DETAILED FINDINGS ABOUT THE ASSESSMENT AND INVESTIGATION OF THE CAUSE OF HYPONATRAEMIA

Fluid status assessment

Accurate fluid assessment can be challenging, even for clinicians and specialists experienced in the management of hyponatraemia. The initial assessment of fluid status is usually undertaken by the resident doctors who may have limited experience in this area. In addition, use of point-of-care ultrasound (PoCUS) by appropriately trained healthcare professionals can help to determine a patient's fluid status.[14] Where possible, fluid status should be assessed by clinical assessment and by using dynamic measures (for example response to passive leg raise) and should not rely on static PoCUS assessment of the inferior vena cava (IVC) diameter and/or collapse during inspiration alone. It is important that all healthcare professionals are trained in assessing patient's fluid status.

In this study PoCUS was only used to assess fluid status in three patients as it is an emerging application amongst non-radiologist clinicians, not currently widely used due to the lack of availability of technology and appropriately trained clinicians.[15-19]

The 2014 European guidelines for the assessment and management of hyponatraemia do not include fluid status assessment as a requirement for the assessment of patients with hyponatraemia, reflecting the difficulty of accurately performing this at the bedside.[10] Other guidelines which have conflicting advice and advocate the use of clinical fluid status assessment as part of the assessment of the cause(s) of hyponatraemia.[20-22] However, at the time of some of these guidelines were written, PoCUS assessment for fluid status was not widely available and may reflect why it was not included in them.

In total, 57/248 (23.0%) patients with hyponatraemia did not have a fluid status assessment documented in their medical records during the initial assessment, with no indication that any assessment had been undertaken (unknown in 22). Of those who did have an assessment 11/191 (5.6%) were incomplete or inadequate. Data on the grade of the clinician undertaking the initial fluid assessment was not collected, so it was not possible to determine whether this impacted on the adequacy of the fluid assessment.

Fluid status and sodium balance should be reassessed during the admission, to monitor the effectiveness of any treatment(s) and/or whether the diagnosis for the cause of the hyponatraemia needs to be reconsidered. The frequency of this reassessment needs to be directed by an appropriately trained and experienced senior decision-maker. In addition, this needs to be clearly documented so that those involved with the care of the patient out of hours are aware of the management plan.

There were 85/205 (41.5%) patients admitted with hyponatraemia, and 14/62 (22.6%) who developed postoperative hyponatraemia who did not have evidence of appropriate monitoring (essential for determining the type of hyponatraemia) and documentation of fluid balance (T4.1).

Table 4.1 Appropriate fluid balance monitoring	Emergency		Postoperative	
	Number of patients	%	Number of patients	%
Yes	120	58.5	48	77.4
No	85	41.5	14	22.6
Subtotal	**205**		**62**	
Unknown	65		22	
Total	**270**		**84**	

Reviewer assessment form data

To reduce postoperative hyponatraemia it is essential for surgeons, anaesthetists and specialties involved in the patient's care to address the factors that increase the risk of developing hyponatraemia (e.g. excessive postoperative IV fluid administration). Central to reducing the risk of hyponatraemia is an active, documented fluid balance monitoring plan, as well as supervised and regular monitoring of blood sodium levels postoperatively to detect any developing hyponatraemia. The accuracy of the documentation of fluid balance may depend on how it is recorded.

In 26/156 (16.7%) hospitals both electronic and paper charts were used (T4.2). This practice may increase the risk to patients due to the potential for duplicate recording, which can lead to over- or under-estimating a patient's actual fluid intake and/or output, resulting in inappropriate changes to oral or IV fluids.

Table 4.2 Type of fluid balance charts	Number of hospitals	%
Electronic	76	52.4
Paper	43	29.7
Electronic and paper	26	17.9
Subtotal	**145**	
Unknown	11	
Total	**156**	

Organisational questionnaire data

The data on whether patients reviewed in this study had electronic, paper or a combination of fluid balance charts was not collected to be able to determine their impact on whether fluid balance was monitored appropriately.

Accuracy of completion of fluid balance charts was audited in only 51/83 (61.4%) hospitals, and just 39/83 hospitals reported that any quality improvement projects had been undertaken in the previous five years related to fluid management. Where they had been completed, the improvement themes identified were around resident doctor training and support for the use of intravenous (IV) fluids in both general medicine and surgery, strategies to implement NICE Clinical Guideline CG174 (Intravenous fluid therapy in adults in hospital)[18] and training and compliance with fluid balance documentation.

It was reported from only 26/156 (16.7%) hospitals that there was an IV fluid lead in place as recommended by NICE,[23] and in 63/156 (40.4%) it was unknown, suggesting that the overall proportion of hospitals with an IV fluid lead was much lower.

