McCarthy, Miriam

From: Fisher, Ruth
Sent: 02 November 2004 17:01
To: Browne, Andrew
Cc: McCarthy, Miriam
Subject: FW: PQ3292/04: Mrs Robinson - Hyponatraemia - Further infor. received from Norman Morrow

Andrew
For your information.
Ruth

-----Original Message-----
From: Morrow, Norman (Pharm.)
Sent: 02 November 2004 14:24
To: Fisher, Ruth
Subject: RE: PQ3292/04: Mrs Robinson - Hyponatraemia - Immediate

uth

I have attached a document from the School of Pharmacy providing a brief synopsis of the ground covered in the pharmacy undergraduate course allied to fluids and electrolytes.
I trust this is helpful.

Norman Morrow

CLT- serum electrolytes.doc

-----Original Message-----
From: Fisher, Ruth
Sent: 29 October 2004 15:22
To: Patterson, Nicki; Morrow, Norman (Pharm.)
Subject: FW: PQ3292/04: Mrs Robinson - Hyponatraemia - Immediate
Importance: High

Nicki
Norman

In regard to the attached PQ can you confirm whether or not nurses and pharmacists receive teaching/training on fluids and electrolytes?

Thanks
Ruth
Extn 20545

-----Original Message-----
From: Browne, Andrew
Sent: 29 October 2004 14:21
To: Fisher, Ruth
Subject: FW: PQ3292/04: Mrs Robinson - Hyponatraemia

Ruth
A question for Nicki Patterson and Norman Morrow to follow up but probably not important to the PQ.
Andrew

-----Original Message-----
From: McCarthy, Miriam
Sent: 29 October 2004 13:56
To: Browne, Andrew
Subject: FW: PQ3292/04: Mrs Robinson - Hyponatraemia

DHSSPS

Andrew,

Are you content with ammendments--were you able to establish whether nurses and pharmacists also receive

teaching/training?

Miriam

-----Original Message-----

From: Browne, Andrew
Sent: 29 October 2004 13:34
To: Lindsay, Sharon
Cc: Campbell, Dr Henrietta; Hamilton, Andrew; Sullivan, Dean; McCarthy, Miriam; Fisher, Ruth; McCaw, Darren
Subject: PQ3292/04: Mrs Robinson - Hyponatraemia

<< File: PQ3292-04 HYPONATRAEMIA.doc >>
Sharon
Draft reply attached.
Andrew

DHSSPS

SERUM ELECTROLYTES

Five electrolytes are of major interest. These are sodium, potassium, chloride, magnesium and calcium. Where applicable, some basic information on the signs and symptoms, together with the aetiology and treatment of electrolyte abnormalities, are presented.

1 SODIUM (136 - 145 mmol/l)

Sodium is the main cation in extracellular fluid and, as such, contributes significantly to serum osmolality. Because sodium ions make such a contribution to osmotic pressure in the body, total body sodium plays an important role in determining extracellular fluid volume. Sodium intake from the diet is primarily as added salt at the table or as salt used in cooking. Many convenience foods have high sodium loads especially canned soups. These items should be avoided by those patients who have been prescribed a low sodium diet, e.g. in the treatment of hypertension. Certain drugs can also produce a large body load of sodium, e.g. carbenicillin sodium contains 5.4 mmol of sodium per gram of product; the usual dose is 5 grams every four to six hours. ***[This was also recently brought to note about Co-codamol effervescent tablets which contain 18 mmol/tab]*** This could be of significant importance to the very ill patient, leading to hypernatraemia and hypokalaemia.

The major route of sodium excretion is via the kidneys. Renal conservation of sodium, in the absence of sodium intake, is virtually complete. Anti-diuretic hormone maintains normal serum osmolality ***[ADH secretion stimulated by angiotensin II, arterial and venous barorecoeptors, volume receptors, stress (inc. pain)and drugs e.g. morphine]*** while aldosterone controls total body sodium [***aldosterone retains Na/H_2O at the distal renal tubule]***. Thirst is a further potent regulator of serum sodium concentration.
[Another hormone affecting Na reabsorption is Natriuretic peptide hormone, which is secreted by the cardiac atria in response to atrial stretch e.g. associated with volume expansion). It is natriuretic i.e. increases Na secretion and reduced the aldosterone concentration.

