Guide to Rehydration (First 24 hours, excluding newborn) Basic fluid will be 0.18 per cent saline in 4 per cent dextrose.

	HYPOTONIC OR ISOTONIC DEHYDRATION Na<150 mmol/l		HYPERTONIC DEHYDRATION Na>150 mmol/l	
	1. IF shocked or >5% dehydration give initial infusion 20 ml/kg in 1–2 hours of 0.9% saline or plasma. Continue as in 2.		1. Give 20 ml/kg 0.9% saline or plasma in 1–2 hours.	
	2. Unless initial infusion required, START with 0.18% saline 4% dextrose at 150 ml/kg/day		2. Continue with 0.18% saline 4% dextrose at 100 ml/kg/day	
Wt (kg)	*ml/24 hr*	*ml/hr*	*ml/24 hr*	*ml/hr*
2	300	12	200	9
3	450	19	300	13
4	600	25	400	18
5	750	31	500	23
6	950	40	600	27
7	1050	44	700	32
8	1200	50	800	36
9	1350	56	900	41
10	1500	63	1000	45

For additions of bicarbonate or potassium and adjustment of sodium intake, see text.

Management

(1) Rehydration: Despite misleading appearances many infants require initial repletion of the circulating blood volume using plasma or 0.9 per cent saline, 20 ml/kg over 1–2 hours. Thereafter maintenance solutions should be hypotonic, preferably 0.18 per cent saline in 4 per cent dextrose at a rate no faster than 100 ml/kg/24 hour. Once initial rehydration is achieved and circulation restored consider using oral route.
(2) Urine collection: measure output, examine for deposit; if oliguria estimate urinary urea and electrolytes.
(3) If shocked record B.P. Normal values should be consistently obtained following initial infusion.

Delay correction of metabolic acidosis until initial rehydration is complete.

Potassium supplements are added as detailed under "Hypotonic and isotonic dehydration". If oliguria persists and hypernatraemia has resulted from excess sodium chloride administration, peritoneal dialysis may be required.

Potassium therapy

Plasma concentration is a poor guide to the total body potassium, particularly in the presence of dehydration and acidosis, in both of which it is raised. Potassium should be given orally if possible and not commenced until urine flow is established and the blood urea is falling. If given intravenously add 3 mmol/kg/24 hr not exceeding a maximum concentration of 40 mmol/l. ECG monitoring is useful in difficult cases. As intravenous therapy is being discontinued an oral rehydration preparation, e.g. Dextrolyte, should be chosen which also contains potassium, to continue to repair the total body deficit. The management of hyperkalaemia is detailed in Chapter VIII, page 208.

Metabolic acidosis

Metabolic acidosis will tend to correct spontaneously as the circulation and renal function improve and the electrolyte loss subsides. Estimation of the bicarbonate needed to correct acidosis is not precise. Over-rapid and full correction may increase CSF acidosis and produce apnoea. Theoretically, the amount of bicarbonate required is described by the formula:

base deficit (mmol/l)×body weight (kg)×0.3=mmol for full correction.

Not more than half this amount should be given over 24 hours. In the face of severe progressive acidosis (base deficit greater than 10 mmol/l), imminent collapse or situations where acidosis is unlikely to disappear with other I.V. fluids, an initial 0.5 mmol/kg may be given rapidly over 10 minutes and further 0.5 mmol/kg over the next hour. The remainder is then over 24 hours if indicated by acid/base balance.

Metabolic alkalosis

This may occur following persistent vomiting as in pyloric stenosis, in cases of potassium loss and sodium retention, and following the administration of excess alkali. It is usually a self-correcting condition if renal function is good and the precipitating cause eliminated.

If alkalosis is severe and tetany is imminent, it may be necessary to give ammonium chloride. The amount used is:

mmol chloride=base excess (mmol/l)×0.3×body wt (kg)

[Inj. ammomium chloride is 0.89 per cent (1/6 molar) containing 167 mmol of each ion per litre.]

During a period of alkalosis excess potassium is lost in the urine. Extreme degrees of non-respiratory alkalosis may prove resistant to therapy until any potassium deficit is corrected.

Blood volume

The infant's blood volume is 8 per cent (80 ml/kg) of the body weight, so that a transfusion of 20 ml/kg whole blood will raise the haemoglobin by approximately 25 per cent.

Correction of continuing losses

These losses are usually from the gastro-intestinal or renal tract. Replacement should be contemporaneous, the fluids being of similar composition to those lost, their volume being calculated every 6–8 hours. In small infants with large continuing losses, waiting for a 24-hr period to calculate replacement is too long. Fortunately diarrhoea usually stops when the intravenous infusion has started and the replacement fluids calculated (at 5 per cent, etc) are generous. Mild ongoing diarrhoea is usually ignored and in practice its likely sodium content corresponds approximately to 0.18 per cent saline.

Electrolyte composition of alimentary fluids (mmol/l)

	$H+$	Na^+	K^+	Cl^-	HCO_3^-
Gastric	40–60	20–80	5–20	100–150	—
Small Bowel	—	100–140	5–15	90–130	20–40
Biliary	—	120–140	5–15	80–120	30–50
Diarrhoea	—	40	40	40	40

In sodium depletion due to renal loss the urinary sodium concentration is high (>40 mmol/l); when unrelated to renal disease urinary sodium concentration is extremely low (<10 mmol/l).

Composition of solutions for intravenous use in mmol/l

	Na^+	K^+	Cl^-
Sodium chloride injection (0.9%)	150	—	150
Sodium chloride (0.18%) and dextrose (4%) injection	30	—	30
Hartmann's	130	4	104
Citrated plasma	150	12	55

1 ml of 8.4% sodium bicarbonate contains
1 mmol bicarbonate
1 mmol sodium

1 ml Strong potassium chloride BP contains
2 mmol of potassium
2 mmol of chloride

1 ml 30 per cent sodium chloride contains
5 mmol of sodium
5 mmol of chloride

1 ml Inj. calcium gluconate BP 10% contains
0·225 mmol Ca^{++}

Calculation of millimoles

One millimole=molecular weight in milligrams.

Useful atomic weights:

hydrogen	1·0	magnesium	24·3
carbon	12·0	phosphorus	31·0
nitrogen	14·0	chlorine	35·5
oxygen	16·0	potassium	39·1
sodium	23·0	calcium	40·1

For example:
1 mmol NaCl=58·5 mg. 1 mmol $NaHCO_3$=84 mg.
1 mmol KCl=74·6 mg. 1 mmol NH_4Cl=53·5 mg.

Warning

When HYPERTONIC solutions are being infused the site of the indwelling catheter or needle must remain visible and be examined frequently for extravasation. The use of pumps to infuse such solutions is particularly hazardous. Extensive full-thickness skin loss can follow extravasation. The extravasation of calcium salts or 8·4 per cent $NaHCO_3$ will also lead to skin necrosis and ulceration.

M. PANTER-BRICK

Parenteral nutrition

Parenteral nutrition (PN) is required in infants and children whose gastro-intestinal function cannot support normal growth, or when the gut must be "rested" e.g. in protracted diarrhoea, necrotising enterocolitis or severe inflammatory bowel disease.

Techniques

PN may be given via either a central or peripheral vein and in some instances an artery or arteriovenous fistula. Catheters should be inserted using a strict aseptic technique and such catheters should only be used for PN and not for blood sampling or drug administration.

Composition of fluid

Bags of fluid containing amino acids, glucose, macro and micro elements and vitamins should be made up by a pharmacist in a laminar flow unit. The volume of fluid required, nutrient content and the amounts of micro and macro elements and vitamins can be ascertained from Tables 13 and 14.

Patients should be fed initially for their actual weight and then when gaining weight by increments for their expected weight. Abnormal losses should be taken into account. Intralipid*† is dispensed separately.

The equivalent of these fluid and nutrient allowances is shown in Table 15 for commercially available fluids.

TABLE 13 Intravenous Fluid in the Newborn
Requirements according to age in days and birth weight.

Day of life	Volume (ml/kg)
1	60
2	75
3, 4	90
5+	120

add 30 ml/kg/day of water if naked baby under radiant heater or phototherapy lights.

*Intralipid given over 20 hours per day; wait until infusion stopped four hours before taking blood samples.
†KabiVitrum.

The constituents and volume of the fluids should be adjusted for patients with heart, renal and liver disease or failure, as well as those with metabolic and nutritional abnormalities. The daily volume of IV fluid should not normally exceed 120 ml/kg, but exceptions have to be made, particularly when parenteral nutrition is infused through a peripheral vein.

Amino acids This regime uses Vamin* with 10 per cent glucose, a crystalline L-amino acid solution with a profile similar to that of egg protein and one of the most suitable of those available for use in children. The amount should be reduced if the plasma urea and ammonia become elevated. In renal failure consider using essential amino acid solution, e.g. NephrAmine (Boots)

Carbohydrate Only glucose should be used. The amount infused should be reduced if glycosuria>½ per cent or blood glucose>9·7 mmol/l (175 mg%) occurs. It is usually not necessary to use insulin if the glucose is introduced slowly. The concentration of glucose in the solution may be increased if the volume of infusate needs to be decreased without decreasing the calories.

Fat This should be given as 10 per cent Intralipid. This is an isotonic fat emulsion prepared from fractionated soybean oil with egg phosphatides. The fat is 85 per cent unsaturated and polyunsaturated triglycerides. Small-for-dates and pre-term babies are liable to have a reduced plasma clearance. Fat intake should be restricted to 2 g/kg/day. It should not be given if the plasma bilirubin>100 μmol/l, unless the bilirubin-binding capacity of the plasma can be measured. Neither should Intralipid be given when the patient has an uncontrolled infection. Energy derived from fat should not normally exceed 40 per cent of the total energy.

*KabiVitrum.

TABLE 14 Parenteral Nutrition
Total requirements of nutrients and electrolytes per kg body weight calculated according to age or weight of infant or child and by day from start of parenteral nutrition

	Day of Parenteral Nutrition	*Fluid Volume (ml/kg)*	*Non N_2 (kcal/kg)*	*Amino acids (g/kg)*	*N_2 (g/kg)*	*Glucose (g/kg)*	*Fat (g/kg)*	*Sodium (mmol/kg)*	*Potassium (mmol/kg)*
Requirements in newborn	Day 1	See Table 13	42	0.5	0.07	8	1	3	2.5
	2		50	0.75	0.10	10	1	3	2.5
	3		60	1.0	0.13	10	2	3	2.5
	4		68	1.5	0.20	12	2	3	2.5
	5		86	2.0	0.27	14	3*	3	2.5
	6		91–99	2.5	0.34	14–16	3.5	3	2.5
Requirements for babies > 1 month of age and <10 kg	Day 1	150	42	0.5	0.07	8	1	3	2.5
	2	150	50	1.0	0.13	10	1	3	2.5
	3	150	68	1.5	0.20	12	2	3	2.5
	4	150	72	2.0	0.27	13	2	3	2.5
	5 and over	150	86	2.5	0.34	14	3	3	2.5
10–30 kg	Day 1 and 2	60–100	33	1.0	0.13	4.5	1.5	2–3	2–3
	3 and over	60–100	48–62	2.0	0.27	7–8	2–3	2–3	2–3
30 kg+	Day 1 and 2	40–75	18	1	0.13	2	1	2–3	2–3
	3 and over	40–75	32–62	1.5	0.20	3–8†	2–3	2–3	2–3

(*a*) Vitamins as in text.
(*b*) Macro and micro elements:
If weight <10 kg add Ped-el 4 ml/kg/day
>10 kg add Addamel 0.2 ml/kg/day
after checking text p.64.

† Increase glucose by 1 g/kg body wt/day if tolerated.
* Do not exceed 2 g/kg in preterm infants.

TABLE 15 Scheme for Parenteral Nutrition
(using commercially available products) All values are given per kg body weight per day

	Day of Parenteral Nutrition	*Post-gestational age (days)*	*Fluid volume (ml)*	*Vamin 9 Glucose† (ml)*	*5% dextrose (ml)*	*10% dextrose (ml)*	*10% Intralipid (ml)*	*Additional*	
								Na (mmol)	*K (mmol)*
Neonates including	1	3	90*	7	—	73	10	2.7	2.4
Low Birth Weight	1	4 & 5	120*	7	60	43	10	2.7	2.4
according to day	1	6 and over	150*	7	120	13	10	2.7	2.4
of age and length	2	4 & 5	120*	10	20	80	10	2.5	2.3
of parenteral	2	6 and over	150*	10	80	50	10	2.5	2.3
nutrition in days	3	5	120*	14	—	86	20	2.3	2.2
	3	6 and over	150*	14	60	56	20	2.3	2.2
	4	6 and over	150*	21	20	89	20	2.0	2.1
	5	over 6	150*	28	—	112	30	1.6	1.9
	6	over 6	150*	35	—	105–125	35	1.25	1.8
Infants	1		150	7	120	13	10	2.7	2.4
>1 month	2		150	14	80	46	10	2.3	2.2
<10 kg	3		150	21	20	89	20	2.0	2.1
	4		150	28	—	102	20	1.6	1.9
	5 and over		170	35	—	105	30	1.25	1.8
10–30 kg	1 and 2		60	14		31	15	2.0	2.0
	3 and over		90-100	28		42–52	20–30	1.5	1.5
>30 kg	1 and 2		36	14	12		10	2.0	2.0
	3 and over		50–75	21	—	9–59‡	20–25	1.5	1.5

(*a*) Vitamins as in text.
(*b*) Macro and micro elements:
If weight less than 10 kg add Ped-el 4 ml/kg/day. When weight more than 10 kg add Addamel 0.2 ml/kg/day after checking text p.64.

*If under radiant heater or receiving phototherapy allow 30 ml/kg/day of extra water. This may be achieved in most instances by reducing the 10 per cent dextrose by 30 ml/kg and increasing 5 per cent dextrose by 60 ml/kg.
†Vamin with 10 per cent dextrose.
‡Volume of fluid may be decreased by substituting 20 per cent for 10 per cent dextrose.

If Intralipid is not used, the infant's skin should be coated with sunflower oil twice daily to prevent essential fatty acid deficiency.

Macro and micro elements Vamin contains insufficient amounts of Na^+, K^+, Ca^{++}, Mg^{++}, and Cl^-. Sodium and potassium should be given as indicated in the Tables and increased to compensate for abnormal losses. Ped-el* contains Ca^{++}, Mg^{++}, Fe^{++}, Zn^{++}, Mn^{++}, Cu^{++}, F^-, I^-, P^{3-}, and Cl^- and is given to infants of up to 10 kg body weight in a dose of 4 ml/kg/day. Additional P^{3-} 0·4 mmol/kg for preterm infant as Addiphos.* It is added to the Vamin-dextrose solution when renal function has become established. There is no suitable electrolyte mixture commercially available for children over 10 kg, but Addamel* 0·2 ml/kg/day may be added to the Vamin-dextrose; it does not contain phosphate and has insufficient calcium. Trace element mixtures should only be given when renal function is established. Deficiencies particularly of zinc and copper are liable to occur in diarrhoeal states.

Vitamins Water-soluble vitamins should be given as Solivito*. The vial should be reconstituted with 5 ml of 10 per cent dextrose and added to the Vamin-dextrose or Intralipid in a dose of 0·5 ml/kg/day to a maximum of 5 ml/day. The solution must be protected from the light. Vitamins A, D and K can be given in the form of Vitlipid Infant* 1 ml/kg/day to a maximum of 4 ml/day for patients up to 10 years, then normal Vitlipid. This must be added to the Intalipid. Preterm infants may need extra vitamin E.

Infusion system

This is shown diagrammatically in Fig. 3. Stringent asepsis is essential. Connections between the catheter and the infusion system should be soaked in isopropyl alcohol (70%) for 3 minutes before disconnection and reconnection of the fresh system. Millipore filters should be inserted on any air inlets. Fluid should not be allowed to enter the air inlet tubing. An air eliminator/particle filter should be included in the circuit as shown.

Nutrient solutions and the system should be changed daily and samples taken for bacteriology as indicated in Table 16.

* KabiVitrum.

Monitoring

Table 16 gives the daily and weekly routines for monitoring of neonates, infants and children. Close liaison with a clinical chemistry department with facilities for microanalytic estimations is essential.

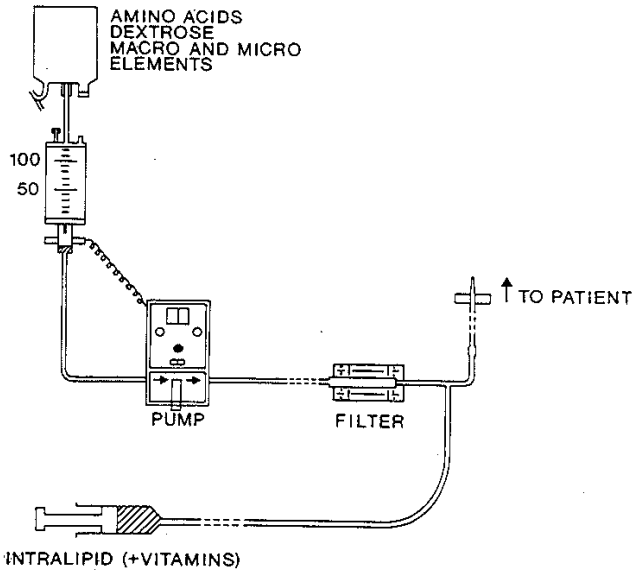


Fig. 3. Diagram of system used to administer parenteral nutrition.

Complications

See Table 17. Most of these are avoidable.

Infection Attention to detail and strict asepsis is essential.

Hypophosphataemia This may occur despite apparently adequate phosphate supplementation. The regimes described adequate phosphate supplementation. The regimes described here contain P^{3-} in barely adequate amounts when Ped-El is used and none when Addamel is used as a source of elements.

TABLE 16 Recordings, Routines and Investigations while on Parenteral Nutrition

Daily	*3 × Weekly*	*Once Weekly (or as indicated)*
Weight Chart input/output* Change I.V. infusion sets and filters		Length+head circumference Skinfold thickness
Bacteriology		Blood culture CSF exam+culture Urine culture } if infection is suspected
Culture (*a*) Specimen of Vamin/dextrose electrolyte from burette or container (*b*) Filter (*c*) Specimens from Intralipid container and tubing		
Urine Chemistry		
Urine glucose (6-hourly for 1 week, then daily)	Urine electrolytes	
Plasma Chemistry		
Dextrostix (6-hourly for 1 week, then daily)		Albumin Transaminases
Electrolytes (daily for 1st week)	Urea and electrolytes (when stabilised)	Bilirubin (if clinically jaundiced) Calcium and phosphorus Alkaline phosphatase
H^+ Plasma turbidity	H^+	Magnesium Glucose
Haematology		Haemoglobin, haematocrit, WBC+differential and platelets Screening for blood coagulation defect (if indicated).

*Account for volume and constituents of concurrent infusions and enteral intake.

TABLE 17 Complications of Parenteral Nutrition in Childhood

Infection
Activation of infection
Extravasation and tissue necrosis (See below)
Electrolyte disturbances
Hypophosphataemia
Anaemia
Hypoxia
Thrombocyte and neutrophil dysfunction
Hypo and hyperglycaemia
Trace element deficiencies
Hyperammonaemia
Hyper and hypocalcaemia
Essential fatty acid deficiency
Hepatic dysfunction
Increased folic acid requirement
Metabolic acidosis

WARNING:
When HYPERTONIC solutions are being infused the site of the indwelling catheter or needle must remain visible and be examined frequently for extravasation. The use of pumps to infuse such solutions is particularly hazardous. Extensive full-thickness skin loss can follow extravasation.

Phosphate is present as phospholipid in Intralipid although there is some suggestion that it may not be biologically available. If plasma levels fall below normal, phosphate should be given in a dose of 0·25–0·5 mmol/kg/day as the potassium salt in the daily requirement of dextrose solution. This cannot be mixed with Vamin or the trace element mixtures.

Hypoglycaemia To avoid this problem parenteral nutrition should never be stopped abruptly. Drips should be resited immediately and cessation of parenteral nutrition should be a gradual one as enteral feeding is increased.

Commencing enteral nutrition

This should be commenced cautiously and increased in volume and nutrient content depending on gastro-intestinal function. The parenteral nutrition should be reduce accordingly.

C. A. HUGHES.

Enclosure (9)

ADVANCED PAEDIATRIC LIFE SUPPORT

The Practical Approach

Advanced Life Support Group

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APPENDIX

B

Fluid and electrolyte management

Fluid and electrolyte management is an essential part of both the immediate and the ongoing care of all sick children. In this appendix we will look at the following:

1. Normal requirements.
2. Dehydration.
3. Diabetic ketoacidosis.
4. Hypervolaemia.
5. Specific electrolyte problems.

NORMAL REQUIREMENTS

Volume

Blood volume is about 100 ml/kg at birth falling to about 80 ml/kg at 1 year. Total body water varies from just under 800 ml/kg in the neonate to about 600 ml/kg at 1 year; after this it varies little. Of this about two-thirds (400 ml/kg) are intracellular fluid; the rest is extracellular fluid. Thus initial expansion of vascular volume in shock can be achieved with relatively small volumes of fluid: 20 ml/kg will usually suffice. However, this volume is only a fraction of that required to correct dehydration if the fluid has been lost from all body compartments. In fact, 20 ml/kg is 2% of body weight. Clinically, dehydration, which is distributed across the fluid compartments rather than being restricted to the vascular compartment, is not detectable until it is greater than 5% (50 ml/kg).