Where there was an IV fluid lead, the majority (4/17; 9 unknown) did not have formal time in their job plan to undertake this role. Having IV fluid leads in place with appropriate job planned time could improve the documentation of fluid assessment and fluid balance. It is important that the NICE Guidance is implemented and the impact of this on patient care and outcomes are audited.

Other investigations

Despite guidance from the Society of Endocrinology regarding necessary investigations, clinicians often rely on local clinical guidelines to inform their decisions on appropriate investigations to help identify the cause of the hyponatraemia, as advice on what investigations should be undertaken and when often differs between different national and international guidelines.[10,20,21] Currently there are no nationally agreed 'care bundles' that could improve the appropriateness and timeliness of investigations being undertaken in patients with hyponatraemia.

Imaging

The majority (222/270; 82.2%) of patients admitted as an emergency had some form of imaging undertaken during their admission (T4.3) and this altered the management for only 11 patients with emergency admission-related hyponatraemia. Imaging undertaken in patients with hyponatraemia, particularly where it is related to syndrome of inappropriate antidiuretic hormone secretion (SIADH) is to identify an underlying malignancy as the cause.

Table 4.3 Imaging undertaken during admission for emergency admissions hyponatraemia patients	Number of patients	%
CT scan of head	132	50.0
Chest X-ray	120	45.5
CT scan of thorax	35	13.3
CT scan of abdomen/pelvis	35	13.3
Other (specified)	22	8.3
Abdomen ultrasound	15	5.7
MRI of head	13	4.9

Reviewer assessment form data; answers may be multiple; n=270

Case note review suggested that additional imaging should have been undertaken in 21/270 (7.8%) patients. Most commonly a chest X-ray in seven patients; all of whom had a long smoking history and therefore would be at risk of having underlying lung cancer as a cause of their hyponatraemia.

CASE STUDY

An older patient was admitted to hospital with a 7-month history of chronic hyponatraemia. There was a failure to adequately assess the patient's fluid status and review their medications. Despite a long smoking history and unknown cause of chronic hyponatraemia, no imaging was undertaken during the admission. The patient was readmitted to hospital two months later and was found to have lung cancer with liver metastasis.

The reviewers considered that this demonstrated a deficiency in establishing the cause of a history of chronic hyponatraemia. Simple imaging may have diagnosed the underlying cause in patient with a known history of smoking.

Blood tests

Data from the clinical questionnaires showed that liver function tests were most commonly performed (F4.1).

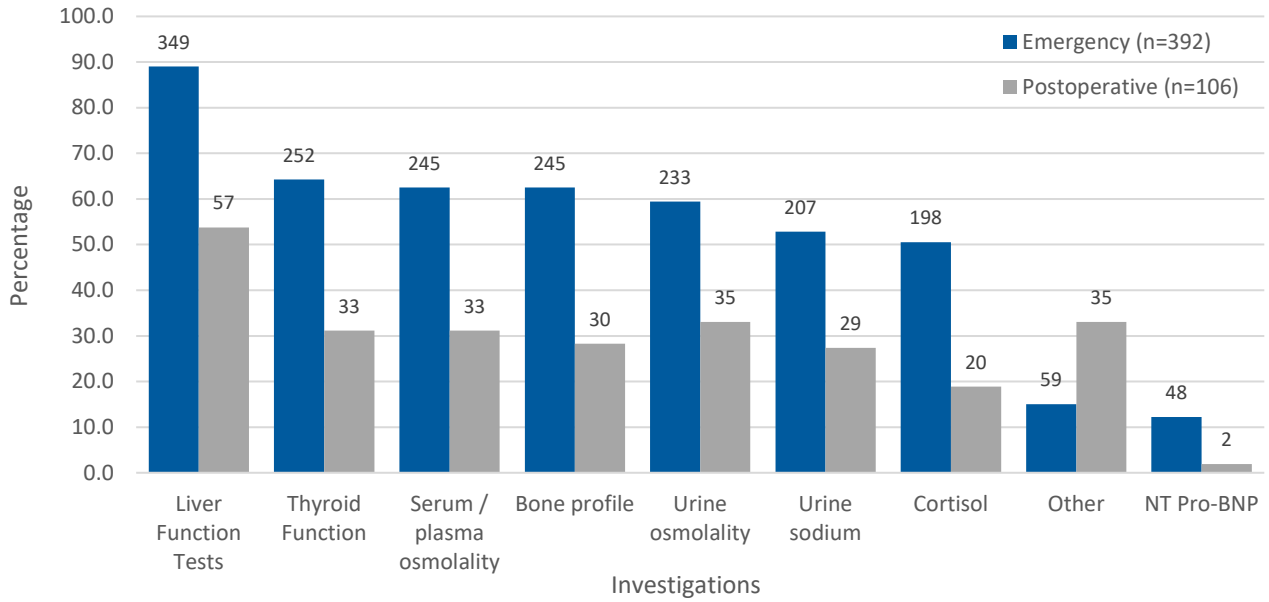

Figure 4.1 Investigations undertaken in emergency and postoperative hyponatraemia patients
Clinician questionnaire data