1.1 Normonatraemia with problems

This occurs when the body gains or loses water and sodium in the proper ratio to one another ***[such isotonic losses are commonly associated with burns or haemorrhages]*** and is easily correctable by sodium and fluid restriction or administration of sodium and fluids.

1.2 Hyponatraemia

A low serum sodium means that there is overhydration, too little sodium in the body, or a mixture of both these factors. This is a frequently reported abnormality. The signs and symptoms include lethary, tetany and convulsions (likely if serum sodium drops below 110 mmol/1). Mild hyponatraemia is usually symptom free.

Aetiology

(i) **abnormal losses of sodium-containing fluid with inadequate replacement**, e.g. replacement of fluid loss in diarrhoea (isotonic) with water alone, renal salt wasting states like the diuretic phase of acute renal failure, diabetic ketoacidosis and excessive sweating
(ii) **migration of sodium into the cells due to failure of the sodium pump**. This is a common feature of chronic debilitating diseases and may also be a consequence of potassium depletion
(iii) **water overload or primary water retention in excess of sodium,** e.g. in compulsive water drinkers ***[rarely a problem as adults can excrete up to 2 ml/min]***, in SIADH (syndrome of inappropriate anti-diuretic hormone secretion) ***[a syndrome of several causes, including chest infections and some tumours]***, or in overload with hypotonic intravenous fluids leading to expansion of the extracellular fluid

(iv) **artefact,** e.g.. dilution of blood sample with intravenous fluid from an infusion into the same arm, or failure to correct for a marked hyperlipidaemia by expressing the results in relation to plasma water. Hyperglycaemia may also give rise to a falsely depressed sodium concentration.

DRUGS – All types of antidepressants (esp. SSRIs) in elderly especially, NSAIDs, diuretics

Treatment - correct the underlying disorder. Fluid and salt administration or restriction are required depending on the aetiology of the condition.

1.3 Hypernatraemia

Hypernatraemia nearly always indicates water depletion. The condition does not occur often since thirst is normally an irresistible stimulus. Patients who are comatose can, however, develop this abnormality. It should also be stressed that the thirst reflex is depressed in the elderly. The signs and symptoms of hypernatraemia include fever, hypertension, CNS irritability, congestive heart failure and dehydration. Coma will occur above 155-160 mmol/l.

Aetiology

[Primary mineralocorticoid excess – Cushings and Conns syndrome)
[Secondary mineralocorticoid excess – CHF, Nephrotic syndrome, hepatic cirrhosis with ascites]

(i) loss of water in excess of sodium - patients losing large amounts of water from kidneys, lungs or skin without proper replacement , diabetes insipidus
(ii) decreased fluid intake, e.g.. in states of unconsciousness, in the very young or elderly due to neglect, or if water is unavailable as in a desert
(iii) increased sodium intake or retention in excess of water, e.g.. steroid therapy or renal failure.

[Drug induced – clonidine, lactulose, methyldopa, oral contraceptives]
[Drug induced as a result of a nephrogenic diabetes insipidus like syndrome where renal tubules are unresponsive to ADH – therefore polyuria, polydipsia or dehydration result (especially with Lithium and phenytoin)

Treatment - administer water by mouth or fluid intravenously in the form of Dextrose 5% w/v. Treat any precipitating factor.

2 POTASSIUM (3.5 - 5.0 mmol/l) – *[Total amount 3000 mmol in body]*

Potassium is the main intracellular fluid cation with its concentration in intracellular fluid being about 20-30 times that of extracellular fluid. There is a tendency for K+ to diffuse out of cells down this concentration gradient just as there is a tendency for Na+ to diffuse into cells. Potassium is lost from the cells whenever dehydration occurs and returns to the cells when the state of hydration is corrected. Potassium distribution is also influenced by acid-base disturbances. In acidosis the movement of potassium is out of the cells in exchange for hydrogen ions while alkalosis causes potassium to move into the cells. Insulin promotes the entry of potassium and glucose into the cells. Conversely in conditions of relative lack of insulin, e.g. poorly controlled diabetes mellitus, there is a marked loss of potassium from the intracellular fluid. In catabolic states, whenever there is a breakdown of intracellular protein, e.g.. post major surgery, during a severe infection, or in starvation states, potassium is lost from the cells. Conversely, potassium is taken up in anabolic states.