Much is spoken about normal fluid requirements, although in truth there is no such thing. We are all aware as adults that if we drink little we do not get dehydrated and if we drink lots we merely diurese. Healthy children's kidneys are just as capable of maintaining fluid balance. Fluids in neonates are often prescribed on the basis of 150 ml/kg/day, but this is not related to fluid needs and is merely the volume of standard formula milk required to give an adequate protein and calorie intake. What is required clinically is a simple means of prescribing fluid such that patients are maintained in a well-hydrated state and passing reasonable quantities of urine. This formula then has to be modifiable in order to take account of the need for rehydration of dry patients and prevention of overhydration in patients in whom either renal function is impaired or fluid restriction is indicated (e.g. meningitis, cerebral oedema). Fluid requirement can be divided into four types:

1. For replacement of *insensible losses* through sweat, respiration, gastrointestinal loss, etc.
2. For replacement of *essential urine output*, the minimal urine output that allows excretion of urea, etc.
3. Extra fluid to maintain a *modest state of diuresis*.
4. Fluid to replace *abnormal losses* such as blood loss, severe diarrhoea, diabetic polyuria losses, etc.

A formula for calculating this fluid requirement is given in Table B.1. It is useful because it is simple, can be applied to all age ranges and is easily subdivided. The formula gives total fluid requirements, i.e. types (1)+(2)+(3) above. Of this total *one-fifth* represents insensible losses, the *second fifth* represents essential urine output, and the last *three-fifths* represent extra fluid to maintain urine output. Thus this formula is easily adaptable to all states of hydration (Table B.2).

Table B.1. Normal fluid requirements

Body weight	Fluid requirement per day (ml/kg)	Fluid requirement per hour (ml/kg)
First 10 kg	100	4
Second 10 kg	50	2
Subsequent kilogram	20	1

Table B.2. Subdivisions of total fluids

Fraction of total fluid	Function	Amount given	Type of hydration
First fifth	Insensible loss	One-fifth	Insensible losses only
Second fifth	Essential urine output	Two-fifths	Severe fluid restriction
Last three-fifths	Maintenance of urine output	Three-fifths	Moderate fluid restriction
		Four- or five-fifths	Adequate fluids
		Six- to ten-fifths	Induced diuresis

For the very sick and those with renal impairment, a combination of insensible losses plus urine output, given on an hourly basis, is often the best method of fluid management.

Electrolytes

To speak of normal electrolyte requirements is as artificial as speaking of normal fluid requirements. There are obligatory losses of electrolytes in stools, urine, and sweat, and these require replacement. Any excess is simply excreted in the urine. Table B.3 shows the electrolyte content of various body fluids and Table B.4 gives electrolyte "requirements" if there are not excessive losses from any compartment. In truth these "requirements" represent quantities that, if given, maintain homoeostasis without recourse to the various hormonal and renal tubular mechanisms for maintaining the extracellular fluid composition.

Table B.3. Electrolyte contents of body fluids

Fluid	Na^+ (mmol/l)	K^+ (mmol/l)	Cl^- (mmol/l)	HCO_3^- (mmol/l)
Plasma	135–141	3·5–5·5	100–105	24–28
Gastric	20–80	5–20	100–150	0
Intestinal	100–140	5–15	90–130	15–65
Diarrhoea	7–96	34–150	17–164	0–75
Sweat	<70	6–15	<70	0–10

Table B.4. Normal water, electrolyte, energy, and protein requirements

Body weight	Water (ml/kg/day)	Sodium (mmol/kg/day)	Potassium (mmol/kg/day)	Energy (kcal/kg/day)	Protein (g/kg/day)
First 10 kg	100	2–4	1·5–2·5	110	3·00
Second 10 kg	50	1–2	0·5–1·5	75	1·50
Subsequent kilogram	20	0·5–1	0·2–0·7	30	0·75

Tables B.5 and B.6 show commonly available intravenous fluids with their composition. Their usage is described in the appropriate sections below. The main point to note is the need for precise prescription. It is clear that "dextrose saline" can refer to any one of several different preparations.

Table B.5. Commonly available crystalloid fluids

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

Table B.6. Commonly available colloid fluids

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

DEHYDRATION

Dehydration is the result of abnormal fluid losses from the body which are greater than the amount that the kidneys can compensate for. The natural mechanisms for compensation have the primary aim of maintaining circulating volume and blood pressure at all cost (Figure B.1). Thus most patients with dehydration maintain their central circulation satisfactorily. Loss of central circulatory homoeostasis constitutes *hypovolaemic shock* and is dealt with in Chapter 9.

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

1. Isotonic dehydration.
2. Hyponatraemic dehydration.
3. Hypernatraemic dehydration.

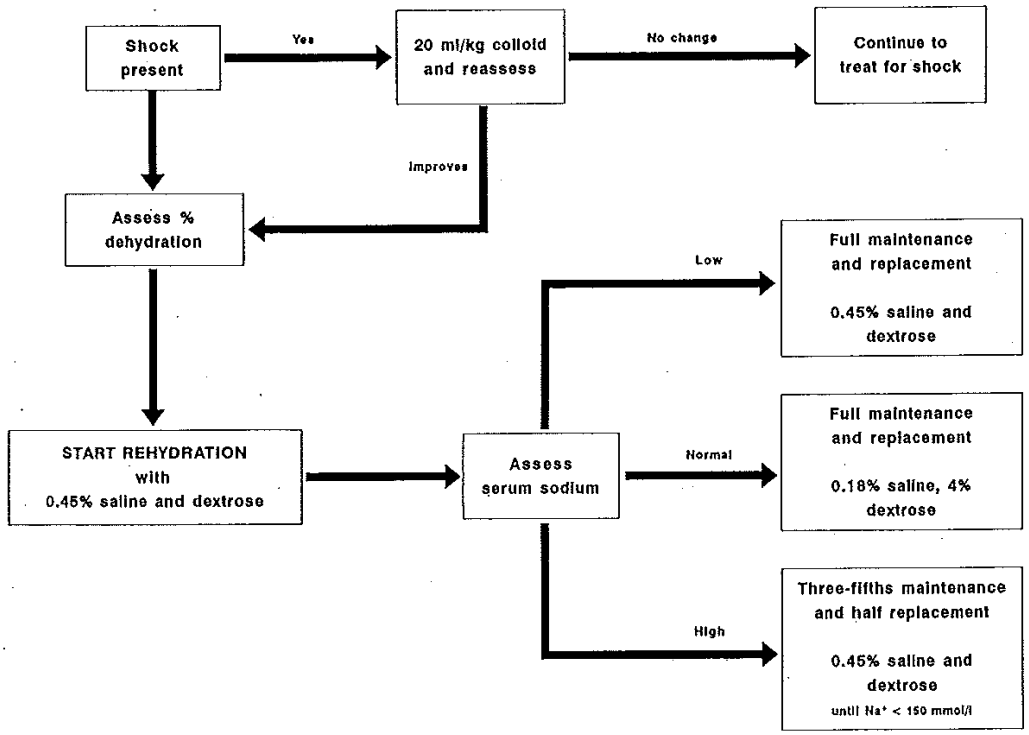

Figure B.1. Algorithm for the management of dehydration in the first 24 hours

In all three types there is a total body deficit of salt and water. Between the three types the relative amounts of salt and water loss vary. Table B.7 shows the symptoms and signs of dehydration and gives a guide towards the assessment of the degree of dehydration. On the whole, the more severe the dehydration the more probable that hypovolaemia is going to be a problem; most patients with more than 10% dehydration are hypovolaemic at presentation. However, speed of fluid loss is important. Slow, prolonged losses can give rise to massive dehydration without hypovolaemia; similarly

acute, severe loss can present as hypovolaemia without apparent significant dehydration. The latter is not infrequently the case in acute gastroenteritis in infants, where acute fluid loss into the bowel causes hypovolaemia and the patient can present even before any diarrhoea has occurred.

Table B.7. Symptoms and signs of dehydration

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

Management of dehydration

Mild dehydration (<5%) can often be managed with oral fluids as long as vomiting is not a major problem. It can be seen from Table B.5 that 0·18% saline, 4% dextrose gives maintenance amounts of sodium and water. Natural renal homoeostatic mechanisms will cause sodium and water retention, so the use of maintenance fluids in normal quantities is sufficient – the deficits will be corrected through an initial lower urine output.

Once the deficit exceeds 5% it is most appropriate to replace this. The deficit is calculated using the following formula:

$$\text{Percentage dehydration} \times \text{Weight} \times 10 \text{ (in kg)}$$

Thus a 10 kg infant who is 7·5% dehydrated requires the replacement of 750 ml. In all types of dehydration, the deficit is mainly extracellular fluid loss and ought to be replaced as normal (physiological) (0·9%) saline. If not, dilutional hyponatraemia will occur. In the absence of the need for immediate circulatory support, this replacement ought to take place over 24 hours to prevent rapid fluid shifts. In addition to the replacement of the deficit, normal maintenance fluids with 0·18% saline, 4% dextrose ought to be given. In practice, in both isotonic and hyponatraemic dehydration, it is often easiest to give the first 24-hour total fluid (deficit + maintenance) as 0·45% saline with dextrose. Following this, isotonic dehydration therapy can be continued with 0·18% saline, 4% dextrose. In hyponatraemic states continuation of 0·45% saline with dextrose will usually correct the sodium deficit easily. All fluids will require potassium. In the knowledge that significant hypokalaemia does not occur until considerable total body potassium depletion has occurred, this usually means giving twice the normal maintenance quantities, i.e. 20 mmol/500 ml.

In hypernatraemic dehydration there is a greater degree of water loss than salt loss. This leads to a higher serum osmolality, dragging fluid from the intracellular to the extracellular space, and maintaining the circulation. Thus the total fluid deficit is often higher than one would predict clinically, and there is a total body sodium deficit. The

deficit ought, as before, to be replaced with normal (physiological) saline and maintenance given with 0·18% saline, 4% dextrose. Again, this can be simplified to initially just giving 0·45% saline. The risk of hypernatraemic dehydration is that over-rapid reduction in the serum sodium will lead to rapid shifts of water into the cells leading to cerebral oedema and convulsions. This can be averted by slowing down the rate of rehydration and replacing the deficit over 48 hours, giving maintenance at three-fifths the normal total. After the first 48 hours, the fluid can be changed to 0·18% saline, 4% dextrose, and once the serum sodium is below 150 mmol/l the rate of administration can be increased to normal.

The very sick child

In the very sick it cannot be assumed that normal homoeostatic mechanisms will work. The patient may be progressing into renal failure and be oliguric or inappropriately polyuric. In such cases the best management is to:

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

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

DIABETIC KETOACIDOSIS

Diabetic ketoacidosis is a special case in which a relative or absolute lack of insulin leads to an inability to metabolise glucose. This leads to hyperglycaemia and an osmotic diuresis. Once urine output exceeds the ability of the patient to drink, dehydration occurs. In addition, without insulin, fat is used as a source of energy leading to the production of large quantities of ketones and metabolic acidosis. The latter is initially compensated for by hyperventilation and a respiratory alkalosis but, as the condition progresses, the combination of acidosis, hyperosmolality, and dehydration leads to coma. Diabetic ketoacidosis is often the first presentation of diabetes, but may be a problem in known diabetics who have decompensated through illness, infection, or non-adherence to their treatment regimens.

History

The history is usually of weight loss, abdominal pain, vomiting, polyuria, and polydipsia.

Examination

Children are usually severely dehydrated with deep and rapid (Kussmaul's) respiration. They have the fruity smell of ketones on their breath. Salicylate poisoning and uraemia are differential diagnoses to be excluded. Infection often precipitates decompensation in both new and known diabetics, and must be sought.

Management

Assess
*A*irway
*B*reathing
*C*irculation

Give 100% oxygen. Place on a cardiac monitor (hypokalaemia can cause dysrhythmias).

Site an intravenous drip and begin fluid replacement. If shock is present, treat as discussed in Chapter 9.

Take blood for:

- Glucose.
- Urea and electrolytes, creatinine, Ca^{2+}, PO_4^{3-}.
- Full blood count and differential white cell count.
- Culture.
- Blood gases.

Take urine for:

- Culture.
- Sugar.
- Ketones.

Fluids

The principles of fluid management outlined above work as well for diabetic ketoacidosis as for any other cause of dehydration. However, as there is hyperglycaemia it is best not to give dextrose initially. Thus, having calculated deficit, maintenance, and 24-hour requirement, this can initially be given all as normal (physiological) saline, switching to 0·45% saline or 0·18% saline with dextrose once the blood sugar has fallen. With the osmotic diuresis, which will persist until the blood sugar falls, calculated fluid requirements will be an underestimate. However, this is beneficial as over-rapid rehydration is likely to cause cerebral oedema.

Insulin

Insulin ought to be given by continuous infusion. The initial dose is 0·1 unit/kg/h. Once the blood sugar falls to less than 10 mmol/l, glucose must be added to the intravenous drip. *Do not stop using insulin. This is the child's prime requirement.* Administer the insulin by separate line. Add 50 units of soluble insulin to 50 ml saline. This solution is 1 unit/ml (0·1 unit/kg/hour is equal to 0·1 × weight in kg, as ml/hour). Thus a 20 kg child would have 2 ml/hour, a 35 kg child 3·5 ml/hour. This often needs decreasing to 0·05 unit/kg/hour when blood sugar starts to fall. In a very young diabetic (under 5 years) start with the smaller dose.

Acidosis

The acidosis of diabetic ketoacidosis is initially compensated for by hyperventilation. Once the blood pH falls below 7·1, CNS depression can occur and this can prevent compensation. Almost always acidosis will correct with correction of fluid balance and cessation of ketosis with insulin therapy. Bicarbonate should be avoided unless the blood

pH is less than 7·0, or less than 7·1 and not improving after the first few hours of fluid and insulin therapy. Many formulae exist relating the base excess to the child's weight and the bicarbonate requirement. However, because of the logarithmic relationship between $[H^+]$ and pH (see Figure A.1), a dose of 2·5 ml/kg of 8·4% $NaHCO_3$ will correct the pH to 7·2 or 7·3 in all cases. This should be administered slowly over 2 hours by infusion. Re-check the pH after the first hour and stop the infusion if the pH is above 7·15 as the rest will correct naturally.

Other treatments

A nasogastric tube is essential as acute gastric dilatation is a common complication. Depending on the level of consciousness, bladder catheterisation may be required. Antibiotics may be indicated.

Monitoring progress

Use a flow sheet to record vital signs, neurological status, input and output of fluids, blood results, and insulin infusion rates. Record urine ketones and glucose. Initially, while intravenous insulin is in use, check blood sugar, biochemistry, and acid–base status 2-hourly.

Complications

Major complications	
Cerebral oedema	Prevent by slow normalisation of sugar and hydration; monitor neurological status hourly; treat cerebral oedema with hyperventilation and mannitol (see Chapter 12)
Hypokalaemia	
Cardiac dysrhythmias	Usually secondary to electrolyte disturbances
Acute renal failure	Uncommon

Any of the complications in the box requires intensive monitoring on an intensive care unit.

HYPERVOLAEMIA

Hypervolaemia and fluid overload in children is a rarer problem than dehydration. The causes tend to be either cardiac failure or renal failure. Occasionally water intoxication following deliberate ingestion or misuse of desmopressin (DDAVP) occurs.

Fluid overload from renal failure presents with raised venous pressure, a triple rhythm, and pulmonary crepitations or crackles, and is treated initially with diuretics. They may be ineffective and treatment will then require the use of oxygen, vasodilators, and urgent dialysis.

Water intoxication will usually present as convulsions secondary to cerebral oedema and hyponatraemia. Treatment is along the usual lines for patients with convulsions and coma (see Chapters 11 and 12). Severe fluid restriction will be necessary, and if hyponatraemia is severe (<120 mmol/l), fluids ought to be given as 0·9% saline. Diuretics, particularly mannitol, which causes a free water diuresis and reduces cerebral oedema, are sometimes of value.

Mild oedema may occur in any of these conditions; severe oedema does not and, when present, is usually a manifestation of nephrotic syndrome. This is important as patients with nephrotic syndrome are intravascularly fluid depleted and diuretics are contraindicated.

SPECIFIC ELECTROLYTE PROBLEMS

Sodium

Sodium is the major extracellular cation. Its movement is inextricably linked to that of water. Disorders of sodium balance are therefore the same as over- and under-hydration, and are dealt with earlier in this appendix.

Potassium

Unlike sodium, potassium is mainly an intracellular ion, and the small quantities measurable in the serum and extracellular fluid represent only a fraction of the total body potassium. However, the exact value of the serum potassium is important as cardiac arrhythmias can occur at values outside the normal range. As the majority of potassium is stored intracellularly, this acts as a large buffer to maintain the serum value within its normal narrow range. Thus, hypokalaemia is usually only manifest after significant total body depletion has occurred. Similarly, hyperkalaemia represents significant total body overload, beyond the ability of the kidney to compensate. The exception to both these statements is the situation in which the cell wall pumping mechanism is breached. A breakdown of the causes of hyper- and hypokalaemia is given in Table B.8.

Table B.8. Causes of hypo- and hyperkalaemia

Hypokalaemia	Hyperkalaemia
Diarrhoea	Renal failure
Alkalosis	Acidosis
Volume depletion	Adrenal insufficiency
Primary hyperaldosteronism	Cell lysis
Diuretic abuse	Excessive potassium intake

Hypokalaemia

Hypokalaemia is rarely a great emergency. It is usually the result of excessive potassium losses from acute diarrhoeal illnesses. As total body depletion will have occurred, large amounts are required to return the serum potassium to normal. The fastest way of giving this is with oral supplementation. In cases where this is unlikely to be tolerated, intravenous supplements are required. However, strong potassium solutions are dangerous and can precipitate arrhythmias; thus the concentration of potassium in intravenous solutions ought not to exceed 80 mmol/l. Fortunately, this is not usually a problem as renal conservation of potassium aids restoration of normal serum levels.

Patients who are alkalotic, hyperglycaemic (but not diabetic), or are receiving insulin from exogenous sources, will have high intracellular potassium stores. Thus hypokalaemia in these cases is the result of a redistribution of potassium rather than potassium deficiency, and treatment of the underlying causes is indicated.

Hyperaldosteronism is a cause of hypokalaemic alkalosis. Patients with this condition will have salt and water retention, and will be hypertensive on presentation. Isolated hyperaldosteronism is the body's natural response to hypovolaemia and salt deficiency. Thus secondary hyperaldosteronism is a common cause of hypokalaemic alkalosis. As there is primary salt and water deficiency the patient is not usually hypertensive. The most common causes are diarrhoeal illness and salt-losing conditions such as cystic fibrosis. External loss of cerebrospinal fluid and fluid loss from intestinal ostomies or drains are other causes. Although potassium replacement is required in this condition, the main thrust of therapy has to be with salt and water replacement to re-expand the circulation and cut down on aldosterone production.

Hyperkalaemia

Hyperkalaemia is a dangerous condition. Although the normal range extends up to 5·5 mmol/l it is rare for arrhythmias to occur at levels below 7·5 mmol/l. The most common cause of hyperkalaemia is renal failure – either acute or chronic. Hyperkalaemia can also result from potassium overload, loss of potassium from cells due to acidosis or cell lysis, hypoaldosteronism, and hypoadrenalism.

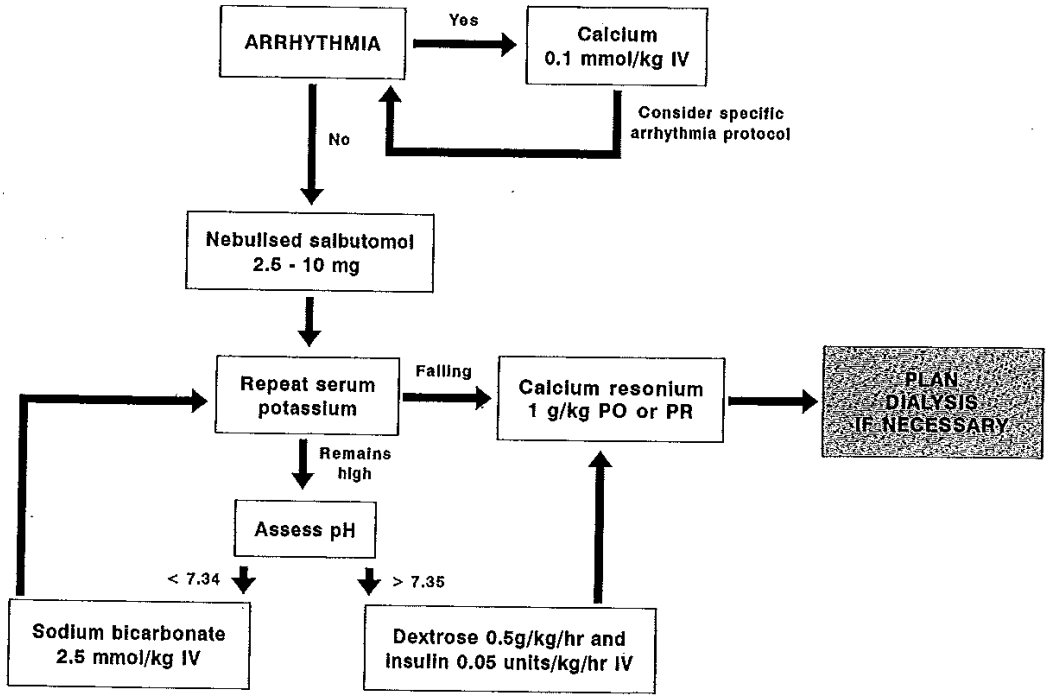

Figure B.2. Algorithm for the management of hyperkalaemia

The immediate treatment of hyperkalaemia is shown schematically in Figure B.2. If there is no immediate threat to the patient's life because of an arrhythmia, then a logical sequence of investigation and treatment can be followed. β_2-Stimulants, such as salbutamol, are the immediate treatment of choice for hyperkalaemia. They act by stimulating the cell wall pumping mechanism and promoting cellular potassium uptake. They are best administered by nebuliser. The dose to be given is shown in Table B.9. The serum potassium will fall by about 1 mmol/l with these dosages.