There is a small loss of potassium in the faeces and through the skin; however, the main loss of potassium from the body is via the kidney *[Potassium is almost completely reabsorbed at the*

proximal tubule, but secreted at the distal convoluted tubule in response to the need to maintain membrane neutrality (Na reabsorption here causes an imbalance in charge)]. Even in the presence of a total body deficit of potassium, the kidney is unable to conserve the cation completely. It is clear, therefore, that the kidney responds less well to potassium restriction than to sodium restriction. The kidney does respond well, however, to increased potassium intake ***[normal dietary intake 60-200 mmol per day – more than adequate to replace losses].***

Serum potassium may not accurately reflect cellular potassium stores, i.e. there may be a large total body deficit of potassium with only a relatively small drop in serum potassium concentrations ***[Only 2% of the exchangeable total body potassium is in the ECF].*** It is for this reason that the measurement of intracellular potassium, e.g. red blood cell potassium, has been suggested as a more meaningful alternative to serum potassium measurement. Aldosterone and cortisol influence the distribution of potassium between intraand extracellular fluid. Aldosterone also gives rise to sodium retention, potassium depletion and alkalosis.

2.1 Hypokalaemia

Mild hypokalaemia is rarely symptomatic. More severe hypokalaemia results in muscle weakness ***[hypotonia, paralytic ileus, depression and confusion]***, ECG changes (flattening or inversion of the T wave, sagging of the ST segment) ***[also, a prolonged P-R interval]***, is often associated with metabolic alkalosis, and leads to increased sensitivity to the toxic effects of digoxin.

Aetiology

[Primary or secondary hyperaldosteronism]
[Renal tubular damage- nephrotoxics e.g. gentamicin]

(i) inadequate intake of potassium post-operatively and in starvation
(ii) abnormal losses, e.g. nasogastric suction, vomiting, diarrhoea, fluid from a fistula, or in urine during renal tubular acidosis
(iii) combination of (i) and (ii)
(iv) iatrogenic - several drugs can induce hypokalaemia including diuretics, aspirin, terbutaline, glucocorticoids, carbenicillin sodium, amphoteracin and insulin (in excess) ***[drugs which affect aldosterone can induce hypokalaemia – corticosteroids mimic aldosterone, increasing K loss. Most common drug cause of hypokalaemia are thiazide and loop diuretics which increase the concentration of Na at the distal convoluted tubule]***
(v) acid-base disturbances - alkalosis redistributes potassium by shifting it into the cells and results in loss into urine

Treatment – replace potassium with intravenous potassium chloride when alkalosis is present. This will give a rapid correction of the hypokalaemia. Care must be taken to control the rate of administration of potassium since this cation is toxic to the heart. The maximum rate of administration should not exceed 10-15 mmol/hour. The solution is normally administered in a saline solution as, if given in dextrose, blood glucose and insulin fluctuations will make it difficult to monitor the potassium replacement. If the condition does not require rapid correction, the potassium should be given orally.

2.2 Hyperkalaemia

Levels of potassium **above 6.5 mmol/1 are dangerous** and should be treated as a **medical emergency**. Hyperkalaemia is caused by the ingestion or administration of potassium in excess of the body's ability to eliminate it via the kidney. Symptoms, which include muscular weakness and paresthesias, do not usually occur until the serum concentration is greater than 6 mmol/l. ECG changes associated with hyperkalaemia include peaked T waves, prolonged PR and QRS intervals and diminished P waves. There is also a significant bradycardia.

Aetiology

(i) renal insufficiency *[common cause]*
(ii) redistribution of potassium between extracellular and intracellular fluid, e.g. hypoaldosteronism as in Addison's disease, acidosis
(iii) increased tissue catabolism
(iv) increased fragility of blood cells, e.g. leukaemia, haemolytic anaemia
(v) incorrect collection or storage of specimens
(vi) iatrogenic, e.g. potassium supplements plus spironolactone, captopril and other ACE inhibitors plus diuretics that antagonise aldosterone (spironolactone) ***[or are potassium sparing e.g. amiloride or triamterene]***, transfusion with old blood

Treatment – ***[Long-term]*** treatment of hyperkalaemia includes the use of polystyrene sulphonate resins (e.g. calcium polystyrene sulphonate), sodium bicarbonate, and glucose and insulin infusions. ***[If due to renal failure, patient is put on low K^+ diet]*** Dialysis may be carried out in an emergency situation or when renal function is very poor. ***[Emergency treatment – calcium gluconate (or chloride) 10% solution, with 10 mls over 5 minutes (this antagonises the effect of hyperkalaemia on cardiac tissue). This is followed by 50 g of glucose and 20 units of soluble insulin by IV infusion to lower the K^+ levels (normally works within 30 minutes).]***

Three main instances for monitoring potassium concentrations in patients are during diuretic therapy, during digoxin therapy, and during treatment with intravenous and TPN fluids.

3 CHLORIDE (95 - 105 mmol/l)

As far as human physiology is concerned, chloride metabolism generally follows that of sodium, and usually chloride deficiency or excess is not a clinical problem in itself. A reduction in plasma sodium is associated with a reduction in plasma chloride and vice-versa. As a general rule, therefore, the concentration of this electrolyte does not give much additional information to the practitioner, but has a place during the monitoring of, e.g. metabolic acidosis due to chronic renal failure or treatment with carbonic anhydrase inhibitors.

4 MAGNESIUM (0.7 - 1.2 mmol/l) *[essential constituent of many enzyme systems]*

Magnesium is primarily located intracellularly and in bone. Only about *1 %* is found in the serum. Like potassium, marked alterations in the body's content of magnesium can occur with little or no change in plasma magnesium concentration. Magnesium depends on the kidney for its elimination. ***[magnesium is not well absorbed from the GI tract and hence is used as an osmotic laxative in the form of Magnesium sulphate or hydroxide]***

4.1 Hypomagnesaemia

This is usually part of a deficiency state involving other electrolytes, e.g. sodium and potassium ***[hypomagnesaemia may also cause hypocalcaemia (often) as well as hyponatraemia and hypokalaemia]***. As with hypokalaemia, an increased toxicity to digitalis glycosides is expected. The symptoms are similar to hypocalcaemia and involve neuromuscular hyperexcitability including irritability, tremor, tetany and convulsions.

Aetiology

(i) excessive loss of gastrointestinal fluids, e.g. severe diarrhoea *[Mg lost in large amounts in GI fluids]*
(ii) long term therapy with magnesium free parenteral fluids
(iii) diuretic therapy may deplete magnesium as well as potassium
(iv) chronic alcoholism and/or cirrhosis of the liver can lead to depletion of body magnesium stores
(v) primary hypomagnesaemia is a rare autosomal recessive disease of nuclear aetiology.

Treatment - the main treatment is to correct the underlying cause of the condition. Magnesium chloride or sulphate is occasionally needed to correct magnesium deficiency in alcoholism or after prolonged diarrhoea or vomiting which has been treated with parenteral fluid and nutrition without magnesium supplements. A solution of magnesium chloride or sulphate (35-50 mmol) may be added to 1 litre of 5% Dextrose or other isotonic solution and given over a period of 12 to 24 hours. Repeated measurements of plasma magnesium are advisable to determine the rate and duration of the infusion. The dose should be reduced in renal failure. Measurement of magnesium is indicated in normocalcaemic tetany.

4.2 Hypermagnesaemia *[RARE]*

There are no symptoms attributable to magnesium excess unless the plasma magnesium concentration exceeds 2.5 mmol/l. Nausea and vomiting, muscle weakness and impaired consciousness may then develop. There may also be impaired neuromuscular transmission and cardiac conduction.