Table B.9. Salbutamol dose by age

Age (years)	Salbutamol dose (mg)
≤2·5	2·5
2·5–7·5	5
>7·5	10

Sodium bicarbonate is also effective at rapidly promoting intracellular potassium uptake. The effect is much greater in the acidotic patient (in whom the hyperkalaemia is likely to be secondary to movement of potassium out of the cells). The dosage is the same as that used for treating acidosis and 2·5 ml/kg of 8·4% $NaHCO_3$ is usually effective. It is mandatory also to check the serum calcium, because, particularly in patients with profound sepsis or renal failure, hyperkalaemia can be accompanied by marked hypocalcaemia. The use of bicarbonate in these situations can provoke a crisis by lowering the ionised calcium fraction, precipitating tetany, convulsions or hypotension, and arrhythmias.

Insulin and dextrose are the classic treatment for hyperkalaemia. They are not, however, without risks. It is very easy to precipitate hypoglycaemia if monitoring is not adequate. Large volumes of fluid are often used as a medium for the dextrose and, particularly in the patient with renal failure, hypervolaemia and dilutional hyponatraemia can then be a problem. Many patients are quite capable of significantly increasing endogenous insulin production in response to a glucose load, and this endogenous insulin is just as capable of promoting intracellular potassium uptake. It thus makes sense to start treatment with just an intravenous glucose load and then to add insulin as the blood sugar rises. The initial dosage of glucose ought to be 0·5 g/kg/hour, i.e. 2·5 ml/kg/hour of 20% dextrose. Once the blood sugar is above 10 mmol/l, insulin can be added if the potassium is not falling. The dosage of insulin is initially half that used in diabetic ketoacidosis, i.e. 0·05 unit/kg/hour. This can then be titrated according to the blood sugar.

The above treatments are the fastest means of securing a fall in the serum potassium, but all work through a redistribution of the potassium into cells. Thus, the problem is merely delayed rather than treated in the patient with potassium overload. The only ways of removing potassium from the body are with dialysis or ion-exchange resins such as polystyrene sulphonate resins, e.g. Calcium Resonium. If it is anticipated that the problem of hyperkalaemia is going to persist, then the use of these treatments ought not to be delayed. Dialysis can only be started when in an appropriate environment. Ion-exchange resins can be used at the outset. The dosage of Calcium Resonium is 1 g/kg as an initial dose either orally or rectally, followed by 1 g/kg/day in divided doses.

In an emergency situation where there is an arrhythmia (heart block or ventricular arrhythmia), then the treatment of choice is intravenous calcium. This will stabilise the myocardium, but will have no effect on the serum potassium. Thus the treatments discussed above will still be necessary. The dosage is 0·5 ml/kg of 10% calcium gluconate (i.e. 0·1 mmol/kg Ca^{2+}). This dose can be repeated twice.

Calcium

Some mention of disorders of calcium metabolism is relevant as both hyper- and hypocalcaemia can produce profound clinical pictures.

Hypocalcaemia

Hypocalcaemia can be a part of any severe illness. Specific conditions that may give rise to hypocalcaemia include severe rickets, hypoparathyroidism, pancreatitis,

rhabdomyolysis, and citrate infusion (in massive blood transfusions). Acute and chroni renal failure can also present with severe hypocalcaemia. In all cases hypocalcaemia ca produce weakness, tetany, convulsions, hypotension, and arrhythmias. Treatment i that of the underlying condition. In the emergency situation, however, intravenou calcium can be administered. As most of the above conditions are associated with a tota body depletion of calcium, and as the total body pool is so large, acute doses will ofte only have a transient effect on the serum calcium. Continuous infusions will often b required, and must be given through a central venous line because calcium is so irritan in peripheral veins. In renal failure, high serum phosphate levels may prevent the serum calcium from rising and dialysis is often indicated in these circumstances.

Hypercalcaemia

Hypercalcaemia usually presents as long-standing anorexia, malaise, weight loss, failure to thrive, and vomiting. Causes include hyperparathyroidism, hypervitaminosis D or A, idiopathic hypercalcaemia of infancy, malignancy, thiazide diuretic abuse, and skeletal disorders. Initial treatment is with volume expansion with normal (physiological) saline. Following this, investigation and specific treatment are indicated.

Enclosure (10)

RBHSC RENAL TRANSPLANT GUIDELINES

UKT will phone the on-call Consultant Nephrologist with kidney offer (call will go to Barbour Ward - 028 90636621 or 028 90633398).

Consultant will ring parents (phone nos in dialysis room red box).
Recipient co-ordinators available office hrs - office 028 90263846, bleep 07623 652169 via BCH switch. (Dolores Elliot, Sharon McCarron, Pamela Stronge).

Kidney will arrive to Ward 11T (Transplant) Belfast City Hospital (028 90263652). Transport organised via UKT and BCH Transplant Ward (Consultant may need to liaise).

CHECK LIST ON ADMISSION

A) HISTORY

Wt = Ht =

BSA (see nomogram) =

Pre transplant urine output mls / 24hours

Pre transplant fluid allowance mls / 24hours

Underlying diagnosis
Urological problems
Bladder Function
Recent contact with infectious diseases
When last dialysed
Vaccination history
CMV status donor/recipient
Cytotoxic antibody status
Tube feeds - what and how much
What central line sites used previously
List drugs (consultant will probably stop anti-hypertensives and non essential drugs such as Alfacalcidol and phosphate binders)

B) EXAMINATION

Clinical examination to establish fit for surgery
State of nutrition
State of hydration
BP
Catheter exit site appearance

C) **INVESTIGATIONS** (insert peripheral cannula at same time)

Clotted sample - 5ml by Hospital transport 7111-6402 (come to dialysis room at back of Barbour Ward) to Ward 11T (Transplant), BCH for tissue typing X-match (tissue type lab 028 90263846 or via BCH switchboard 028 90329241).

FBP, DWCC, coagulation screen
U/E, creatinine, Ca, albumin
Group and X-match 4 units CMV -ve blood
Virology for up to date CMV and EBV (IgG). Check notes for other virology
Skin swabs -nasal, perineum or groin, exit sites e.g. gastrostomy or dialysis line (for LRD should already be performed-check result. May need decolonisation)
PD fluid and urine for culture
Urine for U and E
CXR
ECG if on anti-hypertensives

PLAN:-

1. Fast and consent
2. Shower
3. Dialysis if k+>5 mmol/L
4. Fluid management to be determined by Consultant Nephrologist
5. If prolonged fast - maintenance IV fluids (give insensible losses (= 300 ml/m^2/day) and output usually as 2.5% dextrose 0.45% sodium chloride solution
   D/W consultant if Na < 133. Repeat U/E at time of going to Theatre
6. CHECK CMV Ig G STATUS and order Valganciclovir if indicated (for post-op use).

DONOR	RECIPIENT	ACTION
positive	negative	valganciclovir
positive	positive	valganciclovir
negative	positive	valganciclovir
negative	negative	No treatment

Valganciclovir should be taken with food. The dose is calculated as below (Vaudry et al, 2009). The dose should be administered orally once daily and therapy should begin within 10 days of transplantation and continue until day 100 post-transplantation.
Formulation: 250 mg in 5 mls, 450 mg tablets.

Dose (mg) = 7 x BSA x GFR (Schwartz formula) - Maximum dose 900mg daily.

GFR = Height (cms) x40 / creatinine.

IMMUNOSUPPRESSION PRE OP

1. TACROLIMUS

0.15 mg/kg as a single oral dose 1-4 hrs pre-operatively.
For LRD dose 20.00 hrs night before and 08.00 on day of transplant.

2. MYCOPHENOLATE MOFETIL (MMF)

day 1 - 14	**600 mg/m^2** twice daily orally or iv (max adult dose 1000 mg twice daily)
after day 15	**300 mg/m^2** twice daily orally

Single oral dose 1-4 hrs pre-operatively. For LRD dose 20.00 hrs and 08.00.

3. BASILIXIMAB

2nd TRANSPLANTS, THOSE AT HIGH RISK OF REJECTION OR BARDET-BIEDL SYNDROME PATIENTS MAY RECIEVE ON CONSULTANT DECISION BASILIXIMAB

Basiliximab (Simulect)

Dose: patients will be given two infusions of Basiliximab.
< 35kg: 10mg infused on Day 0 within 2 hours prior to surgery and on Day 4 after transplantation
> 35kg: 20mg infused on Day 0 within 2 hours prior to surgery and on Day 4 after transplantation

Reconstitution & Administration
Simulect is provided as a vial containing 20mg Basiliximab powder + ampoule of water for injections.
Add 5ml water for injection to the vial containing 20mg Basiliximab powder.
Shake the vial gently to dissolve the powder. After reconstitution the solution should be used immediately (at least within 24hours if stored in a refrigerator).
The reconstituted solution is isotonic and must be further diluted:
10mg dose must be diluted to at least 25 mls (20mg to at least 50 mls) with sodium chloride 0.9% or glucose 5%, infuse over 20-30minutes.

IN THEATRE

Assess hydration, check electrolytes and ABG x 2 hourly
IV Co-Amoxiclav 30 mg/kg on induction
S/C Heparin with Surgeons consent after induction

- Tinzaparin (Innohep®) 50 I.U. s/c per kg post vascular anastamosis, and 24hrly.
- Multidose vial 10,000 I.U. per ml -2 people to check dose.

Triple lumen CVP catheter
IA line in small children
Start Dopamine 2 - 3 µg/kg/min
Use N saline, 4.5% albumin or blood (as appropriate) to raise CVP to 8-12 mmHg prior to removal of vascular clamps.
Blood Transfusion can result in sensitisation, therefore **transfuse only if actively bleeding or Hb <8 g/dl.**
Use CMV negative blood.

10 - 15 MINS PRIOR TO RELEASE OF CLAMPS

- 0.5 g/kg (2.5 ml/kg) 20% Mannitol
  (alternative 4 mg/kg Frusemide)

- 10 mg/kg Methylprednisolone (max 500 mg)

POST-OPERATIVE

1. FLUIDS

Replace urine output and insensible losses (300 ml/m^2/day) EACH HOUR as 0.45% saline 2.5% dextrose (subtract volume of infusions).

Anticipate hypovolaemia (low peripheral temp., low CVP, tachycardia, low BP).

Boluses of N saline or 4.5 % albumin (5 - 10 ml/kg) over 20 mins to maintain CVP and BP.

2. OBSERVATIONS

a. CVP between 8 - 10 mmHg

b. BP decided on individual basis

c. Optimal urine output to be decided on an individual basis
In polyuric patients this will be around 4 ml/kg/hr initially falling to 2 ml/kg/hr when stable. In previously anuric patients far lower outputs may be acceptable if ATN has occurred. Check transplant troubleshooter for management guidelines on output and BP.

3. DRUGS POST OP

a. TACROLIMUS (Prograf®)

0.3 mg/kg divided into 2 doses (0.15 mg/Kg each) to be given orally and started within 24 hours of transplant.
I/V tacrolimus dose is 1/5 of total oral dose i.e. 0.06mg/kg/day given as continuous infusion over 24 hours in N saline or 5% Dextrose via non PVC giving set or syringe and tubing and change daily.
Infusion concentration in range 0.004 - 0.1 mg/ml

Capsules sizes available 0.5 mg 1 mg 5 mg
Suspension available as non-licensed special of 5.0mg in 5mls

b. METHYLPREDNISOLONE

60mg/m^2/day as bd dose for first 5 days and then reduce (see separate sheet).

c. MYCOPHENOLATE MOFETIL (MMF or Cellcept).

day 1 - 14	**600 mg/m^2** twice daily orally or iv (max adult dose 1000 mg twice daily)
after day 15	**300 mg/m^2** twice daily orally

Side effects (mostly diarrhoea) could be lessened by dividing the same daily dose in to 3 or 4 doses.
Give same iv/oral dose.
Dose reductions will be needed if low WBC, PLT or haemoglobin.
Available in liquid 1000 mg/5mls, 250 mg capsules or 500mg tablets

d. DOPAMINE - 2 -3 µg/kg/min

e. RANITIDINE 1 mg/kg/bd IV oral 2 mg/kg bd (until minimum steroid dose achieved) 150 mg tablets or syrup 75mg in 5 mls).

f. CO-TRIMOXAZOLE as pneumocystis prophylaxis.120-480 mg (450mg/ m^2orally) Mon, Wed, Fri bd for 3 - 6 months

g. TINZAPARIN (Innohep®) 50 I.U per kg given sub cut following vascular anastamosis, and then 24hrly for 1 week or until fully ambulant.
Multidose vial contains 10,000 I.U. per ml -2 people to check dose.
In children < 20 kg consider following this with Aspirin 37.5 mg once daily for 6 months.

h. VALGANCICLOVIR if CMV +ve donor or recipient (see above).

i. ANALGESIA

Epidural OR Morphine 10 - 20 µg/kg/hr infusion (half BW (kg) in mg in 50 ml at 1 - 2ml/hr)

TRANSPLANT TROUBLESHOOTER

The following is a guide to what measures need to be taken when vital signs fall outside the prescribed values.

	TEMP GAP	CVP	BP	
1	↑	↓	↓	**Consider Volume**
2	↑/N	↑/N	↑	**Consider Vasodilator**
3	↑/N	↑/N	↓	**Consider Dobutamine**

Volume:

5-10 mls/kg/stat N saline or 5% albumin (max bolus of 500 mls) & reassess.

- **Extreme caution where patient is 0_2 dependent - may indicate evolving Pulmonary oedema**

Vasodilators:

Hydralazine: 0.2 to 1.0 mg/kg/IV stat then hourly infusion at same dose. Nicardipine: 0.5-2□g/kg/hour more potent vasodilator than hydralazine.

Dobutamine:

10-20 ug/kg/min for hypotension/low cardiac output

INVESTIGATIONS

1. U/E, creatinine, glucose, Ca x 6 hourly for 48 hours
   x 12 hourly next 48 hours
   x daily, thereafter if situation is stable
2. Doppler renal USS post-op and repeat daily
3. FBP, DWCC, (and CD3 count if on ATG) daily.
4. Chest X-ray initially for CVP line position and daily for 2 -3 days.
5. Urine - culture, U/E and creatinine daily.
6. Twice daily weight.
7. Tacrolimus levels analysed daily Mon-Fri (BCH). When on oral drugs send daily for 10 days and thereafter Tue and Fri.
8. CMV AND EBV PCR (EDTA purple top to virology) Mondays from Day 14.
9. Tissue type bloods at 2,4,6,8 weeks -8mls (red top clotted to BCH tissue type lab).
10. DMSA scan at 1 month.

IMMUNOSUPPRESSION POST TRANSPLANT

1. STEROIDS

Day 1-5 Methylprednisolone 60 mg/m^2/day as bd dosage x 5 days (may be changed to same dose oral Prednisolone as soon as tolerated)

Day 6 Prednisolone 30 mg/m^2/day as 2 divided doses

Day 14 Prednisolone 20 mg/m^2/day as 2 divided doses

Day 21 Prednisolone 10 mg/m^2/day as 2 divided doses

Week 4-8 Prednisolone 10 mg/m^2 DAILY as one dose

Week 8-12 Prednisolone 5 mg /m^2 DAILY

Week 12+ Prednisolone 10 mg/m^2 alternate days

2. MYCOPHENOLATE MOFETIL (MMF)

day 1 - 14	**600 mg/m^2** twice daily orally or iv (max adult dose 1000 mg twice daily)
after day 15	**300 mg/m^2** twice daily orally

3. TACROLIMUS

0.3 mg/kg divided into 2 doses (each of 0.15mg/Kg) to be given orally and started within 24 hours of transplant. Observe precautions of using non PVC giving sets and syringes and flushing N/G and G tubes with water after administration:

Levels Initial target range 10 - 15 ng/ml. After six months range 5-10 ng/ml

FOLLOW UP

After discharge	- alternate days x 1 week
Until week 6	- twice weekly
Week 6 - 10	- weekly
Week 10 - 12	- fortnightly
> 3 months	- monthly
> 1 year	- 6 - 8 weekly

NB See within 4 days of any DOSE CHANGES

REJECTION

ASSESSMENT OF REJECTION

> 10% rise in serum creatinine is a significant change. It could be due to:-

1. Laboratory error.
2. Rejection
3. Tacrolimus toxicity
4. Other drug toxicity
5. Infection (especially UTI)
6. Obstruction - exclude with renal USS/MAG 3 renogram.

All such rises in creatinine should be reported to the consultant and repeated **immediately.** If a 10% rise is confirmed and rejection suspected then initial treatment would be:

Methylprednisolone 15 mg/kg/IV for 3 days (maximum 500 mg)

This should only be prescribed after discussion with Consultant.

Other causes of a rise in creatinine should be excluded and diagnosis confirmed whenever possible with a renal biopsy (need coagulation screen and Group and Hold, inform Path technician x 32534).

For patients who have received renal transplant > 1 month previously, oral Prednisolone 3 mg/kg/day is given for 3 days (maximum 150 mg).

ANTI-THYMOCYTE GLOBULIN (MERIEUX-RABBIT)

Given for steroid resistant rejection or as prophylaxis when recipient > 75% cytotoxic antibodies or second transplant when first graft lost early with rejection

Test dose: 0.1 ml (0.5 mg) in 10 ml N. Saline over 1 hour via central line

Therapeutic dose ATG

< 30 kg - 2.5 mg/kg/day

>30 kg - 1.25 - 2.5 mg/kg/day

Diluted in 100 ml saline central line over 8 hours. Chills, fevers and arthralgia common.

ANAPHYLAXIS treated with:

Hydrocortisone 100 mg IV
Chlorpheniramine 5 - 10 mg IV

Adrenaline (0.01 ml/kg of 1 in 1000, 1 m)

Prior to therapeutic dose give Chlorpheniramine IV and Hydrocortisone IV

ATG Monitoring

Aim absolute lymphocyte count 200 - 400 (omit if < 200).

CD3 count daily

(send 0.5 ml EDTA blood to RVH immunology, arrange with X 32689, ask for lymphocyte markers profile 1 which includes CD3)

Aim level 100 - 300 (omit if < 100).

Updated February 2011 Drs McKeever and O'Connor and Professor Savage

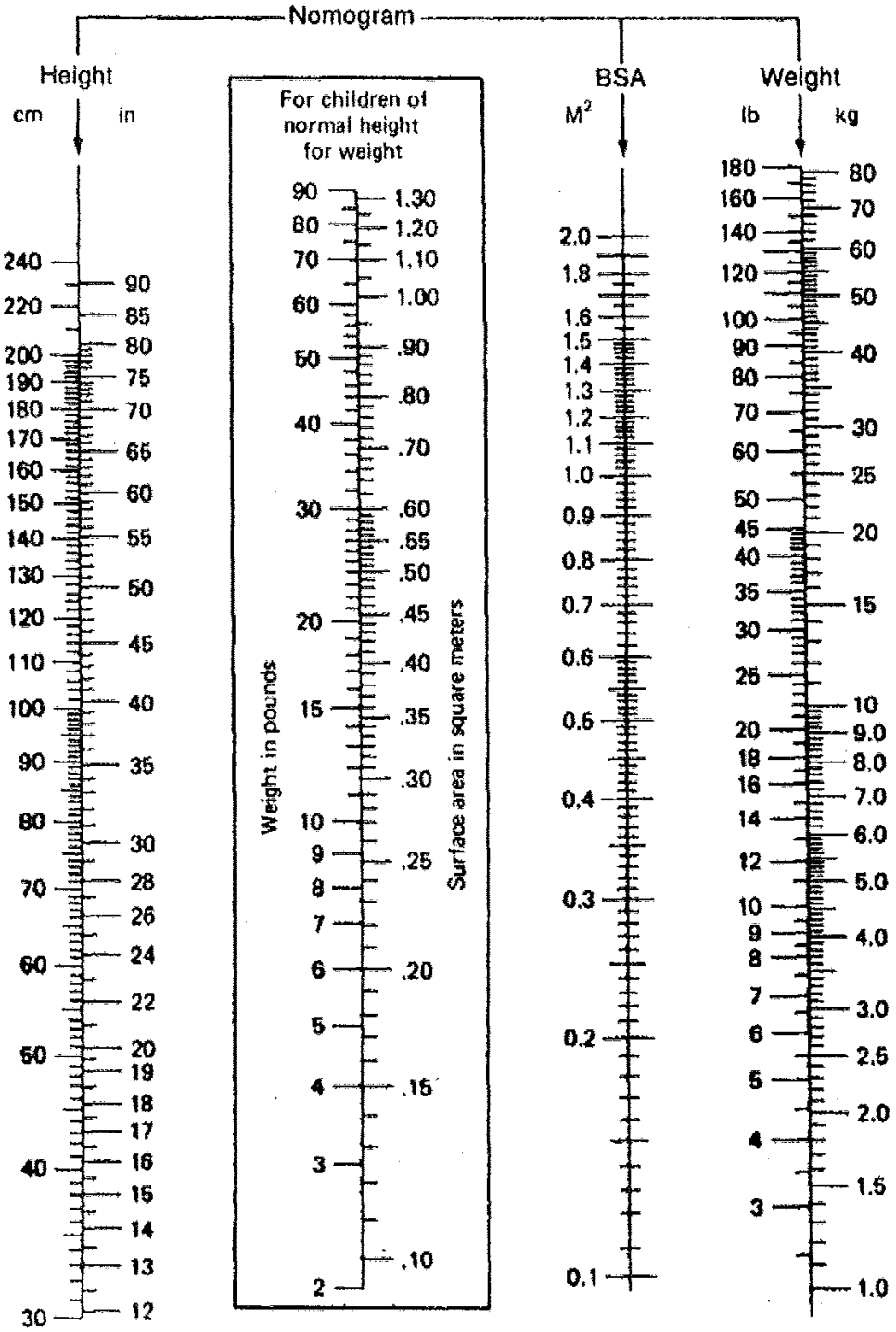

Figure 3–1. West Nomogram (for Estimation of BSA). The BSA is indicated where a straight line connecting the height and weight intersects the BSA column or, if the patient is roughly of normal proportion, from the weight alone (enclosed area). (Nomogram modified from data of E. Boyd by C. D. West; from Vaughan, V. C., and R. J. McKay, eds., *Nelson Textbook of Pediatrics*, 12th ed., Philadelphia: Saunders, 1983.)