Aetiology

(i) acute renal failure
(ii) haemodialysis against hard water with a high magnesium content
(iii) administration of magnesium products to patients with renal failure, e.g. magnesium-containing antacids.

Treatment - eliminate the underlying cause of the condition.

5 CALCIUM (2.25 - 2.6 mmol/l)

Calcium is the most abundant mineral in the body *[1 kg of Ca in average person]*. The normal dietary intake of calcium is about 25 mmol/day although the minimum daily requirement is about half that figure. Calcium absorption tends to decrease with age and higher dietary intake may be required in the elderly. Serum calcium is a measure of bound and free calcium concentrations (the cation is approximately 50% bound to serum albumin) *[46 % bound]*, and, therefore, measured values must be adjusted for hypoproteinaemia. A value of 0.02 mmol/l should be added for every g/l of serum protein below 72 g/l. Homeostasis of calcium is controlled by parathyroid hormone *[this hormone increases serum calcium by actions on osteoclasts, kidney and gut]* and 1,25 dihydroxycholecalciferol. *[In alkalosis, H^+ dissociates from albumin and Ca^{2+} binding increases, decreasing the levels of free Ca – therefore can get the symptoms of hypocalcaemia – opposite in acidosis]*

5.1 Hypocalaemia

Serum concentrations below 2.25 mmol/l but above 1.6 mmol/l are usually symptom free. Tetany usually only occurs below 1.6 mmol/l.

Aetiology

(i) chronic renal failure
(ii) deficient intake or absorption of calcium and/or vitamin D (giving rise to rickets or osteomalacia)
(iii) hypoparathyroidism
(iv) hyperphosphataemia.
[hypoalbinaemia also]
[alkalaemia – e.g. from hyperventilation, increases binding of Ca to albumin and therefore decreases free Ca, which can cause paraesthesia and tetany]
[drugs causing – phenytoin, Phenobarbital,
Treatment - therapy of calcium disorders hinges on the treatment of the underlying cause (e.g. increase vitamin D and calcium intake) or in treatment of the underlying disease mechanism (e.g. in parathyroid disease).

5.2 Hypercalcaemia

Serum concentrations of calcium above 3.75 mmol/l represent a medical emergency with the danger of cardiac arrest. Levels of 2.6-3.75 mmol/l should be treated once the diagnosis is made to avoid renal damage.

Aetiology

(i) hyperparathyroidism ***[most common]***
(ii) hyperthyroidism
(iii) excessive intake of vitamin D or calcium ***[also Vitamin A – causes bone hypertrophy]***
(iv) sarcoid
(v) malignant deposits in bone ***[common – multiple myeloma and carcinomas of bone]].***
[acromegaly]
[Drugs – thiazide diuretics, Li, tamoxifen]

Treatment - see above. Hydration plus other measures if required ***[frusemide (inhibits tubular reabsorption of Ca), correction of dehydration with saline, biphosphonates to decrease bone turnover.***

6 PHOSPHATE (0.8 - 1.4 mmol/l)

Disturbed phosphate concentrations are rarely symptomatic in themselves but may affect calcium metabolism. The phosphate values are higher at birth (1.34-3.36 mmol/l) and in children (1.28-2.24 mmol/l) than in adults. This is associated with increased concentrations of growth hormone in infants and children.

6.1 Hypophosphataemia

Aetiology

(i) hyperparathyroidism

(ii) rickets (vitamin D deficiency)
(iii) steatorrhoea
(iv) Fanconi syndrome (impaired tubular absorption of phosphate)
(v) prolonged ingestion of phosphate-binding antacids.
[chronic alcohol abuse]
[parenterally fed patients]

[Clinically – severe hypophosphataemia causes muscle weakness and wasting and skeletal wasting]

6.2 Hyperphosphataemia

Aetiology

(i) advanced renal insufficiency *[chronic renal failure]*
(ii) true or pseudohypoparathyroidism
(iii) hypervitaminosis D
(iv) hypersecretion of growth hormone.

Treatment - the treatment of phosphate disorders depends on the treatment of the underlying cause of the phosphate imbalance.

CASE STUDIES

1. A 75 year old woman was hospitalised with a chief complaint of increasing shortness of breath and orthopnea over the past week. The patient had been treated previously for congestive heart failure, but has not taken any medication over the past two weeks. The patient was noted to have severe pedal oedema and to be in severe respiratory distress. Serum biochemistry tests revealed the following values: sodium 123 mol/l (136 to 145); potassium 4.1 mmol/l (3.5 to 5.0); chloride 90 mmol/l (95 to 105); carbon dioxide 30 mmol/l (20 to 30); blood urea nitrogen (BUN) 8 mmol/l (1 to 5); glucose 10 mmol/l (3.6 to 11). Why should this patient with a serum sodium concentration of 123 mmol/l not be given sodium chloride to return her serum sodium concentration to normal values?

2. A 27 year old type I (juvenile-onset) diabetic was hospitalised for ketoacidosis. The blood sugar was 91 mmol/l (3.6 - 11); urine output was 135 ml/hr (50); urine was positive (4+) for sugar and ketones. The blood pH was 7.1 and the serum potassium concentration was 4.1 mmol/l. Why should the clinician monitoring this patient be concerned about the "normal" potassium serum concentration?

Browne, Andrew

From: Fisher, Ruth
Sent: 02 November 2004 17:05
To: Browne, Andrew
Cc: McCarthy, Miriam
Subject: FW: PQ3292/04: Mrs Robinson - Hyponatraemia - Further infor. received from Nicki Patterson

Andrew
For your information.
Ruth

-----Original Message-----
From: Patterson, Nicki
Sent: 02 November 2004 13:14
To: Fisher, Ruth
Subject: RE: PQ3292/04: Mrs Robinson - Hyponatraemia - Immediate

Ruth,

discussed by phone on Friday (29/10/04) I can confirm that nursing staff, as part of the pre - registration cirriculum ceive teaching/training on fluids and electrolytes.

If you require any further detail please do not hesitate to contact me.

Nicki Patterson
Ext[redacted]

-----Original Message-----
From: Fisher, Ruth
Sent: 29 October 2004 15:22
To: Patterson, Nicki; Morrow, Norman (Pharm.)
Subject: FW: PQ3292/04: Mrs Robinson - Hyponatraemia - Immediate
Importance: High

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Norman

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Thanks
Ruth
Extn [redacted]

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Sent: 29 October 2004 14:21
To: Fisher, Ruth
Subject: FW: PQ3292/04: Mrs Robinson - Hyponatraemia

Ruth
A question for Nicki Patterson and Norman Morrow to follow up but probably not important to the PQ.
Andrew

-----Original Message-----
From: McCarthy, Miriam
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To: Browne, Andrew
Subject: FW: PQ3292/04: Mrs Robinson - Hyponatraemia

Andrew,

Are you content with ammendments--were you able to establish whether nurses and pharmacists also receive teaching/training?

Miriam

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Sent: 29 October 2004 13:34
To: Lindsay, Sharon
Cc: Campbell, Dr Henrietta; Hamilton, Andrew; Sullivan, Dean; McCarthy, Miriam; Fisher, Ruth; McCaw, Darren
Subject: PQ3292/04: Mrs Robinson - Hyponatraemia

<< File: PQ3292-04 HYPONATRAEMIA.doc >>
Sharon
Draft reply attached.
Andrew

DHSSPS

074-039-172

Browne, Andrew

From: Fisher, Ruth
Sent: 29 October 2004 15:40
To: Browne, Andrew
Cc: McCarthy, Miriam
Subject: RE: PQ3292/04: Mrs Robinson - Hyponatraemia

Andrew
Nicki Patterson has confirmed that nurses receive training on fluids and electrolytes.
Dr Morrow is to check on the position in regard to pharmacists & will let me know the outcome.
Ruth

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From: Browne, Andrew
Sent: 29 October 2004 14:21
To: Fisher, Ruth
Subject: FW: PQ3292/04: Mrs Robinson - Hyponatraemia