NURSING PRE-TRANSPLANT CHECK LIST

NAME:

Height	
Weight	
BP	
MSU and urine U/E sent	
Skin swabs and Shower	
PD sample for microscopy , DWCC and culture	
Drain PD fluid	

Drug	**Given**	**Time**
Tacrolimus		
Mycophenolate Mofetil		
Basiliximab		

Does Valganciclovir need ordered according to CMV status?

Have immunosuppressive drugs and Innohep® heparin been labelled for transfer to ICU?

THEATRE CHECK LIST FOR TRANSPLANT PATIENT

NAME:

Height	
Weight	
SA	

	Donor	**Recipient**
CMV status		
EBV status		
Blood Group		

Pre theatre bloods - record time most recent serum electrolytes taken = _____

Na	Hb	PT	Urine Na
K	WCC	PTTK	
Urea	Plats		
Creat		Is blood available in theatre ?	Usual 24 hr urine output =
Ca			
Albumin			

Drugs in ward pre-transplant	**Dose**	**Given**	**Time**
Tacrolimus			
Mycophenolate Mofetil			
Basiliximab			

Prescribe drugs for use in theatre

Co-amoxiclav (on induction) (30mg/kg, max 1.2g)	
Methylprednisolone (10mg/kg, max 500mg)	
Mannitol 20% (0.5 g/kg = 2.5ml/kg)	
Heparin Tinzaparin (Innohep®) 50 I.U./kg.	

Enclosure 11

Peritoneal dialysis clinical practice guidelines for children and adolescents

Note that the current version is DRAFT number 1, 7/2/07.

This document has been adapted for paediatric patients from the Renal Association Standards for Peritoneal Dialysis in Adults (www.renal.org/guidelines/index.html) which was written by Prof Simon Davies. Although the large majority of standards for adults also apply to children, there are some areas that differ significantly. There are also some important areas not covered in the adult standards, such as the specific requirements of the growing child and the need for a structured process for transfer of adolescents to adult services. For this reason, the renal association adult guidelines have been adapted where necessary. All standards that are taken directly from the adult guidelines are shown in regular font and paediatric guidelines are in red italics.

The lead author of this paediatric guideline was Dr Lesley Rees, along with Dr Sally Feather and Dr Rukshana Shroff. Please send feedback to L.Rees@ich.ucl.ac.uk

CONTENTS

Summary of Clinical Practice Guidelines for Peritoneal Dialysis

1. Equipment and Resources

1.1 The dialysis unit should have sufficient specialist support staff to fulfil the criteria listed by the Renal Workforce Planning Group 2002.

Paediatric standard 1
PD for children should take place in specialised paediatric centres able to provide multidisciplinary support

1.2 Access to other paediatric sub-speciality services should be easily available.

1.3 Adolescents need to be prepared for transfer to adult services. It is important that the process is begun in good time, and that there is an appropriate transfer policy that is agreed by both the referring and receiving centres.

Paediatric standard 2
A transfer process for adolescents must be in place and agreed by referring and receiving units

1.4 Peritoneal dialysis should be delivered in the context of a comprehensive and integrated service for renal replacement therapies, including haemodialysis (including temporary backup facilities), transplantation and conservative care. Both continuous ambulatory peritoneal dialysis (CAPD) and automated peritoneal dialysis (APD), in all its forms should be available. Dedicated PD nursing staff (1 W.T.E. per 20 patients) should be part of the multidisciplinary team

1.5 All equipment used in the delivery and monitoring of therapies should comply with the relevant standards for medical electrical equipment [BS-EN 60601-2-39:1999, BS5724-2-39:1998, IEC 60601-2-39:1998, Particular requirements for the safety – specification for peritoneal dialysis equipment]. Tubing sets and catheters should carry the "CE" mark to indicate that the item conforms to the essential requirements of the Medical Devices Directive (93/42/EEC) and that its conformity has been assessed in accordance with the directive.

1.6 Fluids for peritoneal dialysis are required to satisfy the current European quality standards as indicated in the European good manufacturing practice and the European Pharmacopoeia Monograph "Solutions for Peritoneal Dialysis". Manufacturing facilities are required to meet the relevant standards (ISO 9001/2 and EN 46001/2). Product registration files must be submitted to and product approval given by the Medicines Control Agency.

1.7 The use of disconnect systems should be standard unless clinically contraindicated.

1.8 Biocompatible PD solutions (normal pH, low concentrations of glucose degradation products) should be used in patients experiencing infusion pain. *Also, they should be considered in patients who are likely to remain on PD for a significant period of time, (e.g. those who do not have a suitable living related donor and poor matchability scores, thus anticipating a longer than usual wait for a deceased donor kidney).*

2. Preparation for Peritoneal Dialysis

2.1 All patients should, where possible, be adequately prepared for renal replacement therapy and this should include receiving information and education about PD

treatment, delivered by experienced members of the MDT *who have paediatric renal training.* Patients commencing RRF in an unplanned fashion for whatever reason should receive this information once appropriate.

2.2 Where possible, timing of PD catheter insertion should be planned to accommodate patient convenience, commencement of training between 10 days and 6 weeks and before RRT is essential to enable correction of early catheter-related problems without the need for temporary haemodialysis.

2.3 Dialysis centres should have a dedicated team approach to catheter insertion. This is more important than the type of catheter or the implantation technique used. *In children, the most important thing is that the surgeon undertaking the procedure is appropriately trained and skilled.*

Paediatric standard 3
Peritoneal dialysis catheter insertion should be undertaken by appropriately trained and skilled staff.

2.4 Peri-operative catheter care and catheter complications (leaks, hernias, obstruction) should be managed according to the International Society of Peritoneal Dialysis guidelines

3. Solute Clearance

3.1 Both residual urine and peritoneal dialysis components of small solute clearance should be measured at least six monthly or more frequently if clinically indicated. Both urea and/or creatinine clearances can be used to monitor dialysis adequacy and should be interpreted within the limits of the methods.

3.2 A combined urinary and peritoneal Kt/Vurea of 1.7/week or a creatinine clearance of 50L/week/1.73m2 should be considered as minimal treatment doses. The dose should be increased in patients experiencing uraemic symptoms.

Paediatric standard 4
A Kt/Vurea of 1.7/week and creatinine clearance 50L/week/1.73m^2 should be the minimum for children.

4. Ultrafiltration and fluid management

4.1 Peritoneal membrane function should be monitored regularly (6 weeks after commencing treatment and at least annually or when clinically indicated) using a peritoneal equilibration test (PET) or equivalent. Daily urine and peritoneal ultrafiltration volumes, with appropriate correction for overfill, should be monitored at least six-monthly.

4.2 Dialysis regimens resulting in fluid re-absorption should be avoided. Patients with high or high average solute transport, at greatest risk of this problem, should be considered for APD and icodextrin use.

4.3 Dialysis regimens resulting in routine utilisation of hypertonic (3.86%) glucose exchanges should be avoided. Where appropriate this should be achieved by using icodextrin or diuretics.

4.4 Treatment strategies that favour preservation of renal function should be adopted where possible. These include avoidance of episodes of dehydration, and the use of diuretics, ACEi and ARBs.

4.5 Anuric patients who consistently achieve a daily ultrafiltration of less than 750 ml/1.73m^2 should be closely monitored and the benefits of modality switch considered.

5. Infectious complications

1. Prevention Strategies

5.1.1 PD units should undertake regular audit of their peritonitis and exit-site infection rates, including causative organism, treatment and outcomes. They should enter into active dialogue with their microbiology department and infection control team to develop optimal local treatment and prevention protocols.

Paediatric standard 5
All units should collect and audit data on the incidence of exit site infection.

Paediatric standard 6
All units should collect and audit data on the incidence of peritonitis.

5.1.2 Flush-before-fill dialysis delivery systems should be used.

5.1.3 Patients should undergo regular revision of their technique and receive intensified training if this is below standard.

5.1.4 Initial catheter insertion should be accompanied by antibiotic prophylaxis.

5.1.5 Invasive procedures should be accompanied by antibiotic prophylaxis and emptying the abdomen of dialysis fluid for a period commensurate with the procedure.

5.1.6 Topical antibiotic administration should be used to reduce the frequency of *Staph. aureus* and Gram negative exit-site infection and peritonitis.

2. Treatment

5.2.1 Exit site infection is suggested by pain, swelling, crusting, erythema and serous discharge; purulent discharge always indicates infection. Swabs should be taken for culture and initial empiric therapy should be with oral antibiotics that will cover *S. aureus and P. aeruginosa. If there is pain and redness that is tracking along the tunnel, particularly if associated with an exit site infection, a tunnel infection should be suspected. An exit site swab should be taken and the PD fluid sent for microscopy and culture. An ultrasound of the tunnel may show evidence of inflammation. A tunnel infection that fails to respond to antibiotics is an indication for removal of the PD catheter.*

5.2.2 Methicillin resistant organisms (MRSA) will require systemic treatment (e.g vancomycin) and will need to comply with local infection control policies.

5.2.3 Initial treatment regimens for peritonitis should include cover for bacterial Gram positive and Gram negative organisms until result of culture and antibiotic sensitivities are obtained.

6. Metabolic Factors

6.1 Standard strategies to optimise diabetic control should be used; these should be complemented by dialysis prescription regimens that minimise glucose, including glucose free solutions (icodextrin and amino-acids), where possible.

6.2 Central obesity can worsen or develop in some PD patients. The risk of this problem, and associated metabolic complications, notably increased atherogenicity of lipid profiles and insulin resistance, can be reduced by avoiding excessive glucose prescription and using icodextrin.

6.3 Awareness of the effects of Icodextrin on assays for estimation of amylase and glucose (using glucose dehydrogenase) should be disseminated to patients, relatives, laboratory and clinical staff.

7. Laboratory and clinical indices

7.1 Monitoring of biochemical and haematological parameters should be performed monthly or at each clinic visit if less often than monthly

7.2 Serum bicarbonate concentrations should be between 20 and 26mmol/l.

7.3 Serum potassium should be between 3.5 and 6.5mmol/l.

7.4 Serum phosphate should be within, and preferably nearer to the 50th centile, for the age appropriate normal range (appendix, page 37).

7.5 Serum calcium, adjusted for serum albumin, should be within the age appropriate normal range.

7.6 Serum albumin corrected calcium x phosphate product should be less than *<4.5 mmol^2/L^2 (K/DOQI guidelines) or <5 mmol^2/L^2 (European PD working group advice).*

7.7 The optimum range for serum PTH levels is controversial. There is emerging evidence that levels should be maintained at less than twice the upper limit of normal for the intact PTH assay used.

7.8 *Although aluminium is not a recommended phosphate binding agent in children, the* serum aluminium concentration should be measured every three months in all patients receiving oral aluminium hydroxide. No patient whose ferritin level is <100 µg/l should have a serum aluminium concentration >60 µg/l (2.2 µmol/l).

7.9 Pre-dialysis haemoglobin concentration should be greater than the lower limit of the age appropriate normal range.

7.10 Ferritin levels should be between 100 and 800mcg/L. However, given the increased risk of thrombotic events with higher ferritin levels that have been shown in recent trials, the revised K/DOQI guidelines for the management of anaemia recommend that the serum ferritin levels should be maintained between 100 – 500mcg/L in adults. In the absence of paediatric studies, in patients at risk of thrombosis (e.g. those with heavy proteinuria, arteriovenous fistulae or synthetic grafts), serum ferritin levels above 500mcg/L should be avoided.

7.11 Height and head circumference (in those under 2 years of age) should be measured monthly, and the rate of growth checked against normal centiles. Dry weight should be estimated regularly, at least monthly or every 2 weeks in infants. Pubertal stage should be assessed every 3 months in those over 10 years of age, or sooner if clinically indicated.

Paediatric standard 7

Growth and development should be measured regularly as part of the assessment of dialysis adequacy

Paediatric standard 8
Dry weight needs regular reassessment in the growing child

7.12 An assessment of school progress, both in the hospital and locally, should be made annually.

7.13 Blood pressure should be maintained within the age appropriate normal range.

8. Access to and withdrawal from dialysis

8.1 All children with chronic kidney disease should be considered for renal replacement therapy by stage 4

CKD should be suspected in children with: bilateral renal anomalies on antenatal scans (many children with CKD are now diagnosed antenatally); a creatinine above the normal age appropriate range; bilateral renal defects on scans e.g. for UTI; a family history of CKD; persistent proteinuria; or after an episode of acute renal failure. All such children should be referred to a paediatric nephrologist. Early referral provides the opportunity for delaying the progression of CKD by treating hypertension and proteinuria, for optimising growth and preventing renal bone disease. Importantly, it also allows for timely forward planning for renal replacement therapy.

8.2 If there is no medical contraindication the choice of initial dialysis modality should be based on patient choice. However, although patient choice is paramount, guidance from unit staff is necessary: venous access can be difficult to achieve and maintain in those less than 5 years of age, and needling of a fistula can be particularly difficult in an uncooperative patient. Also, PD is likely to preserve residual renal function for longer. For these reasons, as well as social ones, PD is recommended in young children.

8.3 After full education and counselling a small proportion of families may opt for active non-dialytic management of advanced chronic kidney disease, including nutritional, medical and psychological support, rather than plan to initiate dialysis. *This decision may need to be discussed with an independent ethics advisor if a consensus on further management is not reached amongst members of the medical and nursing teams or amongst the parents and medical teams.* The numbers of patients not taken on to dialysis and the reasons for this decision should be subject to audit.

8.4 Renal replacement therapy should commence when a patient with an eGFR < 15ml/min/1.73m^2 has symptoms or signs of uraemia, fluid overload, malnutrition *and/or growth failure* in spite of medical therapy or before an asymptomatic patient has an eGFR < 6ml/min/1.73m^2.

8.5 Any decision to discontinue dialysis should be made jointly by the patient (when age appropriate) and their carers and the responsible consultant nephrologist and renal team and the family practitioner. The decision, and the reasons for it, must be recorded in the patient's notes. Renal units should develop guidelines for palliative care of such patients, including liaison with community services.

9. Summary of the most important paediatric clinical practice guidelines

Paediatric standard 1
PD for children should take place in specialised paediatric centres able to provide multidisciplinary support (good practice)

Paediatric standard 2
A transfer process for adolescents must be in place and agreed by referring and receiving units (good practice)

Paediatric standard 3
Peritoneal dialysis catheter insertion should be undertaken by appropriately trained and skilled staff (good practice)

Paediatric standard 4
A Kt/Vurea of 1.7/week and creatinine clearance 50L/week/1.73m^2 should be the minimum for children (good practice)

Paediatric standard 5
All units should collect and audit data on the incidence of exit site infection (good practice)

Paediatric standard 6
All units should collect and audit data on the incidence of peritonitis (good practice)

Paediatric standard 7
Growth and development should be measured regularly as part of the assessment of dialysis adequacy (good practice)

Paediatric standard 8

Dry weight needs regular reassessment in the growing child (good practice). A paediatric renal dietician should regularly review patients and provide guidelines on the nutritional requirement in each case.

Summary of Audit Measures for Peritoneal Dialysis

1. *Adequacy of staffing levels (medical, surgical, radiological, anaesthetic, nursing, dietetic, play therapists, psychosocial, pharmacy, and schooling)*
2. *Presence of a transfer process for adolescents that is agreed by referring and receiving units*
3. Availability of modality choice
4. Monitoring of modality switching
5. Systems in place to check medical equipment
6. Systems in place to ensure purchase of dialysis fluid fulfil legal requirements
7. Use of non-standard systems with documentation of clinical indication
8. Use of biocompatible solutions and indication for use
9. Audit of care pathway for dialysis preparation to include information given, when and who delivers it.
10. Audit of care pathway for catheter insertion to include timeliness and need for temporary haemodialysis
11. Catheter complications and their resolution
12. Frequency of solute clearance (residual and peritoneal) estimation
13. Cumulative frequency curves for the total solute clearance
14. Frequency of measurement of membrane function, residual urine and peritoneal ultrafiltration volume
15. Identify patients with fluid reabsorption in long dwell
16. Routine annual audit of infection prevention strategies
17. Routine annual audit of infection outcomes (exit site and peritonitis rates)
18. Cumulative frequency curves of plasma bicarbonate
19. Processes in place to increase awareness of interference of assays by icodextrin metabolites
20. *Cumulative frequency curves of pre-dialysis serum calcium, phosphate calcium x phosphate product and PTH concentration*
21. *Cumulative frequency curves of pre-dialysis haemoglobin concentration*
22. *Height, weight, head circumference and pubertal progression*
23. *School attendance*
24. *Cumulative frequency curves of BP predialysis*
25. *An audit of cases where PD was refused or withdrawn and the indications for this.*

Rationale for clinical practice guidelines for paediatric patients on peritoneal dialysis

Introduction

In the UK peritoneal dialysis is usually the first choice dialysis modality because it interferes less with the child's day-to-day life, particularly in those who may live a long way from their paediatric renal unit. The ratio of children on peritoneal dialysis compared to haemodialysis is approximately 2:1; of the 173 children who received dialysis in the UK in 2003, 62% were on peritoneal dialysis (Report of the Paediatric Renal Registry, Seventh Annual Report of the UK Renal Registry, December 2004).

The National Service Framework Part 1: Dialysis and Transplantation is a document that is applicable to both adult and paediatric services (1). It stresses the importance of a family-centred approach to the care of children. It also stresses the importance of the team needed to provide such services i.e. medical, surgical, anaesthesiology, radiology, nursing, dietetic, play therapy, psychological, social work and pharmacists, all of whom need the special skills necessary to treat such children. However, this document was not designed to set standards for clinical care.
Owing to the small numbers of children with CKD stage 5, and, in particular, the very small numbers on dialysis, there are very few analyses to help in the management of paediatric patients, and paediatric nephrologists have to rely on extrapolation of data from adult studies. Clinical practice guidelines for adults on peritoneal dialysis have been developed in Australasia, Canada, Europe and the USA as well as the UK (2-11). Guidelines for children on peritoneal dialysis have also been published (12-17).

These guidelines serve to identify and promote best practice in the delivery of haemodialysis and have set clinical standards to allow comparative audit of the key aspects of the haemodialysis prescription, laboratory data and patient outcomes. The reports of the UK Renal Registry, Scottish Renal Registry and NHS Quality Improvement Scotland have demonstrated the benefits of performing regular audit to improve clinical standards in haemodialysis (2-4).

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1. Equipment and Resources

1.1 Peritoneal Dialysis should be delivered in the context of a comprehensive and integrated service for renal replacement therapies, including haemodialysis (including temporary backup facilities), transplantation and conservative care. Both continuous ambulatory peritoneal dialysis (CAPD) and automated peritoneal dialysis (APD), in all its forms should be available. Dedicated PD nursing staff (1 W.T.E. per 20 patients) should be part of the multidisciplinary team (Good Practice)

Evidence from observational studies or registry data, with all its limitations, indicates that peritoneal dialysis (PD) used in the context of an integrated dialysis programme is associated with good clinical outcomes, certainly comparable to haemodialysis in the medium term (HD) (1-5). The only randomised study (NECOSAD), comparing HD to PD as a first treatment showed no differences in 2 year quality adjusted life years or 5 year mortality, but the number randomised was insufficient to generalize this observation; notably, most patients in this national study had sufficient life-style preferences related to one modality to decline randomisation (6). PD has a significant technique failure rate however, so patients need to be able to switch treatment modality (to either temporary or permanent HD) in a timely manner, which has implications for HD capacity.

PD modalities (CAPD v. APD) have a different impact on life-style; one randomised study found that APD creates more time for the patient to spend with family or continue employment but is associated with reduced quality of sleep (7). APD is the preferred modality for children. There are medical indications for APD (see sections 2, 3 and 4), but generally modality choice is a lifestyle issue.

The success of a PD programme is dependent upon specialized nurses with appropriate skills in assessing and training patients for PD, monitoring of treatment and with sufficient resources to provide continued care in the community. A recent randomised trial of more intensive training has shown that this reduces peritonitis risk (8) (see section 5). Several studies have documented the benefits of home visits in identifying new problems, reducing peritonitis and non-compliance (9-11). It is usually possible for a WTE PD nurse to deliver this quality of care with a case load of 20 PD patients (see recommendations of the National Renal Workforce Planning Group, 2002).

Audit measure 1 - Adequacy of staffing levels (medical, surgical, radiological, anaesthetic, nursing, dietetic, play therapists, psychosocial, pharmacy, and schooling)

Audit measure 2 - Presence of a transfer process for adolescents that is agreed by referring and receiving units

Audit measure 3 - Availability of modality choice
Audit measure 4 - Monitoring of modality switching

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1.2 All equipment used in the delivery and monitoring of therapies should comply with the relevant standards for medical electrical equipment [BS-EN 60601-2-39:1999, BS5724-2-39:1998, IEC 60601-2-39:1998, Particular requirements for the safety – specification for peritoneal dialysis equipment]. Tubing sets and catheters should carry the "CE" mark to indicate that the item conforms to the essential requirements of the Medical Devices Directive (93/42/EEC) and that its conformity has been assessed in accordance with the directive.

Audit Measure 5 - Systems in place to check medical equipment

This is a legal requirement

1.3 Fluids for peritoneal dialysis are required to satisfy the current European quality standards as indicated in the European good manufacturing practice and the European Pharmacopoeia Monograph "Solutions for Peritoneal Dialysis". Manufacturing facilities are required to meet the relevant standards (ISO 9001/2 and EN 46001/2). Product registration files must be submitted to and product approval given by the Medicines Control Agency.

Audit Measure 6 - Systems in place to ensure purchase of dialysis fluid fulfil legal requirements.

1.4 The use of disconnect systems should be standard unless clinically contraindicated (evidence).

Audit Measure 7 - Use of non-standard systems with documentation of clinical indication

Disconnect systems have been shown through randomised trials to be associated with a lower peritonitis risk, especially in infections due to touch contamination (1)

1. MacLeod A, Grant A, Donaldson C, et al. Effectiveness and efficiency of methods of dialysis therapy for end-stage renal disease: systematic reviews. *Health Technol Assess* 1998;2(5):1-166.

1.5 Biocompatible PD solutions (normal pH, low concentrations of glucose degradation products) should be used in patients experiencing infusion pain (evidence). Also, they should be considered in patients who are likely to remain on PD for a significant period of time (those who do not have a suitable living related donor and poor matchability scores, thus anticipating a longer than usual wait for a deceased donor kidney).

Audit Measure 8 - Use of biocompatible solutions and indication for use

A minority of patients commencing PD will experience infusion pain, often severe enough to consider discontinuing the therapy. A double blind randomised study demonstrated that pain could be prevented by using a normal pH, bicarbonate-lactate buffered dialysis fluid (Physioneal) (1). Subsequent clinical experience has found that the benefit of this more biocompatible solution on infusion pain results in immediate and sustained benefit, and is probably applicable to other biocompatible solutions.

The evidence of clinical benefit from the routine use of biocompatible solutions is more controversial. Standard solutions are clearly bio-incompatible, with low pH (~5.2), lactate rather than bicarbonate buffer, high osmolality and high concentrations of glucose which also result in high concentrations of glucose degradation products (GDPs). Many *in vitro* and *ex vivo* studies have demonstrated the relative toxicity of these solutions, with all of the biocompatible features playing their part (2-7). There is also strong observational evidence that (a) detrimental functional changes to the membrane occur with time on treatment, which are more exaggerated in patients using solutions with high glucose concentration early in their time on therapy (8, 9) and (b) morphological changes occur that are related to time on treatment which include membrane thickening and vascular scarring (10). Time on treatment is also the greatest risk factor for encapsulating peritoneal sclerosis (EPS) (11, 12).

These observations have led all the main dialysis companies to develop and market 'biocompatible' solutions, with normalization of pH, reduction of GDPs and a variable approach to buffering. In randomised clinical trials these solutions have been shown to improve the dialysate concentrations of biomarkers considered to be indicators of mesothelial cell and possibly membrane health (13-16). Systemic benefits possibly include reduced circulating advanced glycation end-products (16) and better glycaemic control in diabetics (17). *However, the long-term effects of metabolic alkalosis on the developing skeleton are not known, and an alkaline pH can predispose to soft tissue calcification.* Data is currently lacking on hard clinical endpoints such as technique failure, functional membrane change or patient survival. One non-randomised study has found an improved patient but not technique survival; patients in this study using biocompatible solutions were younger, suggesting a selection bias that may not be fully adjusted for, so caution should be exercised in the interpretation of this study (18).

Currently there is insufficient evidence to recommend that all patients should be treated with biocompatible solutions, especially as this may have a significant cost implication. A selective approach to their use should be considered. Working on the assumption

that the primary benefit of biocompatible solutions is membrane protection then there is evidence indicating that function membrane changes become more significant at 4 years of treatment, even in patients commencing PD with good residual renal function and low use of hypertonic exchanges (9). Likewise the incidence of EPS is rare before this period of time on treatment. This issue remains controversial at this stage and further studies are required.

An area of difference between paediatric and adult PD is that fill volumes vary with size. Surface area is preferable to body weight, which may underestimate the optimal fill volume in younger children, and should be between 1200 and 1400ml dialysate/m^2 body surface area (19).

1. Mactier RA, Sprosen TS, Gokal R, et al. Bicarbonate and bicarbonate/lactate peritoneal dialysis solutions for the treatment of infusion pain. *Kidney Int* 1998;53(4):1061-7.
2. Liberek T, Topley N, Jorres A, et al. Peritoneal dialysis fluid inhibition of polymorphonuclear leukocyte respiratory burst activation is related to the lowering of intracellular pH. *Nephron* 1993;65(2):260-5.
3. Jorres A, Bender TO, Finn A, et al. Biocompatibility and buffers: effect of bicarbonate-buffered peritoneal dialysis fluids on peritoneal cell function. *Kidney Int* 1998;54(6):2184-93.
4. Jörres A, Topley N, Steenweg L, Müller C, Köttgen E, Gahl GM. Inhibition of cytokine synthesis by peritoneal dialysate persists throughout the CAPD cycle. *Am J Nephrol* 1992;12(1-2):80-5.
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9. Davies SJ. Longitudinal relationship between solute transport and ultrafiltration capacity in peritoneal dialysis patients. *Kidney Int* 2004;66:2437-45.
10. Williams JD, Craig KJ, Topley N, et al. Morphologic changes in the peritoneal membrane of patients with renal disease. *J Am Soc Nephrol* 2002;13(2):470-9.
11. Rigby RJ, Hawley CM. Sclerosing peritonitis: the experience in Australia. *Nephrol Dial Transplant.* 1998;13(1):154-9.
12. Lee HY, Kim BS, Choi HY, et al. Sclerosing encapsulating peritonitis as a complication of long-term continuous ambulatory peritoneal dialysis in Korea. *Nephrology (Carlton).* 2003;8(Suppl):S33-9.
13. Rippe B, Wieslander A, Musi B. Long-term results with low glucose degradation product content in peritoneal dialysis fluids. *Contrib Nephrol* 2003(140):47-55.
14. Jones S, Holmes CJ, Krediet RT, et al. Bicarbonate/lactate-based peritoneal dialysis solution increases cancer antigen 125 and decreases hyaluronic acid levels. *Kidney Int* 2001;59(4):1529-38.
15. Jones S, Holmes CJ, Mackenzie RK, et al. Continuous dialysis with bicarbonate/lactate-buffered peritoneal dialysis fluids results in a long-term improvement in ex vivo peritoneal macrophage function. *J Am Soc Nephrol* 2002;13(Suppl 1):S97-103.
16. Williams JD, Topley N, Craig KJ, et al. The Euro-Balance Trial: the effect of a new biocompatible peritoneal dialysis fluid (balance) on the peritoneal membrane. *Kidney Int* 2004;66(1):408-18.
17. Marshall J, Jennings P, Scott A, Fluck RJ, McIntyre CW. Glycemic control in diabetic CAPD patients assessed by continuous glucose monitoring system (CGMS). *Kidney Int* 2003;64(4):1480-6.
18. Lee HY, Park HC, Seo BJ, et al. Superior patient survival for continuous ambulatory peritoneal dialysis patients treated with a peritoneal dialysis fluid with neutral pH and low glucose degradation product concentration (Balance). *Perit Dial Int.* 2005;25(3):248-55.

19. White CT, Gowrishankar M, Feber J, Yiu V; Canadian Association of Pediatric Nephrologists (CAPN); Peritoneal Dialysis Working Group. Clinical practice guidelines for pediatric peritoneal dialysis. Pediatr Nephrol. 2006 Aug;21(8):1059-66

2. Preparation for Peritoneal Dialysis

2.1 All patients should, where possible, be adequately prepared for renal replacement therapy and this should include receiving information and education about PD treatment, delivered by an experienced member of the MDT. Patients commencing RRF in an unplanned fashion for whatever reason should receive this information once appropriate. (Good practice)

Audit Measure 9: Audit of care pathway for dialysis preparation to include information given, when and who delivers it.

The arguments and rationale for this guideline relate to the National Service Framework for Renal Services, Part 1. The reader is referred to standard 2, Preparation and Choice pp. 21-23.

2.2 Where possible, timing of PD catheter insertion should be planned to accommodate patient convenience, commencement of training between 10 days and 6 weeks, (unless using the Moncrief catheter) and before RRT is essential to enable correction of early catheter-related problems without the need for temporary haemodialysis. (Good practice)

Audit Measure 10: Audit of care pathway for catheter insertion to include timeliness and need for temporary haemodialysis

The arguments and rationale for this guideline relate to the National Service Framework for Renal Services, Part 1. The reader is referred to standard 3, Elective Dialysis Access Surgery, pp. 24-26. The Moncrief catheter is buried subcutaneously and is designed to be left in this position, where it can remain for many months, until required (1). *If the catheter needs to be used within 7 days of insertion, fill volumes should start at 500ml/m^2 body surface area in order to reduce the chances of dialysate leak, with its associated risk of tunnel and peritoneal infection.*

1. Gokal R, Alexander S, Ash S, et al. Peritoneal catheters and exit-site practices toward optimum peritoneal access: 1998 update. (Official report from the International Society for Peritoneal Dialysis). *Perit Dial Int* 1998;18(1):11-33.

2. *White CT, Gowrishankar M, Feber J, Yiu V; Canadian Association of Pediatric Nephrologists (CAPN); Peritoneal Dialysis Working Group. Clinical practice guidelines for pediatric peritoneal dialysis. Pediatr Nephrol. 2006 Aug;21(8):1059-66*

2.3 Dialysis centres should have a dedicated team approach to catheter insertion. This is more important than the type of catheter or the implantation technique used. (Good practice)

An experienced team approach to catheter insertion is recommended by all available guidelines; in the case of the European guidelines this is given a level A evidence although no randomised trial has been published comparing *ad hoc* arrangements with those of a dedicated experienced team (1). This approach should be combined with regular audit of outcomes. Several randomised trials have been performed comparing

different catheter designs and insertion techniques. These are fully reviewed elsewhere (1-4). Whilst there are theoretical advantages in choosing different catheters, e.g. double *v.* single cuff to reduce leakage, coiled *v.* straight to reduce catheter migration, when put to the test in randomised trials no significant benefit of one over another has been demonstrated. Equally, there may be clear logistic benefits of one approach to catheter insertion over another, e.g. laparoscopic *v.* open surgical *v.* Seldinger that reflect local expertise and facilities but no studies have demonstrated a clear benefit. Evidence would suggest that a downwards-directed exit site is associated with less infection and a caudally directed angle of the catheter in the deep tunnel, especially if this is made through the rectus muscle, is associated with reduced likelihood of catheter migration (5).

Similarly for children, there is no evidence showing any difference in the incidence of complications and the number of cuffs. However, in young children care is necessary to avoid placement of the distal cuff too near the exit site as cuff extrusion can occur. For this reason, it is recommended that there should be at least a 2cm distance between the distal cuff and the exit site. There is also no evidence to support the use of swan necked in comparison to straight catheters or a coiled in comparison to a straight intraperitoneal segment. However, downward or lateral pointing exit sites have been shown to be associated with a decreased incidence of peritonitis in 2 studies (6). Furthermore, there is no evidence in the paediatric literature to demonstrate any benefit of omentectomy, although there is some evidence in adults. The most important issue, therefore, is that the catheter is inserted by experienced staff who are aware of these issues.

Paediatric standard 3
Peritoneal dialysis catheter insertion should be undertaken by appropriately trained and skilled staff (good practice)

1. Dombros N, Dratwa M, Feriani M, et al. European best practice guidelines for peritoneal dialysis. 3 Peritoneal access. *Nephrol Dial Transplant.* 2005;20(Suppl 9):ix8-ix12.
2. Gokal R, Alexander S, Ash S, et al. Peritoneal catheters and exit-site practices toward optimum peritoneal access: 1998 update. (Official report from the International Society for Peritoneal Dialysis). *Perit Dial Int* 1998;18(1):11-33.
3. Canadian Guidelines for treatment wth peritoneal dialysis. *J Am Soc Nephrol* 1999;Suppl 13.
4. Flanigan M, Gokal R. Peritoneal catheters and exit-site practices toward optimum peritoneal access: a review of current developments. *Perit Dial Int.* 2005;25(2):132-9.
5. Crabtree JH, Burchette RJ, Siddiqi NA. Optimal peritoneal dialysis catheter type and exit site location: an anthropometric analysis. *ASAIO J.* 2005;51(6):743-7.
6. White CT, Gowrishankar M, Feber J, Yiu V; Canadian Association of Pediatric Nephrologists (CAPN); Peritoneal Dialysis Working Group. Clinical practice guidelines for pediatric peritoneal dialysis. Pediatr Nephrol. 2006 Aug;21(8):1059-66

2.4 Peri-operative catheter care and catheter complications (leaks, hernias, obstruction) should be managed according to the International Society of Peritoneal Dialysis guidelines, www.ispd.org (Good practice)

Audit Measure 11: Catheter complications and their resolution

For management of the catheter in the peri-operative period, for catheter related problems including leak (internal and external), poor flow, obstruction and hernias the guidelines developed by the International Society of Peritoneal Dialysis should be used, www.ispd.org (1, 2). Catheter problems due to increased intra-peritoneal pressure, especially leaks, hernias and prolapse are an important medical indication for the use of APD either temporarily or permanently; poor flow or catheter related flow pain should be treated with tidal APD.

1. Flanigan M, Gokal R. Peritoneal catheters and exit-site practices toward optimum peritoneal access: a review of current developments. *Perit Dial Int.* 2005;25(2):132-9.
2. Crabtree JH. Rescue and salvage procedures for mechanical and infectious complications of peritoneal dialysis. *Int J Artif Organs.* 2006;29(1):67-84.

3. Solute Clearance

3.1 Both residual urine and peritoneal dialysis components of small solute clearance should be measured at least six monthly or more frequently if clinically indicated. Both urea and/or creatinine clearances can be used to monitor dialysis adequacy and should be interpreted within the limits of the methods. (Good practice)

Audit Measure 12: Frequency of solute clearance (residual and peritoneal) estimation.

Small solute clearance is one of the measurements of adequate dialysis treatment. Salt and water removal and acid-base balance are considered in sections 4 and 6 respectively. There are two issues in measuring small solute clearance that need to be taken into consideration. First, the relationship to clinical outcomes of residual renal versus peritoneal small solute clearance is quantitatively different. Observational studies have shown that preserved renal clearance, in fact just urine volume, is associated with improved survival, independent of other known factors such as age and comorbidity (1, 2). Randomised controlled trials designed to replace this residual renal function with peritoneal clearance did not show a proportional survival benefit (3, 4). Second, there are two potential surrogate solutes, urea and creatinine, that can be used to measure solute clearance in PD patients. There is no clear evidence as to which is the more useful clinically, and both have their problems. Current advice, therefore, is that either or both can be used, but clinicians should be aware of their differing limitations. Urea clearances are limited by the difficulty in PD patients of estimating V accurately, whilst peritoneal creatinine clearances are affected by membrane transport characteristics (see Appendix).

1. Churchill DN, Taylor DW, Keshaviah PR. Adequacy of dialysis and nutrition in continuous peritoneal dialysis: association with clinical outcome. *J Am Soc Nephrol* 1996;7:198-207.
2. Bargman JM, Thorpe KE, Churchill DN. Relative contribution of residual renal function and peritoneal clearance to adequacy of dialysis: a reanalysis of the CANUSA study. *J Am Soc Nephrol* 2001;12(10):2158-62.
3. Paniagua R, Amato D, Vonesh E, et al. Effects of Increased Peritoneal Clearances on Mortality Rates in Peritoneal Dialysis: ADEMEX, a Prospective, Randomized, Controlled Trial. *J Am Soc Nephrol* 2002;13(5):1307-20.
4. Lo WK, Ho YW, Li CS, et al. Effect of Kt/V on survival and clinical outcome in CAPD patients in a randomized prospective study. *Kidney Int* 2003;64(2):649-56.

3.2 A combined urinary and peritoneal Kt/Vurea of 1.7/week or a creatinine clearance of 50L/week/1.73m2 should be considered as minimal treatment doses. The dose should be increased in patients experiencing uraemic symptoms (Evidence)

Audit Measure 13: Cumulative frequency curves for the total solute clearance

Two randomised controlled trials (ADEMEX and Hong Kong) have evaluated the impact of peritoneal solute clearances on clinical endpoints (1, 2). Neither found that an increase of peritoneal Kt/V_{urea} >1.7 was associated with an improvement in survival. Only one of these studies (ADEMEX) measured creatinine clearance, which was the solute used to make decisions in this case; patients in the control group achieved an average peritoneal creatinine clearance of 46L/1.73m^2/week and a total (urine plus renal) of 54L/1.73m^2/week. In setting a recommendation for minimal peritoneal clearances, to be achieved in anuric patients, the previous Renal Association guideline of Kt/V > 1.7 and creatinine clearance >50L/1.73m^2/week is supported by both the randomised and observational data. In the Hong Kong study, patients randomised to a Kt/V <1.7, whilst their mortality was not significantly worse they had a significantly higher drop out rate, more clinical complications and worse anaemia. One observational longitudinal study demonstrated that patients develop malnutrition once the Kt/V falls below 1.7 with a three-fold increase in the death rate (3). The NECOSAD study found that a creatinine clearance of <40L/wek or a Kt/V urea <1.5 was associated with increased mortality in anuric patients (4).

The vast majority of PD patients will be able to reach these clearance targets, especially if APD is employed (5). These guidelines must however be viewed as recommendations for *minimal* overall clearance. In patients with residual renal function this renal clearance can be subtracted from the peritoneal clearance with confidence that the value of equivalent renal clearances in greater. Equally, in patients achieving these clearances but experiencing uraemic symptoms, or failing to achieve adequate acid base balance (see section 6) the dialysis dose should be increased. Drop out due to uraemia or death associated with hyperkalaemia and acidosis was significantly more common in the control patients in the ADEMEX study (1).

Studies in children include small patient numbers and results are variable, some suggesting a ceiling above which no further improvement in growth and nutritional state occurs because of peritoneal protein losses. It is recommended that the standards for adults should be seen as a minimum for children (6).

1. Paniagua R, Amato D, Vonesh E, et al. Effects of Increased Peritoneal Clearances on Mortality Rates in Peritoneal Dialysis: ADEMEX, a Prospective, Randomized, Controlled Trial. *J Am Soc Nephrol* 2002;13(5):1307-20.
2. Lo WK, Ho YW, Li CS, et al. Effect of Kt/V on survival and clinical outcome in CAPD patients in a randomized prospective study. *Kidney Int* 2003;64(2):649-56.
3. Davies SJ, Phillips L, Russell L, Naish PF, Russell GI. An analysis of the effects of increasing delivered dialysis treatment to malnourished peritoneal dialysis patients. *Kidney Int* 2000;57(4):1743-54.
4. Jansen MA, Termorshuizen F, Korevaar JC, Dekker FW, Boeschoten E, Krediet RT. Predictors of survival in anuric peritoneal dialysis patients. *Kidney Int.* 2005;68(3):1199-205.
5. Brown EA, Davies SJ, Heimburger O, et al. Adequacy targets can be met in anuric patients by automated peritoneal dialysis: baseline data from EAPOS. *Perit Dial Int* 2001;21(Suppl 3):S133-7.
6. Lesley Rees I, Vanessa Shaw. Nutrition in children with CRF and on dialysis. Pediatr Nephrol, in press

4. Ultrafiltration and fluid management

4.1 Peritoneal membrane function should be monitored regularly (6 weeks after commencing treatment and at least annually or when clinically indicated) using a peritoneal equilibration test (PET) or equivalent. Daily urine and peritoneal ultrafiltration volumes, with appropriate correction for overfill, should be monitored six-monthly. (Good practice)

Audit Measure 14: Frequency of measurement of membrane function, residual urine and peritoneal ultrafiltration volume

Assessment of membrane function, specifically solute transport rate and ultrafiltration capacity) is fundamental to PD prescription. (See appendix for methodological description of membrane function tests). This is for the following reasons:

1. There is considerable between-patient variability in both solute transport and ultrafiltration capacity that translates into real differences in achieved solute clearance and ultrafiltration unless they are accounted for in prescription practice (1-5)

2. Membrane function is an independent predictor of patient survival; specifically high solute transport and low ultrafiltration capacity are associated with worse outcomes (6-10)

3. Membrane function changes with time on therapy. There are early changes – usually during the first few weeks of treatment that can be avoided by performing tests 6 weeks after commencing PD. Later changes vary between patients but tend to be increasing solute transport and reduced ultrafiltration capacity; the rate of membrane change is accelerated in patients with earlier loss of residual renal function and greater requirement for hypertonic glucose solutions. (5, 11, 12)

Residual renal function, as discussed above, is one of the most important factors, along with age, comorbidity, nutritional status, plasma albumin and membrane function that predict survival in PD patients. Its rate of loss is variable and clinically significant changes can occur within 6 months. Total fluid removal is associated with patient survival, especially once anuric (9, 13, 14), ADEMEX study, data awaiting publication.