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A question for Nicki Patterson and Norman Morrow to follow up but probably not important to the PQ.
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Miriam

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<< File: PQ3292-04 HYPONATRAEMIA.doc >>
Sharon
Draft reply attached.
Andrew

McCarthy, Miriam

From: Browne, Andrew
Sent: 29 October 2004 14:28
To: Lindsay, Sharon
Cc: Campbell, Dr Henrietta; Hamilton, Andrew; Sullivan, Dean; McCarthy, Miriam; Fisher, Ruth; McCaw, Darren
Subject: FW: PQ3292/04: Mrs Robinson - Hyponatraemia

Sharon
Slightly amended draft as suggested by Miriam McCarthy.
Andrew

-----Original Message-----
From: Browne, Andrew
Sent: 29 October 2004 13:34
To: Lindsay, Sharon
Cc: Campbell, Dr Henrietta; Hamilton, Andrew; Sullivan, Dean; McCarthy, Miriam; Fisher, Ruth; McCaw, Darren
Subject: PQ3292/04: Mrs Robinson - Hyponatraemia

PQ3292-04 IYPONATRAEMIA.do

Sharon
Draft reply attached.
Andrew

DHSSPS

PQ3292/04

DATE FOR ANSWER: TUESDAY 2 NOVEMBER 2004

Mrs Iris Robinson (Strangford). To ask the Secretary of State for Northern Ireland, what action he has taken to (a) improve knowledge of intravenous fluid management and (b) prevent deaths of children from dilutional hyponatraemia in hospitals in the Province and if he will make a statement. **(194641)**

ANGELA SMITH

There is undergraduate teaching on fluids and electrolytes and the topic of fluid management is included during the induction training of junior doctors. Also the Department of Health, Social Services and Public Safety has issued guidance on the prevention of hyponatraemia in children (March 2002) and the Clinical Resource Efficiency Support Team (CREST) has also issued guidance on the management of hyponatraemia in adults (June 2003).

The guidance issued by the Department in March 2002 stresses that hyponatraemia can be extremely serious and potentially fatal. The guidance has been prepared as an A2 sized poster for display in all hospital units where children may receive IV fluids or oral rehydration.

_____ Date: _____

DHSSPS

BACKGROUND NOTE TO PQ3292/04

1. This is one of 2 Parliamentary Questions asked by Iris Robinson MP regarding hyponatraemia and the investigation of hospital deaths in the Province (PQ 3293/04). They are thought to be linked to the death of Lucy Crawford and a recent television programme on the subject.

2. Hyponatraemia can be extremely serious and in the past few years has been responsible for two deaths of children in Northern Ireland that have been widely reported in the media. Hyponatraemia is a problem of water balance and most often reflects the failure to excrete water. Stress, pain and nausea are all potential stimulators of the antidiuretic hormone ADH which inhibits water excretion.

3. Any child receiving IV fluids or oral rehydration is potentially at risk of hyponatraemia. The administration of excess or inappropriate fluid to a sick child may result in serious or life threatening hyponatraemia. There is a particular concern about the use of 0.18% Sodium Chloride in Glucose among children as it has been implicated in cases of hyponatraemia. While it may pose a risk because of the relatively low sodium content no specific fluid is without risk.

4. The Department issued guidance on the prevention of hyponatraemia in children in March 2002. This guidance emphasises that every child receiving intravenous fluids requires a thorough baseline assessment, that fluid requirements must be calculated accurately and fluid balance must be rigorously monitored. Following this advice will prevent children from developing hyponatraemia. CREST has also issued guidance on the management of hyponatraemia in adults (June 2003).

5. The Department is planning to do further work to develop a care pathway for fluid management in children.

6. The Department is presently considering whether any further investigation into the circumstances surrounding the death of Lucy Crawford is required.

Reply prepared by

Ruth Fisher
Eastern Board Unit
Secondary Care Directorate
Tel [redacted]

Andrew Browne
Eastern Board Unit
Secondary Care Directorate
Tel [redacted]

DHSSPS