1. Twardowski ZJ, Nolph KD, Khanna R, et al. Peritoneal Equilibration Test. *Perit Dial Bull* 1987;7:138-47.
2. Smit W, van Dijk P, Langedijk MJ, et al. Peritoneal function and assessment of reference values using a 3.86% glucose solution. *Perit Dial Int* 2003;23(5):440-9.
3. Smit W, Schouten N, van den Berg N, Langedijk MJ, Struijk DG, Krediet RT. Analysis of the prevalence and causes of ultrafiltration failure during long-term peritoneal dialysis: a cross-sectional study. *Perit Dial Int* 2004;24(6):562-70.
4. Selgas R, Bajo MA, Cirugeda A, et al. Ultrafiltration and small solute transport at initiation of PD: questioning the paradigm of peritoneal function. *Perit Dial Int.* 2005;25(1):68-76.
5. Davies SJ. Longitudinal relationship between solute transport and ultrafiltration capacity in peritoneal dialysis patients. *Kidney Int* 2004;66:2437-45.
6. Davies SJ, Phillips L, Naish PF, Russell G. Quantifying comorbidity in Peritoneal Dialysis patients and its relationship to other predictors of survival. *Nephrol Dial Transplant* 2002;17(6):1085-92.
7. Churchill DN, Thorpe KE, Nolph KD, Keshaviah PR, Oreopoulos DG, Page D. Increased

peritoneal membrane transport is associated with decreased patient and technique survival for continuous peritoneal dialysis patients. *J Am Soc Nephrol* 1998;9:1285-92.
8. Rumpsfeld M, McDonald SP, Johnson DW. Higher peritoneal transport status is associated with higher mortality and technique failure in the Australian and New Zealand peritoneal dialysis patient populations. *J Am Soc Nephrol.* 2006;17(1):271-8. Epub 2005 Nov 23.
9. Brown EA, Davies SJ, Rutherford P, et al. Survival of Functionally Anuric Patients on Automated Peritoneal Dialysis: The European APD Outcome Study. *J Am Soc Nephrol* 2003;14(11):2948-57.
10. Brimble KS, Walker M, Margetts PJ, Kundhal KK, Rabbat CG. Meta-analysis: peritoneal membrane transport, mortality, and technique failure in peritoneal dialysis. *J Am Soc Nephrol.* 2006;17(9):2591-8. Epub 006 Aug 2.
11. Heimburger O, Wang T, Lindholm B. Alterations in water and solute transport with time on peritoneal dialysis. *Perit Dial Int* 1999;19 Suppl 2:S83-90.
12. del Peso G, Fernandez-Reyes MJ, Hevia C, et al. Factors influencing peritoneal transport parameters during the first year on peritoneal dialysis: peritonitis is the main factor. *Nephrol Dial Transplant.* 2005;20(6):1201-6.
13. Ates K, Nergizoglu G, Keven K, et al. Effect of fluid and sodium removal on mortality in peritoneal dialysis patients. *Kidney Int* 2001;60(2):767-76.
14. Jansen MA, Termorshuizen F, Korevaar JC, Dekker FW, Boeschoten E, Krediet RT. Predictors of survival in anuric peritoneal dialysis patients. *Kidney Int.* 2005;68(3):1199-205.

4.2 Dialysis regimes resulting in fluid reabsorption should be avoided. (Good practice). Patients with high or high average solute transport, at greatest risk of this problem, should be considered for APD and icodextrin (Evidence)

Audit Measure 15: Identify patients with fluid reabsorption in long dwell

Increased solute transport has been repeatedly shown to be associated with worse survival, especially in CAPD patients (1-4). The explanation for this association is most likely to be because of its effect on ultrafiltration when this is achieved with an osmotic gradient (using glucose or amino-acid dialysis fluids). The reason is twofold: first, due to more rapid absorption of glucose, the osmotic gradient is lost earlier in the cycle resulting in reduced ultrafiltration capacity. Second, once the osmotic gradient is dissipated the rate of fluid reabsorption in high transport patients is more rapid. This will result in significant fluid absorption, contributing to a positive fluid balance, during the long exchange.

These problems associated with high transport can be avoided by using APD to shorten dwell length and by using icodextrin for the long exchange to prevent fluid reabsorption. Several randomised controlled trials have shown that icodextrin can achieve sustained ultrafiltration in the long dwell (5-9) and that this translates into a reduction in extracellular fluid volume (10, 11). Observational studies indicate that high solute transport is not associated with increased mortality or technique failure in APD patients, especially when there is also a high use of icodextrin (3, 12, 13).

1. Davies SJ, Phillips L, Naish PF, Russell G. Quantifying comorbidity in Peritoneal Dialysis patients and its relationship to other predictors of survival. *Nephrol Dial Transplant* 2002;17(6):1085-92.
2. Churchill DN, Thorpe KE, Nolph KD, Keshaviah PR, Oreopoulos DG, Page D. Increased peritoneal membrane transport is associated with decreased patient and technique survival for continuous peritoneal dialysis patients. *J Am Soc Nephrol* 1998;9:1285-92.
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4. Brimble KS, Walker M, Margetts PJ, Kundhal KK, Rabbat CG. Meta-analysis: peritoneal membrane transport, mortality, and technique failure in peritoneal dialysis. *J Am Soc Nephrol.* 2006;17(9):2591-8. Epub 006 Aug 2.

5. Posthuma N, ter Wee PM, Verbrugh HA, et al. Icodextrin instead of glucose during the daytime dwell in CCPD increases ultrafiltration and 24-h dialysate creatinine clearance. *Nephrol Dial Transplant Nephrology, Dialysis, Transplantation* 1997;12(3):550-3.
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8. Ota K, Akiba T, Nakao T, et al. Peritoneal ultrafiltration and serum icodextrin concentration during dialysis with 7.5% icodextrin solution in Japanese patients. *Perit Dial Int* 2003;23(4):356-61.
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12. Brown EA, Davies SJ, Rutherford P, et al. Survival of Functionally Anuric Patients on Automated Peritoneal Dialysis: The European APD Outcome Study. *J Am Soc Nephrol* 2003;14(11):2948-57.
13. Davies SJ. Mitigating peritoneal membrane characteristics in modern PD therapy. *Kidney Int* 2006;in press.

4.3 Dialysis regimes resulting in routine utilisation of hypertonic (3.86%) glucose exchanges should be avoided (Good practice). Where appropriate this should be achieved by avoiding excess dietary salt intake, using diuretics or icodextrin (Evidence).

There is growing evidence that regular use of hypertonic glucose dialysis fluid (3.86%), and where possible glucose 2.27%, is to be avoided. It is associated with acceleration in the detrimental changes in membrane function that occur with time on treatment (1, 2), as well as several undesirable systemic effects including weight gain (3, 4), poor diabetic control (5), delayed gastric emptying (6), hyperinsulinaemia and adverse haemodynamic effects (7). In addition to patient education to avoid excessive salt and fluid intake, where possible the use of hypertonic glucose should be minimised by enhancing residual diureses with the use of diuretics (e.g. frusemide 250mg daily) (8). Substituting icodextrin for glucose solutions during the long exchange will result in equivalent ultrafiltration whilst avoiding the systemic effects of the glucose load (3, 5, 7, 9). Observational evidence would suggest that icodextrin is associated with less functional deterioration in the membrane in APD patients (2).

1. Davies SJ. Longitudinal relationship between solute transport and ultrafiltration capacity in peritoneal dialysis patients. *Kidney Int* 2004;66:2437-45.
2. Davies SJ, Brown EA, Frandsen NE, et al. Longitudinal membrane function in functionally anuric patients treated with APD: Data from EAPOS on the effects of glucose and icodextrin prescription. *Kidney Int* 2005;67(4):1609-15.
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4. Fernstrom A, Hylander B, Moritz A, Jacobsson H, Rossner S. Increase of intra-abdominal fat in patients treated with continuous ambulatory peritoneal dialysis. *Perit Dial Int* 1998;18(2):166-71.
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8. Medcalf JF, Harris KP, Walls J. Role of diuretics in the preservation of residual renal function in patients on continuous ambulatory peritoneal dialysis. *Kidney Int* 2001;59(3):1128-33.
9. Davies SJ, Woodrow G, Donovan K, et al. Icodextrin improves the fluid status of peritoneal dialysis patients: results of a double-blind randomized controlled trial. *J Am Soc Nephrol* 2003;14(9):2338-44.

4.4 Treatment strategies that favour preservation of renal function should be adopted where possible (Good practice). These include avoidance of episodes of dehydration, use of diuretics, ACEi and ARBs (Evidence)

This is the single most important parameter in PD patients, and also the one most likely to change with time. Clinically significant changes can occur within three months. Because secretion of creatinine by the kidney at low levels of function overestimates residual creatinine clearance, it is recommended to express this as the *mean* of the urea and creatinine clearances. Observational and randomised studies have shown that episodes of volume depletion, whether unintentional or in response to active fluid removal with the intent of changing blood pressure or fluid status, are associated with increased risk of loss in residual renal function (1-4). Care should be taken not to volume deplete a PD patient too rapidly or excessively. The use of diuretics to maintain urine volume is not associated with a risk to renal clearances (5). ACE inhibitors, (Ramipril 5mg) (6) and ARBs (valsartan) (7) have been shown in randomised studies to maintain residual diuresis.

1. Jansen MA, Hart AA, Korevaar JC, Dekker FW, Boeschoten EW, Krediet RT. Predictors of the rate of decline of residual renal function in incident dialysis patients. *Kidney Int* 2002;62(3):1046-53.
2. Gunal AI, Duman S, Ozkahya M, et al. Strict volume control normalizes hypertension in peritoneal dialysis patients. *Am J Kidney Dis* 2001;37(3):588-93.
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5. Medcalf JF, Harris KP, Walls J. Role of diuretics in the preservation of residual renal function in patients on continuous ambulatory peritoneal dialysis. *Kidney Int* 2001;59(3):1128-33.
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4.5 Anuric patients who consistently achieve a daily ultrafiltration of less than 750 should be closely monitored and the benefits of modality switch considered (Good practice)

Audit Measure (adult only): Identify patients with a total fluid removal <750 ml per day. Observational studies have consistently shown that reduced peritoneal ultrafiltration is associated with worse survival rates; whilst this is seen in studies with or without residual urine (1), this effect is most marked in anuric patients (2, 3). In the only prospective study to have preset an ultrafiltration target (750 ml/day), patients who remained below this had higher mortality after correcting for age, time on dialysis, comorbidity and nutritional status. It is likely this association is multifactorial, but failure to prescribe sufficient glucose or icodextrin and a lower ultrafiltration capacity of the membrane were factors in this study and should be considered (2, 4). The

European guidelines have suggested a 1 litre minimal daily ultrafiltration target;(5) there is insufficient evidence to say that such a target must be met at this stage. Blood pressure, salt (and fluid) intake, nutritional and fluid status should be taken into account. Nevertheless patients with less than 750 ml ultrafiltration once anuric should be very closely monitored and the potential benefits of modality switch considered.

1. Ates K, Nergizoglu G, Keven K, et al. Effect of fluid and sodium removal on mortality in peritoneal dialysis patients. *Kidney Int* 2001;60(2):767-76.
2. Brown EA, Davies SJ, Rutherford P, et al. Survival of Functionally Anuric Patients on Automated Peritoneal Dialysis: The European APD Outcome Study. *J Am Soc Nephrol* 2003;14(11):2948-57.
3. Jansen MA, Termorshuizen F, Korevaar JC, Dekker FW, Boeschoten E, Krediet RT. Predictors of survival in anuric peritoneal dialysis patients. *Kidney Int.* 2005;68(3):1199-205.
4. Davies SJ, Brown E, Riegel W, et al. What is the link between poor ultrafiltration and increased mortality in anuric APD patients? Analysis of data from EAPOS. *Perit Dial Int* 2006;26(4):458-65.
5. Dombros N, Dratwa M, Feriani M, et al. European best practice guidelines for peritoneal dialysis. 7 Adequacy of peritoneal dialysis. *Nephrol Dial Transplant.* 2005;20(Suppl 9):ix24-ix7.

5. Infectious complications

5.1 Prevention Strategies

5.1.1 PD units should undertake regular audit of their peritonitis and exit-site infection rates, including causative organism, treatment and outcomes. They should enter into active dialogue with their microbiology department and infection control team to develop optimal local treatment and prevention protocols (Good practice)

5.1.2 Flush-before-fill dialysis delivery systems should be used (Evidence)

5.1.3 Patients should undergo regular (annually or more frequently if indicated) revision of their technique and receive intensified training if this is below standard (Evidence)

5.1.4 Initial catheter insertion should be accompanied by antibiotic prophylaxis (Evidence)

5.1.5 Invasive procedures should be accompanied by antibiotic prophylaxis and emptying the abdomen of dialysis fluid for a period commensurate with the procedure (Good practice)

5.1.6 Topical antibiotic administration should be used to reduce the frequency of *Staph. aureus* and Gram negative exit-site infection and peritonitis (Evidence)

Audit Measure 16: Routine annual audit of infection prevention strategies

The rationale underpinning the guidelines in this section is laid out in a series of documents published by the International Society of Peritoneal Dialysis, available on their web-site: www.ispd.org

Prevention strategies: Both the ISPD 2005 guidelines (1) and the NSF Part 1 place increasing emphasis on prevention strategies. Regular audit is essential to this progress and the following standards should be considered as minimal:

1. Peritonitis rates of less than 1 episode per 18 months in adults and 12 months in children (see NSF part 1)
2. A primary cure rate of 80%
3. A culture negative rate of < 20%

Approaches that have been shown to reduce infection rates in randomised studies include increased intensity of training (2), use of flush before fill systems,(3) antibiotic prophylaxis to cover catheter insertion and prevention of exit-site infections (1). Several studies have addressed the latter issue; following demonstration that the risk of *Staph aureus* exit site infection (the organism responsible in 90% of cases) is associated with pre-existing skin carriage, several randomised studies demonstrated that clinical exit-site infection and associated peritonitis could be reduced by either nasal or exit-site application of mupirocin. This has led to the practice of applying mupirocin to all patients;(4, 5) this approach should be discussed with the local microbiology and infection control team. A more recent study, comparing mupirocin with gentamicin cream, found that the latter prevented both *Staph aureus* and *Pseudomonas* exit-site infections and peritonitis episodes (6). This approach should be strongly considered in patients with a known history of *Pseudomonas* infections; again the policy should be discussed and agreed with the local microbiology team.

1. Piraino B, Bailie GR, Bernardini J, et al. Peritoneal dialysis-related infections recommendations: 2005 update. *Perit Dial Int.* 2005;25(2):107-31.
2. Hall G, Bogan A, Dreis S, et al. New directions in peritoneal dialysis patient training. *Nephrol Nurs J.* 2004;31(2):149-54, 59-63.
3. MacLeod A, Grant A, Donaldson C, et al. Effectiveness and efficiency of methods of dialysis therapy for end-stage renal disease: systematic reviews. *Health Technol Assess* 1998;2(5):1-166.
4. Bernardini J, Piraino B, Holley J, Johnston JR, Lutes R. A randomized trial of Staphylococcus aureus prophylaxis in peritoneal dialysis patients: mupirocin calcium ointment 2% applied to the exit site versus cyclic oral rifampin. *Am J Kidney Dis* 1996;27(5):695-700.
5. Piraino B. Staphylococcus aureus infections in dialysis patients: focus on prevention. *Asaio J* 2000;46(6):S13-7.
6. Bernardini J, Bender F, Florio T, et al. Randomized, double-blind trial of antibiotic exit site cream for prevention of exit site infection in peritoneal dialysis patients. *J Am Soc Nephrol.* 2005;16(2):539-45. Epub 2004 Dec 29.

5.2 Treatment

5.2.1 Exit site infection is suggested by pain, swelling, crusting, erythema and serous discharge; purulent discharge always indicates infection. Swabs should be taken for culture and initial empiric therapy should be with oral antibiotics that will cover *S. aureus and P. aeruginosa* (Good practice)

5.2.2 Methicillin resistant organisms (MRSA) will require systemic treatment (e.g vancomycin) and will need to comply with local infection control policies. (Good practice)

5.2.3 Initial treatment regimes for peritonitis should include cover for bacterial Gram positive and Gram negative organisms until result of culture and antibiotic sensitivities are obtained. (Good practice)

Audit Measure 17: Routine annual audit of infection outcomes (exit site and peritonitis rates

The ISPD has developed a simple scoring system for exit site signs and symptoms which is easy to use and gives guidance on when to treat immediately rather than waiting for a swab result. Purulent discharge is an absolute indicator for antibiotic treatment (1). The ISPD has become less dogmatic about the initial choice of antibiotic treatment for peritonitis, provided that gram positive and negative infections are covered. It is recognised that patterns of resistance vary considerably and thus a local policy must be developed.

1. Piraino B, Bailie GR, Bernardini J, et al. Peritoneal dialysis-related infections recommendations: 2005 update. *Perit Dial Int.* 2005;25(2):107-31.

6. Metabolic Factors

6.1 Standard strategies to optimise diabetic control should be used; these should be complemented by dialysis prescription regimens that minimise glucose, including glucose free solutions (icodextrin and amino-acids), where possible. (Good practice)

Glycaemic control can be made worse by glucose absorption across the peritoneal membrane. Dialysis regimes that incorporate less glucose and more glucose free (amino acid, icodextrin) solutions have been shown to improve glycaemic control (1), Paniagua (in press).

1. Marshall J, Jennings P, Scott A, Fluck RJ, McIntyre CW. Glycemic control in diabetic CAPD patients assessed by continuous glucose monitoring system (CGMS). *Kidney Int* 2003;64(4):1480-6.

6.2 Plasma bicarbonate should be maintained within the normal range; this can be achieved in the vast majority of patients by adjusting the dialysis dose and/or dialysate buffer concentration. Occasionally bicarbonate buffered solutions will be required (Good practice).

Audit measure 18: Cumulative frequency curves of plasma bicarbonate

Two randomised controlled trials have suggested that clinical outcomes, including gaining lean body mass and reduced hospital admissions are achieved if the plasma bicarbonate is kept within the upper half of the normal range.(1, 2) Generally this can be achieved by using dialysis fluids with a 40 mmol buffer capacity (lactate or bicarbonate results in similar plasma bicarbonate levels(3)) and ensuring that the dialysis dose is adequate (see section 3 (b), above) (4). However, for solutions with a lower buffering capacity, when patients are switched from an all lactate (35 mmol/l) to a 25 mmol bicarbonate: 10 mmol lactate mix, there is a significant improvement in plasma bicarbonate (24.4 to 26.1 mmol/l), such that a higher proportion of patients will fall within the normal range (5). Whilst bicarbonate solutions may have a role in biocompatibility (see section 1(e), above), they are generally not required to achieve satisfactory acid-base balance. The main reason for using a 35 mmol buffer capacity solution (25:10 bicarbonate:lactate mix) is to avoid excessive alkalinisation (6). The long-term effects of persistent metabolic alkalosis on the developing skeleton are not known, and an alkaline pH can predispose to soft tissue calcification.

Control of acidosis is especially important in malnourished patients who may benefit from the glucose available in dialysis solutions as a calories source. Amino acid solutions were developed in an attempt to address protein calorie malnutrition and several randomised studies have been conducted. In using amino acid solutions it is essential to ensure that acidosis does not develop and to use the solution at the same time as there is a significant intake of carbohydrate (7). Despite demonstration that amino acids delivered in dialysis fluids are incorporated into tissue protein, the randomised trials have failed to show benefit in terms of hard clinical endpoints (8, 9).

1. Stein A, Moorhouse J, Iles-Smith H, et al. Role of an improvement in acid-base status and nutrition in CAPD patients. *Kidney Int* 1997;52(4):1089-95.
2. Szeto CC, Wong TY, Chow KM, Leung CB, Li PK. Oral sodium bicarbonate for the treatment of metabolic acidosis in peritoneal dialysis patients: a randomized placebo-control trial. *J Am Soc Nephrol* 2003;14(8):2119-26.
3. Coles GA, Gokal R, Ogg C, et al. A randomized controlled trial of a bicarbonate- and a bicarbonate/lactate-containing dialysis solution in CAPD. *Perit Dial Int* 1997;17(1):48-51.
4. Mujais S. Acid base profile in patients on PD. *Kidney Int* 2003;Suppl. 83(Deb):in press.
5. Otte K, Gonzalez MT, Bajo MA, et al. Clinical experience with a new bicarbonate (25 mmol/L)/lactate (10 mmol/L) peritoneal dialysis solution. *Perit Dial Int* 2003;23(2):138-45.
6. Dratwa M, Wilkie M, Ryckelynck JP, et al. Clinical experience with two physiologic bicarbonate/lactate peritoneal dialysis solutions in automated peritoneal dialysis. *Kidney Int* 2003;88:S105-13.
7. Kopple JD, Bernard D, Messana J, et al. Treatment of malnourished CAPD patients with an amino acid based dialysate. *Kidney Int* 1995;47(4):1148-57.
8. Li FK, Chan LY, Woo JC, et al. A 3-year, prospective, randomized, controlled study on amino acid dialysate in patients on CAPD. *Am J Kidney Dis* 2003;42(1):173-83.
9. Jones M, Hagen T, Boyle CA, et al. Treatment of malnutrition with 1.1% amino acid peritoneal dialysis solution: results of a multicenter outpatient study. *Am J Kidney Dis* 1998;32(5):761-9.

6.3 Central obesity can worsen or develop in some PD patients. The risk of this problem, and associated metabolic complications, notably increased atherogenicity of lipid profiles and insulin resistance, can be reduced by avoiding excessive glucose prescription and using icodextrin. (Good practice)

Weight gain, or regain, is common after starting peritoneal dialysis and this is associated with a worsening in the lipid profile (1). Randomised studies comparing glucose 2.27% with icodextrin in the long exchange have shown that the latter prevents weight gain, which in body composition studies is at least in part fat weight (2, 3). Recommendations on how to treat dyslipidaemia are published by the ISPD and include the use of statins (4). There is no currently available trial data on the benefit of statins in PD patients with a hard clinical endpoint; the 4D study did not include PD patients and there are good reasons for believing that the PD patient population may be different.

1. Little J, Phillips L, Russell L, Griffiths A, Russell GI, Davies SJ. Longitudinal lipid profiles on CAPD: their relationship to weight gain, comorbidity, and dialysis factors. *J Am Soc Nephrol* 1998;9(10):1931-9.
2. Wolfson M, Piraino B, Hamburger RJ, Morton AR. A randomized controlled trial to evaluate the efficacy and safety of icodextrin in peritoneal dialysis. *Am J Kidney Dis* 2002;40(5):1055-65.
3. Davies SJ, Woodrow G, Donovan K, et al. Icodextrin improves the fluid status of peritoneal dialysis patients: results of a double-blind randomized controlled trial. *J Am Soc Nephrol* 2003;14(9):2338-44.
4. Fried L, Hutchison A, Stegmayr B, Prichard S, Bargman JM. Recommendations for the treatment of lipid disorders in patients on peritoneal dialysis. ISPD guidelines/recommendations. International Society for Peritoneal Dialysis. *Perit Dial Int* 1999;19(1):7-16.

6.4 Awareness of the effects of Icodextrin on assays for estimation of amylase and glucose (using glucose dehydrogenase) should be disseminated to patients, relatives, laboratory and clinical staff.

Audit Measure 19: Processes in place to increase awareness of interference of assays by icodextrin metabolites

Use of icodextrin is associated with circulating levels of metabolites that can interfere with laboratory assays for amylase (or actually suppress amylase activity) (1-4) and for glucose when finger-prick tests that utilise glucose dehydrogenase as their substrate are employed (manufactured by Boehringer Mannheim) (5-8). In the case of amylase, the measured level will be reduced by 90%, leading to the potential failure in the diagnosis of pancreatitis. No adverse events have been reported, but clinicians should be aware of this possibility. If clinical concern remains then plasma lipase can be used. In the case of glucose measurements, the methods using glucose dehydrogenase will *over*-estimate blood glucose levels, leading to a failure to diagnose hypoglycaemia. This has been reported on several occasions in the literature and has contributed to at least one death. Typically these errors occur in places and circumstances in which staff not familiar with peritoneal dialysis work, for example emergency rooms and non-renal wards. A number of solutions to this problem are under active review (e.g. use of alarm bracelets) but it is also the responsibility of health-care professionals to ensure that clinical environments in which their patients using icodextrin may find themselves are notified of this issue on a routine basis.

1. Schoenicke G, Grabensee B, Plum J. Dialysis with icodextrin interferes with measurement of serum alpha-amylase activity. *Nephrol Dial Transplant* 2002;17(11):1988-92.
2. Wang R, Leesch V, Turner P, Moberly JB, Martis L. Kinetic analysis of icodextrin interference with serum amylase assays. *Adv Perit Dial* 2002;18:96-9.
3. Anderstam B, Garcia-Lopez E, Heimburger O, Lindholm B. Determination of alpha-amylase activity in serum and dialysate from patients using icodextrin-based peritoneal dialysis fluid. *Perit Dial Int* 2003;23(2):146-50.
4. Garcia-Lopez E, Anderstam B, Heimburger O, Amici G, Werynski A, Lindholm B. Determination of high and low molecular weight molecules of icodextrin in plasma and dialysate, using gel filtration chromatography, in peritoneal dialysis patients. *Perit Dial Int* 2005;25(2):181-91.
5. Wens R, Taminne M, Devriendt J, et al. A previously undescribed side effect of icodextrin: overestimation of glycemia by glucose analyzer. *Perit Dial Int* 1998;18(6):603-9.
6. Oyibo SO, Pritchard GM, McLay L, et al. Blood glucose overestimation in diabetic patients on continuous ambulatory peritoneal dialysis for end-stage renal disease. *Diabet Med* 2002;19(8):693-6.
7. Mehmet S, Quan G, Thomas S, Goldsmith D. Important causes of hypoglycaemia in patients with diabetes on peritoneal dialysis. *Diabet Med* 2001;18(8):679-82.
8. Janssen W, Harff G, Caers M, Schellekens A. Positive interference of icodextrin metabolites in some enzymatic glucose methods. *Clin Chem* 1998;44(11):2379-80.

7. Laboratory and clinical indices

7.1 Monitoring of biochemical and haematological parameters should be performed monthly or at each clinic visit (Good practice).

Standardised analytical methods of measuring laboratory indices are required if comparative audit against target standards is to be meaningful. Difficulties still arise since laboratories across the UK use different methods to measure serum albumin and different correction factors for adjusting serum calcium levels (1).

1. The Renal Association UK Renal Registry, The Seventh Annual Report, December 2004. (www.renalreg.com)

7.2 Pre-dialysis serum bicarbonate concentrations measured with minimum delay after venepuncture should be between 20 and 26mmol/l. (Good practice)

The main causal factors of metabolic acidosis are inadequate dialysis delivery, *ongoing renal losses*, excessive animal protein (sulphur containing amino acid) intake and high interdialysis weight gains. Whole-body base balance studies in 18 anuric HD patients have highlighted the importance of interdialysis dilution in the aetiology of predialysis acidosis (1). In ill patients metabolic acidosis may also be due to increased protein catabolism, hypotension or hypoxia induced lactate production or bicarbonate losses associated with co-morbid illness. Metabolic acidosis has a range of adverse consequences: an increase in protein catabolism and anti-anabolic effects, negative inotropic effect, loss of bone mineral, insulin resistance, growth retardation in children, reduced thyroxine levels, altered triglyceride metabolism, hyperkalaemia, lower serum leptin levels and greater accumulation of beta-2-microglobulin.

Pre-dialysis venous bicarbonate levels between 17.5 and 20 mmol/l were associated with the lowest risk of death in a large cohort study of 13535 hemodialysis patients whilst the relative risk of death was increased threefold if the pre-dialysis venous bicarbonate was < 15 mmol/l (2). In a DOPPS study of more than 7000 unselected HD patients the corrected mid-week serum bicarbonate concentration averaged 21.9 mmol/l and correlated inversely with the nPCR and serum albumin (3). The adjusted risk of death, hospitalization or malnutrition was higher in patients with serum bicarbonate levels less than 16 or above 24 when compared with patients in the reference group with moderate pre-dialysis acidosis (3). Short-term benefits of correcting pre-dialysis acidosis from below 19mmol/l to 24mmol/l, by either increasing the dialysate bicarbonate concentration (4-7) or the addition of oral bicarbonate supplements (8), have been shown in several small crossover studies. Correction of acidosis reduced whole body protein degradation in a study of 6 patients (4), increased the sensitivity of the parathyroid glands to serum calcium in studies of 21 and 8 patients (5,6), improved triceps skin thickness as an index of nutritional status in 46 patients (7) and increased serum albumin after 3 months in 12 patients without any change in body weight, Kt/V, and nPCR (8). Other studies have shown no increase in serum albumin after correction of acidosis.

1. *Mioni R, Gropuzzo M, Messa M et al. Acid production and base balance in patients in chronic hemodialysis. Cli Sci 201;101:329-37*
2. *Lowrie EG, Lew NL. Death risk in hemodialysis patients: The predictive value of commonly measured variables and an evaluation of death rate differences between facilities. Am J Kidney Dis 1990; 15: 458-482*
3. *Bommer J, Locatelli F, Satayathum S et al. Association of predialysis serum bicarbonate levels with risk of mortality and hospitalisation in the Dialysis Outcomes and Practice Patterns Study (DOPPS). Am J Kidney Dis 2004;44:661-71*
4. *Graham KA, Reaich D, Channon SM et al. Correction of acidosis in hemodialysis decreases whole-body protein degradation. J Am Soc Nephrol 1997;8: 632-7*
5. *Lefebvre A, de Verneioul MC, Gueris J et al. Optimal correction of acidosis changes progression of dialysis osteodystrophy. Kidney Int 1989;36:1112-8*
6. *Graham KA, Hoenich NA, Tarbit M et al. Correction of acidosis in hemodialysis patients increases the sensitivity of the parathyroid glands to calcium. J Am Soc Nephrol;1997;8:627-31*

7. *Williams AJ, Dittmer ID, McArley A, Clarke J. High bicarbonate dialysate in hemodialysis patients: effects on acidosis and nutritional status. Nephrol Dial Transplant 1997;12:2633-7*
8. *Movilli E, Zani R, Carli O et al. Correction of metabolic acidosis increases serum albumin concentrations and decreases kinetically evaluated protein intake in hemodialysis patients: a prospective study. Nephrol Dial Transplant 1998;13:1719-22*

7.3 Serum potassium should be between 3.5 and 6.5 mmol/l (Good practice)

The risk of developing hyperkalaemia is inversely related to renal function. 3-5% of deaths in dialysis patients have been attributed to hyperkalaemia (1). Non-compliance with the PD prescription and/or diet is the main cause of hyperkalaemia in dialysis patients but drug therapy, such as ACE inhibitors, angiotensin receptor blockers, non-steroidal anti-inflammatory drugs, beta-blockers and potassium supplements, may be implicated.

A Cochrane meta-analysis of non-dialytic emergency interventions for hyperkalaemia concluded that intravenous glucose with insulin and nebulised or inhaled salbutamol were effective in reducing serum potassium levels but the studies were limited by the absence of data on cardiac arrhythmia or mortality rates (2). Whilst the combination of salbutamol and intravenous glucose with insulin was probably more effective than either therapy alone the evidence for efficacy of intravenous bicarbonate or potassium exchange resins in this Cochrane review of randomized or quasi-randomised trials was equivocal and neither should be used as monotherapy for severe hyperkalaemia.

1. *Morduchowicz G, Winkler J, Drazne E et al. Causes of death in patients with end-stage renal disease treated by dialysis in a centre in Israel. Isr J Med Sci 1992;28:776-9*
2. *Mahoney BA, Smith WAD, Lo DS, Tsoi K, Tonelli M, Clase CM. Emergency interventions for hyperkalaemia (Cochrane Review). In: The Renal health Library, 2005. Oxford: Update Software Ltd (www.update-software.com)*

7.4 Serum phosphate should be within, and preferably nearer to the 50th centile for age. (Good practice) (The normal range for phosphate declines from birth to adult levels by the age of 3 years)

7.5 Serum calcium, adjusted for serum albumin, should be within the age appropriate normal range. (Good practice)

7.6 Serum albumin corrected calcium x phosphate product should be less than <4.5 mmol^2/L^2 (K/DOQI guidelines) or <5 mmol^2/L^2 (European PD working group advice).

7.7 The optimal range for PTH is controversial. There is emerging evidence that levels should be maintained at less than twice the upper limit of normal for the intact PTH assay used. (Good practice)

Audit measure 21 - Cumulative frequency curves of serum calcium, phosphate calcium x phosphate product and PTH concentrations

7.8 Haemoglobin concentration should be greater than the lower limit of the age appropriate normal range. (Evidence) The target haemoglobin concentration should be 1g/dl higher, to allow for the normal distribution around the mean haemoglobin value of the patient population and intra individual variation of laboratory measurements and hydration status.

Audit measure 22 - Cumulative frequency curves of haemoglobin concentration

7.9 Ferritin levels should be between 100 and 800 mcg/l. *However, given the increased risk of thrombotic events with higher ferritin levels that have been shown in recent trials, the revised K/DOQI guidelines for the management of anaemia recommend that the serum ferritin levels should be maintained between 100 – 500mcg/L in adults. In the absence of paediatric studies, in patients at risk of thrombosis (e.g. those with low serum albumin, arteriovenous fistulae or synthetic grafts), the serum ferritin levels above 500mcg/L should be avoided.*

Haemoglobin levels should be maintained in the age appropriate normal range, aiming for Hb between 11-12gm/dL in children above 2 years of age.

7.8 Growth, wellbeing and school attendance are very important indicators of dialysis adequacy and should be assessed at least monthly in those under two years of age (length, weight and head circumference) and at least 3 monthly in older children (height, weight and pubertal stage, school attendance). Assessment of dry weight may be difficult in the growing child and also needs checking with at least the same frequency, with close collaboration with a paediatric renal dietician.

Audit measure 22 Height, weight, head circumference and pubertal progression

7.9 An assessment of school progress, both in hospital and locally, can be used as an assessment of well-being, and should be made annually

Audit measure 23 – School attendance

7.10 Blood pressure should be maintained within the age appropriate normal range

Audit measure 24 - Cumulative frequency curves of BP pre-dialysis

8. Access to and withdrawal from dialysis

RATIONALE

8.1 All children with CKD should be considered for renal replacement therapy by stage 4 (Good practice)

CKD should be suspected in children with: bilateral renal anomalies on antenatal scans (many children with CKD are now diagnosed antenatally); a creatinine above the normal age appropriate range; bilateral renal defects on scans e.g. for UTI; a family history of CKD; persistent proteinuria; or after an episode of acute renal failure. All such children should be referred to a paediatric nephrologist. Early referral provides the opportunity for delaying the progression of CKD by treating hypertension and proteinuria, for optimising growth and preventing renal bone

disease. Importantly, it also allows for timely forward planning for renal replacement therapy.

In adults, avoiding late referral provides the opportunity for intervention to prevent or reduce the complications of renal failure and time to plan for renal replacement therapy. Patients who have been under nephrology care for more than 1 month are more likely to start HD using an AVF (1). A retrospective analysis of 109,321 incident HD patients in the USA found that the relative risk of death of patients with no pre-dialysis nephrology care was 1.51 and the relative risk of death of patients with one or two months pre-dialysis nephrology care was 1.23 when compared with patients with at least 3 months nephrology pre-dialysis care (2).

1. Rayner HC, Besarab A, Brown WW et al. Vascular access results from the Dialysis Outomes and Practice Patterns Study (DOPPS): performance against Kidney Disease Outcomes Quality Initiative (K/DOQI) Clinical Practice Guidelines. Am J Kidney Dis 2004; 44 (Suppl 3): 22-26
2. Khan SS, Xue JL, Kazmi WH et al. Does pre-dialysis nephrology care influence patient survival after initiation of dialysis? Kidney Int 2005; 67:1038-1046

8.2 If there is no medical contraindication the choice of initial dialysis modality should be based on patient choice. (Good practice)

The provision of patient choice and equity of access to dialysis and transplantation have been reinforced by the National Service Framework Part 1 Dialysis and Transplantation (1). There has been only one small prospective randomized trial comparing HD and peritoneal dialysis in incident patients and this showed no differences in short-term patient outcomes in the small numbers of patients that could be enrolled into the study but the study data were not powered adequately to reach any other conclusion (2). In the absence of evidence that either HD or peritoneal dialysis provide superior patient outcomes the selection of initial dialysis modality should be based on the patient's choice after full education about the different forms of renal replacement therapy that are available, including home HD and live donor and cadaveric transplantation (3).

However, although patient choice is paramount, guidance from unit staff is necessary: venous access can be difficult to achieve and maintain in those less than 5 years of age, and needling of a fistula can be particularly difficult in an uncooperative patient. For these reasons, as well as social ones already discussed, PD is recommended in young children.

1. The National Service Framework for Renal Services Part 1: Dialysis and Transplantation, Department of Health, London, UK, January 2004. (www.doh.gov.uk/nsf/renal/index.htm)
2. Korevaar JC, Feith GW, Dekker FW et al. Effects of starting with hemodialysis compared with peritoneal dialysis in patients new on dialysis treatment: A randomised controlled trial. Kidney Int 2003; 64 :2222-2228
3. National Institute of Clinical Excellence. Full guidance on home compared with hospital haemodialysis for patients with end-stage renal failure October 2002 (www.nice.org.uk)

8.3 After full education and counselling a small proportion of families may opt for active non-dialytic management of advanced chronic kidney disease, including nutritional, medical and psychological support rather than plan to initiate dialysis. The numbers of patients not taken on to dialysis and the reasons for this decision should be subject to audit. (Good Practice)

The decision whether to start or not to start RRT may be difficult (1). It is impossible to set quantitative standards in this difficult area of care, but principles of action can be enunciated and agreed. All patients who are found to have advanced renal failure should be considered for dialysis, and the patient's age, social circumstances or required level of community support should not be a factor leading to exclusion. Nor should lack of facilities for dialysis be acceptable on its own as grounds for exclusion, or fear of litigation a basis for a decision in either direction. Careful medical assessment of any co-morbid conditions from which the patient may suffer is needed, together with whatever medical measures (short of dialysis) are required to correct them or minimise their effects (2). Similarly, patients who have deteriorated will need careful medical and psychological assessment. If it appears that only a brief period of survival of unacceptable quality is likely on dialysis (e.g. less than three months), then the possibility of not starting or stopping dialysis needs to be considered. The interest of the individual patient must remain paramount, and although the opinions of relatives should be consulted, they should not be binding. The responsible consultant nephrologist should solicit views of the patient's family doctor, next of kin, and all carers within the multidisciplinary caring team. The decision to start or not to start RRT must be taken by both the consultant), and the family. The family will need to be fully informed throughout, and to be aware of the options. The most realistic and accurate description of starting or not starting, continuing or not continuing dialysis should be given. The substance of these discussions must be recorded in the patient's notes. If the decision is taken not to initiate, or to stop dialysis, then a management plan of supportive care must be put in place. This must then be carried through in a way that ensures continued support, achieves what seems best from the patient's and family's point of view, and finally enables the patient to die with dignity, when the time comes. Achieving this will often require co-ordinated work with the palliative care team, who should be involved early in the management plan (3). Certain patients who are severely ill, often with conditions affecting several organs, may have a concurrent acute deterioration of their chronic renal failure. The nephrologist, may feel, after discussion, that dialysis is inappropriate given the very poor prognosis from the underlying conditions. Under these circumstances the referring physician would discuss matters with the patient, if possible, and with the family. Guidelines on shared decision-making in the initiation or withdrawal of dialysis have been developed (4).

Two approaches may be taken when a patient presents in uraemia whose ability to cope with, and to enjoy and benefit from dialysis treatment is doubtful. The first approach attempts to make a 'clean' decision on whether or not to start dialysis after a process of consultation and discussion; the second, often called 'trial of dialysis', involves starting a proportion of such patients on dialysis, but with a pre-discussed plan to review whether this should continue beyond a specified point in the near future – usually a few weeks or months. Clearly the expectation is that the outcome in this case will be withdrawal of some patients from dialysis.

1. Tobe SW, Senn JS (for the End-Stage Renal Disease Group). Foregoing renal dialysis: case study and review of ethical issues. Am J Kidney Dis 1996; 28:147–153
2. Campbell ML. Terminal care of ESRD patients forgoing dialysis. ANNA J 1991; 18:202–204
3. Cohen LM, Germian M, Poppel DM et al. Dialysis discontinuation and palliative care. Am J Kidney Dis 2000; 36:140–144
4. Galla JH. Clinical practice guideline on shared decision-making in the appropriate initiation of and withdrawal from dialysis. J Am Soc Nephrol 2000; 11:1340-1342

8.4 Renal replacement therapy should commence when a patient with an eGFR < 15ml/min/1.73m^2 has symptoms or signs of uraemia, fluid overload or malnutrition in spite of medical therapy or before an asymptomatic patient has an eGFR < 6ml/min/1.73m^2. (Good practice)

There are no criteria based on definitive evidence to advise when to start dialysis. In the absence of severe hyperkalaemia or pericarditis there is no definitive evidence to indicate when an asymptomatic patient with advanced renal failure should initiate dialysis. There is consensus that patients should start dialysis when they develop symptoms or signs of fluid overload, hypertension, poor nutrition or uraemia which cannot be controlled by medical therapy such as high dose diuretics, even if their estimated residual renal function is relatively high. Nutritional status and dietary protein intake decrease progressively as renal function declines (1). The medical treatment of the complications of renal failure such as anaemia has improved in the past 10 years and this may explain recent reports of a lack of any relationship between the presence or absence of traditional symptoms of uraemia and residual renal function in patients with stage 5 chronic kidney disease (2). The patients with a higher haemoglobin concentration had fewer symptoms (2) and so relying on the onset of symptoms may result in patients starting dialysis too late. Conversely studies in the Netherlands and Scotland comparing patients who started dialysis at two different levels of residual renal function have shown no advantage to patient survival if adjustments are made for lead time bias in the group of patients starting dialysis with higher residual renal function (3-6). In the multicentre prospective Netherlands study 94 of the 253 incident patients began dialysis later than recommended in the US NKF KDOQI guideline and the adjusted benefit in survival after 3 years on dialysis was 2.5 months in the timely starter group (4). However this benefit may be attributed to lead-time bias since the average delay in initiation of dialysis in the late starter group was 4.1 months. A randomized prospective study to compare 3 year morbidity and mortality after initiating dialysis when patients have a Cockcroft and Gault creatinine clearance of 10-14ml/min/1.73m^2 or 5-7ml/min/1.73m^2 is underway (IDEAL study) (7).

With the evidence that nutritional status deteriorates progressively as renal function declines (1) and symptoms of advanced renal failure are not closely related to the degree of residual renal function in the modern era (2) it is appropriate that international guidelines have attempted to identify the level of residual renal function at which an asymptomatic patient should initiate dialysis. The above considerations fit well with the European Best Practice Guidelines which recommended that renal replacement therapy should commence when a patient with an eGFR < 15ml/min/1.73m^2 has symptoms or signs of uraemia, fluid overload or malnutrition in spite of medical therapy or before an asymptomatic patient has an eGFR < 6ml/min/1.73m^2 (8).

Audit measure 17 – Record of the serum creatinine, the estimated GFR and co-morbidity at initiation of chronic renal replacement therapy (dialysis or transplantation)

1. Ikizler TA, Greene JH, Wingarde RL et al. Spontaneous dietary protein intake during progression of chronic renal failure. J Am Soc Nephrol 1995; 6: 1386-1391
2. Curtis BM, Barrett BJ, Jindal K et al. Canadian survey of clinical status at dialysis initiation 1998-

1999: a multicentre prospective study. Clin Nephrol 2002; 58:282-288
3. Traynor JP, Simpson K, Geddes CC et al. Early initiation of dialysis fails to prolong survival in patients in end-stage renal failure. J Am Soc Nephrol 2002; 13:2125-2132
4. Korevaar JC, Jansen MA, Dekker FW et al. When to initiate dialysis: effect of proposed US guidelines on survival. Lancet 2001; 358:1046-1050
5. Korevaar JC, Dekker FW, Krediet RT. Initiation of dialysis: is the problem solved by NECOSAD? Nephrol Dial Transplant 2003; 18: 1228-1229
6. Termorshuizen F, Korevaar JC, Dekker FW et al. Time trends in initiation and dose of dialysis in end-stage renal disease patients in The Netherlands. Nephrol Dial Transplant 2003; 18: 552-558
7. Cooper BA, Branley P, Bulfone L et al. The Initiating Dialysis Early and Late (IDEAL) study: study rationale and design. Perit Dial Int 2004; 24: 176-181
8. European Best Practice Guidelines for haemodialysis Part 1. Nephrol Dial Transplant 2002; 17: Supplement 7 S1-S111 (http://ndt.oupjournals.org/content/vol17/suppl_7/index.shtml).

8.5 Any decision to discontinue haemodialysis should be made jointly by the patient (when age appropriate) and their carers and the responsible consultant nephrologist and the renal team and the family practitioner. The decision and reasons for it should be recorded in the patient's notes. Renal units should develop guidelines for palliative care of such patients, including liaison with community services. (Good practice)

In addition to patients who clearly present greater than average problems from the outset, there are individuals who have had a period of worthwhile life on dialysis, but whose quality of life worsens because of medical or psychological deterioration, or both simultaneously. Additional difficulty arises when dementia, often fluctuating, or irrecoverable neurological deficit after a cerebrovascular event makes it difficult or impossible to ascertain what the patient's own feelings and wishes might be (1). In practice, the decision to withdraw dialysis has much in common with decision not to start a patient on dialysis. This is because caring staff, patients and relatives all face similar difficult judgements and decisions about the likely quality and quantity of life on dialysis. A similar process to that outlined in deciding whether or not to plan to start dialysis (see above) should be followed when assessing if withdrawal of dialysis is appropriate. There is one study from the UK that suggests that withdrawal from dialysis plays a major role (17%) in overall death rates on dialysis (2), as it does in the USA and Canada (3,4). Recent data from the Dialysis Outcomes and Practice Patterns Study have shown that the rate for withdrawal from HD is 3.5 per 100 patient-years and that not surprisingly "do not resuscitate" orders are associated with older age and nursing home residence (5). In a recent UK study withdrawal of dialysis was the commonest cause of death (38%) in the group of patients commencing dialysis when more than 75 years old (6). Withdrawal of dialysis is an increasing cause of death in dialysis patients and the date of the decision and the reasons for it should be recorded in the patient's casenotes (7). Renal units should develop guidelines for withdrawal of dialysis that include liaison with palliative care and community services.

1. Singer J, Thiel EC, Naylor D et al. Life-sustaining treatment preferences of hemodialysis patients: implications for advance directives. J Am Soc Nephrol 1995; 6:1410–1417
2. Catalano C, Goodship THJ, Graham KA et al. Withdrawal of renal replacement therapy in Newcastle upon Tyne: 1964-1993. Nephrol Dial Transplant 1996; 11:133–139
3. Cohen LM, McCue JD, Germain M, Kjellstrand CJ. Dialysis discontinued: a good death? Arch Intern Med 1995; 155:42–47
4. Friedman EA. The best and worst times for dialysis are now. ASAIO J 1994; 40:107–108
5. Fissell RB, Bragg-Gresham JL, Lopez AA et al. Factors associated with "do not resuscitate" orders and rates of withdrawal from hemodialysis in the international DOPPS. Kidney Int; 2005 68: 1282-

6. Munshi SK, Vijayakumar N, Taub NA et al. Outcome of renal replacement therapy in the very elderly. Nephrol Dial Transplant 2001; 16:1721-1722
7. McLean AM. Dialysis treatment withdrawal – Legal aspects (UK). Nephrol Dial Transplant 1998; 13:1152-1153

APPENDIX

Assessment of Membrane Function

(a) A number of methods to assess peritoneal membrane have been developed, the most commonly used, supported by clinical observation being the Peritoneal Equilibration Test (PET). This test measures two aspects of membrane function, low molecular weight solute transport (expressed as the dialysate:plasma ratio of creatinine at four hours), and the ultrafiltration capacity of the membrane. In the PET as originally described, ultrafiltration capacity is the net volume of ultrafiltration achieved at four hours using a 2.27% glucose exchange (1, 2). In the simplified Standard Permeability Analysis (SPA) test, it is the net volume of ultrafiltration using a 3.86% exchange (3, 4)

(b) Using a standard PET, an ultrafiltration capacity of < 200 mls (includes overfill) is associated with a 50% risk of achieving < 1000 mls ultrafiltration in anuric patients. Using a SPA test, an ultrafiltration capacity of < 400 mls indicates ultrafiltration failure.

(c) The methods of performing PET and SPA tests are well described in the literature, The following points should be remembered in the interpretation of results:

- High concentrations of glucose interfere with many assays for creatinine. It is important to work with the local biochemists to ensure that the appropriate correction for measurement of creatinine in dialysate has been taken into account.
- Remember that dialysis bags are overfilled, mainly due to the additional fluid volume required to perform the 'flush before fill' procedure. Dialysis manufacturers are being encouraged to publish overfill volumes which differ significantly. The typical volume is 100-200ml. The value of 200 ml UF capacity defining ultrafiltration failure quoted above *includes* the flush volume as this is easier for patients to perform (the alternative is weighing before and after flush which is time consuming and difficult).
- The patient should follow their usual dialysate regime, draining out as completely as possible before the test dwell. Large residual volume of dialysate will affect the results.
- Intra-patient variability of the ultrafiltration capacity (~ 20%) is greater than for the solute transport (<10%). Results of the PET/SPA, in particular the ultrafiltration capacity, should always be interpreted in the light of additional

exchanges performed during the same 24-48 hour period (usually collected to assess solute clearance – see below).

- The PET/SPA are not surrogates for measuring solute clearance.

Measurement of Solute Clearance

In measuring solute clearance and planning changes to the dialysis regime, three clinical parameters are essential: Estimates of (1) *patient size*, (2) peritoneal *solute transport* and (3) *RRF*. In each case, the choice of surrogate "toxin", urea or creatinine, interacts with each of these parameters in different ways. At present, there is no clear evidence from the literature that one surrogate is superior to another. Where possible, clinicians should measure both, attempt to reach at least one of the targets, and understand why there appears to be a discrepancy. A number of commercial computer programs exist that are designed to aid dialysis prescription. Whilst some have been validated, good practice dictates that a change in dialysis prescription is checked for efficacy by repeating clearance studies.

(1) Patient Size

In calculating urea clearances, patient size is expressed as an estimate of the total body water (volume of distribution of urea). It is recommended that the Watson formula is used for this (5):

Males: V = 2.447 – 0.09156 * age (years) + 0.1074 * height (cm) + 0.3362 * weight kg)

Females: V = -2.097 + 0.1069 * age (years) + 0.2466 * weight (kg)

Alternatively 58% of body weight (kg) may be used; this is less precise, and will give lower values for Kt/V, especially in obese patients. Creatinine clearances should be corrected for body surface area, normalising to 1.73 m^2.

(2) Peritoneal Solute Transport

Solute transport rates have an important influence on peritoneal creatinine clearance, but not on urea clearance. This means that it is easier to achieve creatinine clearance targets in high transport patients. It should be remembered, however, that these patients might have less satisfactory ultrafiltration. In designing optimum dialysis regimes, patients with low solute transport will require equally spaced medium length dwells, such as are achieved with CAPD and single extra night exchanges (e.g. 5 x 2.5 litre exchanges). Those with high transport are more like to achieve targets with short dwells (APD) plus polyglucose solutions (e.g. 4 x 2.5 litre exchanges overnight, 1 x 2.5 litre evening exchange and 1 x 2.5 litre daytime icodextrin).

(3) Residual Renal Function (RRF)

This is the single most important parameter in PD patients, and also the one most likely to change with time. Clinically significant changes can occur within three months. Because secretion of creatinine by the kidney at low levels of function overestimates residual creatinine clearance, it is recommended to express this as the *mean* of the urea and creatinine clearances.

Estimating Total Ultrafiltration

The total achieved ultrafiltration is best measured from the 24-hour dialysate collections used to calculate solute clearance. For APD patients this is simple as machines now calculate the ultrafiltration volumes precisely. Furthemore, many models store this information over several weeks so that an average value can be obtained. In CAPD patients it is important to remember that each bag is overfilled to achieve flush before fill; the total dialysate drain volume must be measured and sampled from to calculate solute clearance accurately, but the overfill must then be subtracted to calculate the net ultrafiltration. If this is not done then over a 24-hour period the overestimate of ultrafiltration may be anything from 200 to 800 ml depending on manufacturer.(6, 7)

Peritoneal sodium losses are largely determined by convection and are thus proportional to the ultrafiltration volume. Typically 1 litre of ultrafiltration results in 100 mmol of sodium loss in CAPD patients and 70-80 mmol in APD patients.

1. Twardowski ZJ, Nolph KD, Khanna R, et al. Peritoneal Equilibration Test. *Perit Dial Bull* 1987;7:138-47.
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3. Ho-dac-Pannekeet MM, Atasever B, Struijk DG, Krediet RT. Analysis of ultrafiltration failure in peritoneal dialysis patients by means of standard peritoneal permeability analysis. *Peritoneal Dialysis International* 1997;17(2):144-50.
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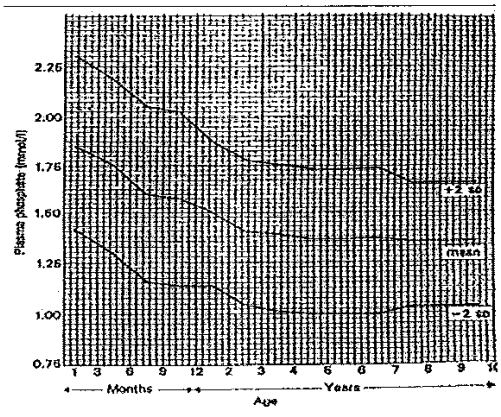
5. Watson PE, Watson ID, Batt RD. Total body water volume for adult males and females estimated from simple anthropometric measurements. *Am J Clin Nutr* 1980;33:27-39.
6. McCafferty K, Fan S. Are we underestimating the problem of ultrafiltration in peritoneal dialysis patients? *Perit Dial Int* 2006;26(3):349-52
7. La Milia V, Pozzoni P, Crepaldi C, Locatelli F. The overfill of bags for peritoneal dialysis as a cause of underestimation of ultrafiltration failure. *Perit Dial Int* 2006;26(4):503-5.

Figure 1. The age appropriate normal range for serum phosphate levels.



Enclosure 12J

Standard Peritoneal Dialysis Prescription

Date				
Dialysis Fluid & Volume				
Therapy volume				
Fill Volume				
Therapy time				
Last Bag Fill				
Maximum UF				
Additives				
Pre Weight				
Post Weight				
Initial Drain				
Total UF				
Dwell Time				
Lost Dwell				
Doctor				
Nurses				

Low Fill Peritoneal Dialysis Prescription

Date				
Min. Drain Vol				
Min. Drain Time				
-ve UF Limit (%)				
+ve UF Limit (mls)				
Dialysis Fluid & Volume				
Therapy Volume				
Fill Volume				
Therapy Time				
Last Bag Fill				
Additives				
Maximum UF				
Pre Weight				
Post Weight				
Initial Drain				
Total UF				
Dwell Time				
Lost Dwell				
Doctor				
Nurse				

CHILD'S NAME

PARENT/GUARDIAN'S NAME

HOME CHOICE TRAINING PROGRAMME.

	OBSERVED	ATTEMPTED						MINIMAL SUPERVISION			PROFICIENT
		DOLL			CHILD						
		1	2	3	1	2	3	1	2	3	
Preparation of equipment 1. Cleaning of machine 2. Cleaning of trolley/tray 3. Setting out requirements Selection of correct fluids **Daily Exit site care** • Signs of infection											

	OBSERVED	ATTEMPTED						MINIMAL SUPERVISION			PROFICIENT
		DOLL			CHILD						
		1	2	3	1	2	3	1	2	3	
Skilled Handling of equipment											
1. Hygienic Environment											
2. Hand washing technique											
3. Lining Machine											
4. Setting programme • Low Fill mode / Standard mode • Total fill volume • Therapy time • Fill volume • Last bag volume											
5. Additives • Heparin • Antibiotics											
6. Connecting patient to machine											
7. Discontinuing Home Choice											
8. Ending therapy early											

	DISCUSSED AND KNOWLEDGEABLE
ASSESSING DRY WEIGHT **RECOGNISING FLUID OVERLOAD** **RECOGNISING PERITONITIS** **ALARMS** – Knowledge of main alarms and action • Reload set • Check lines and bags • Reprime patient line • Check supply line • Check drain line • Low drain volume • Fill / drain not finished • Caution negative UF • Caution positive UF • System error • Verify initial drain **BYPASSING** **MANUAL DRAIN** **POWER FAILURE**	

	DISCUSSED AND KNOWLEDGEABLE
ORDERING SUPPLIES STORAGE OF FLUIDS AND SUPPLIES SAFE DISPOSAL OF SHARPS DISPOSAL OF WASTE	
PROBLEMS • Contaminated equipment • Defective equipment • Impaired drainage • Dehydration • Fluid overload • Leakage at exit site • Blood stained effluent USE OF DRY BAG USE OF CATHETER CLAMP FIBRIN IN LINES	

	DATE AND TIME
PHARMACIST VISIT	

I / We _____ parent/guardian of _____

have completed training to the satisfaction of the renal staff and feel confident and competent that I/we can perform dialysis unsupervised at home.

Parent / Guardian signature and Date

_____

Renal Nurse Signature

_____

Consultant signature

_____

Initial Home visit :-

ROYAL BELFAST HOSPITAL FOR SICK CHILDREN

COMMENCING STANDARD HOME CHOICE DIALYSIS

Requirements

- machine and cassette
- drainage bag
- dialysis fluid
- trolley wipes
- connection shield
- heparin
- 2 ml syringe
- green and blue needle
- sterets

1. Wash hands and collect equipment.

2. Clean machine with mild detergent and water, and tray with alcohol wipes.

3. Take bags out of covers and place on tray - check expiry date, amount, dextrose, green seals, blue cover, leakage and clarity of fluid. If adding antibiotics or heparin see points a. - b. at end of protocol.

4. Switch on at mains and at back of machine. Machine will display PLEASE WAIT then STANDARD MODE ON then PLEASE WAIT. Put bag on heater.

5. Programme machine - Press down arrow and review programme. (If programme unchanged continue on)
   Press red STOP button and number of cycles and dwell time will be displayed.

6. Press green START button twice - LOW FILL MODE OFF then LOAD THE SET displayed.

7. Wash hands (short social wash).

8. Take disposable set out of the bag and close all clamps.(6 in total)

9. Open door, load cassette, close door ensuring lines are inserted correctly and place organiser on door clip.

10. Attach drain line and 'Y' connection.

11. Place bag on heater plate

12. Press green GO button. PLEASE WAIT displayed
SELF TESTING displayed.
When complete CONNECT BAGS displayed

13. Wash hands for one minute - rinse and repeat - dry hands using theatre technique.

14. Connect lines to bags:-
- line with RED clamp must go heater bag
- WHITE clamps to storage bags
- BLUE clamps to last fill bag if being used for different dextrose otherwise use as a white clamp

15. Break GREEN seals.

16. Open all clamps on bags and on patient line.

17. Press the GREEN go button PRIMING will be displayed. (This can take up to 6 mins)

18. When completed, machine now displays CONNECT YOURSELF.

19. Expose tube and open connection shield.

20. Wash hands for one minute and apply hibisol. If hands have touched anything repeat 2 one minute hand washes.

21. Holding patient line in left hand remove minicap and discard, apply connection shield (ensure it is closed by rotating), now connect to catheter.

22. Open roller clamp on catheter.

23.Press GREEN go button INITIAL DRAIN will be displayed. Press down arrow, initial drain volume displayed, if nothing coming out and / or child complaining of pain press RED button, then down arrow until BYPASS displayed then press and repeat - machine will display FILL 1 OF - - .

a) Draw up heparin/antibiotics with green needle, then add to each bag with a new blue needle.
b) Helper places bag on heater plate.

DISCONTINUING HOME CHOICE DIALYSIS

Put on apron
Wash hands- social wash

1. The machine will read END OF THERAPY, press the down arrow and record INITIAL DRAIN, press the down arrow again and record TOTAL UF. Press the down arrow once again and record AVERAGE DWELL TIME. Press the down arrow again and record LOST or ADDED DWELL TIME.

2. Press the green go button. The display will then read CLOSE ALL CLAMPS.

3. Close ALL clamps on lines and roller clamp on patient catheter.

4. Press the green go button, display reads DISCONNECT YOURSELF.

5. Clean trolley/tray with sani cloth and set out minicap.

6. Open mini cap (peel open minicap packaging being careful not to touch the minicap inside, leave it in packaging but accessible).

7. Carry out X 2 one minute hand washes

8. Disconnect patient line and apply mini cap, being extremely careful not to touch exposed end of catheter or inside of minicap. Ensure cap is secured tightly to end of catheter.

9. Unload the used set from the machine by releasing the door lever where the cassette is placed.

10. Press green go button, machine will now read TURN ME OFF. Turn the machine off by flicking switch off at the rear of the machine and at the mains.

11. Dispose of the used cassette, empty dialysis fluid bags and drain line/drainage bags in the correct manner as per trust policy.

Updated January 2011.

COMMENCING STANDARD HOME CHOICE DIALYSIS
(Physioneal)

Aim

To ensure safe and effective dialysis treatment is performed as per individual prescription.

Requirements

- machine and cassette
- drainage bag or extension drain line
- dialysis fluid (as prescribed on prescription sheet)
- trolley wipes (Sani-cloth 70)
- connection shield

  If required
  - Heparin Sodium 1000 units/ml
  - 2 ml syringe
  - green and blue needle
  - Clinell wipes

1. Wash hands and collect equipment.

2. Clean machine with mild detergent and water, and trolley/tray with alcohol wipes.

3. Take bags out of covers and place on trolley/tray – check expiry date, amount, dextrose concentration, green seals, blue cover, leakage and clarity of fluid. If adding antibiotics or heparin see relevant procedure. Hang bags on stand remembering to break seal between bag sections.

4. Switch on at mains and at back of machine. Machine will display PLEASE WAIT then STANDARD MODE ON then PLEASE WAIT. Ensure electrical connections are well secured to prevent interruption in power supply.

5. Programme machine – Press down arrow and review programme. (If programme unchanged continue on)
   Press red STOP button and number of cycles and dwell time will be displayed.

6. Press green START button twice – LOW FILL MODE OFF then LOAD THE SET displayed.

7. Put on apron. Wash hands (short social wash) following 7 step technique.

8. Take disposable set out of the bag and close all clamps.(6 in total, 10 if using 8-prong set)

9. Open door, load cassette, close door ensuring lines are inserted correctly and place organiser on door clip.

10. Attach drainage bag or extension line.

11. Place prescribed bag on heater plate, having first checked that upper compartment has fully emptied. Place at an angle ensuring green seal between compartments is covered with fluid at all times.

12. Press green GO button. PLEASE WAIT displayed SELF TESTING displayed.
When complete machine will display CONNECT BAGS.

13. Wash hands for one minute – rinse and repeat – dry hands using theatre technique.

14. Connect lines to bags:-line with RED clamp must go heater bag
- WHITE clamps to storage bags
- BLUE clamps to last fill bag if being used for different dextrose otherwise use as a white clamp

15. Break GREEN seals.

16. Open all clamps on bags and on patient line.

17. Press the GREEN go button PRIMING will be displayed. (This can take up to 6 mins)

18. When completed, machine now displays CONNECT YOURSELF.

19. Make catheter tube accessible and open connection shield packaging and set onto clean trolley/tray.

20. Wash hands for one minute, rinse and repeat. Dry following theatre technique.

21. Holding patient line in left hand apply connection shield (ensure it is closed by rotating). Hold patient line in right hand, take patient catheter in left hand, remove minicap and discard. Securely connect patient line to catheter using non-touch technique.

22. Press GREEN go button x2 VERIFY INITIAL/ INITIAL DRAIN will be displayed.

23. Open roller clamp on catheter. (If patient line contaminated dispose of set and recommence procedure. If patient catheter contaminated contact hospital immediately).

24. Press down arrow, initial drain volume displayed, if minimal or no drainage and/or child complaining of pain press RED button, then down arrow until BYPASS displayed then press and repeat – machine will display FILL 1 OF - - **CAUTION MUST BE TAKEN WHEN DECIDING WHETHER TO BYPASS THE INITIAL DRAIN AS A PATIENT MAY HAVE A LAST BAG FILL INSITU FROM PREVIOUS DIALYSIS. Contact consultant if pain/no drain and aware there is fluid in the peritoneal cavity(last bag fill).**

February 2011