A higher proportion of postoperative hyponatraemia patients required additional investigations compared to those admitted as an emergency (47/83; 56.6% vs 116/265; 43.8%). Table 4.4 shows the other investigations that were indicated. This difference between emergency admission-related and postoperative hyponatraemia may be due to clinicians incorrectly assuming that managing postoperative hyponatraemia involves only modification of fluid management, rather than considering other potential causes. Specifically, 48/270 (17.8%) emergency admission patients and 33/84 (39.3%) postoperative patients did not have paired (taken at the same time) urine and plasma/serum osmolality measured when it was indicated.

Table 4.4 Additional investigations that were indicated	Emergency admissions		Postoperative hyponatraemia	
	Number of patients	% (n=270)	Number of patients	% (n=84)
Urine sodium	78	28.9	27	32.1
Urine osmolality	72	26.7	36	42.9
Plasma/serum osmolality	48	17.8	33	39.3
Cortisol	38	14.1	11	13.1
Thyroid function	30	11.1	12	14.3
NT pro B-type natriuretic peptide	10	3.7	2	2.4
Other (specified)	10	3.7	1	1.2
Bone profile	9	3.3	8	9.5
Liver function tests	7	2.6	6	7.1

Reviewer assessment form data

Plasma/serum osmolality

The measurement of serum/plasma and/or urine osmolality, along with urine sodium concentrations, are required to assist clinical teams in diagnosing the cause of the hyponatraemia, so results need to be made available as soon as possible. These analyses require laboratory testing and if rapid and reliable point-of-care testing alternatives were available, this could shorten the

timeframe for the results to be available and the timeliness of delivery of the appropriate treatment(s).

The range of urine and serum/plasma osmolality results in emergency admissions are shown in Figures 4.2 and 4.3.

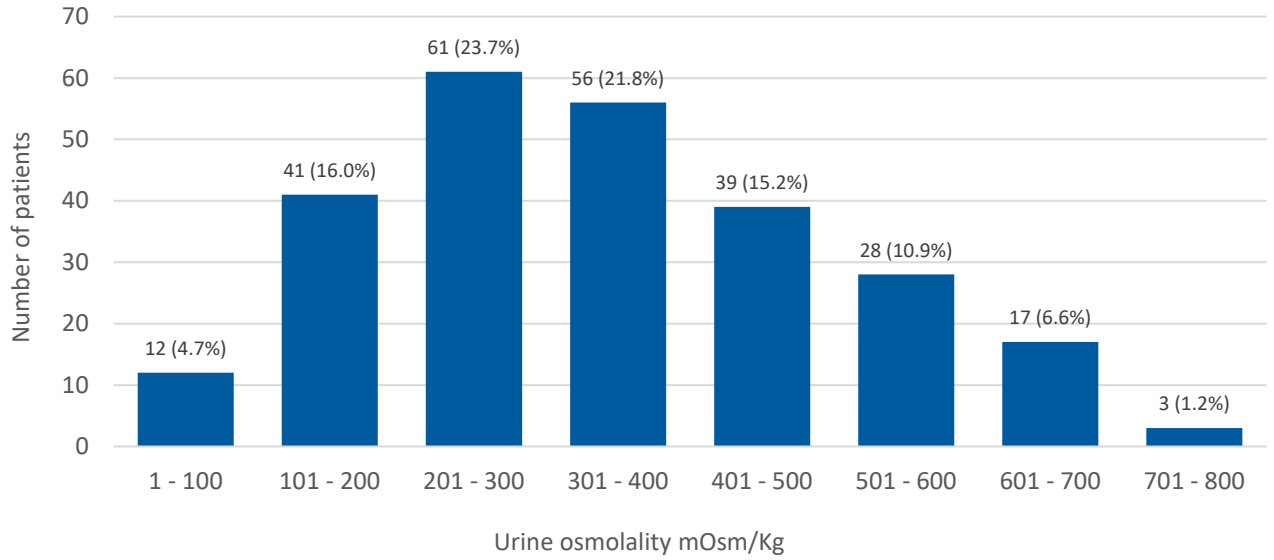

Figure 4.2 Urine osmolality concentrations in emergency admissions
Clinician questionnaire data (n=257)

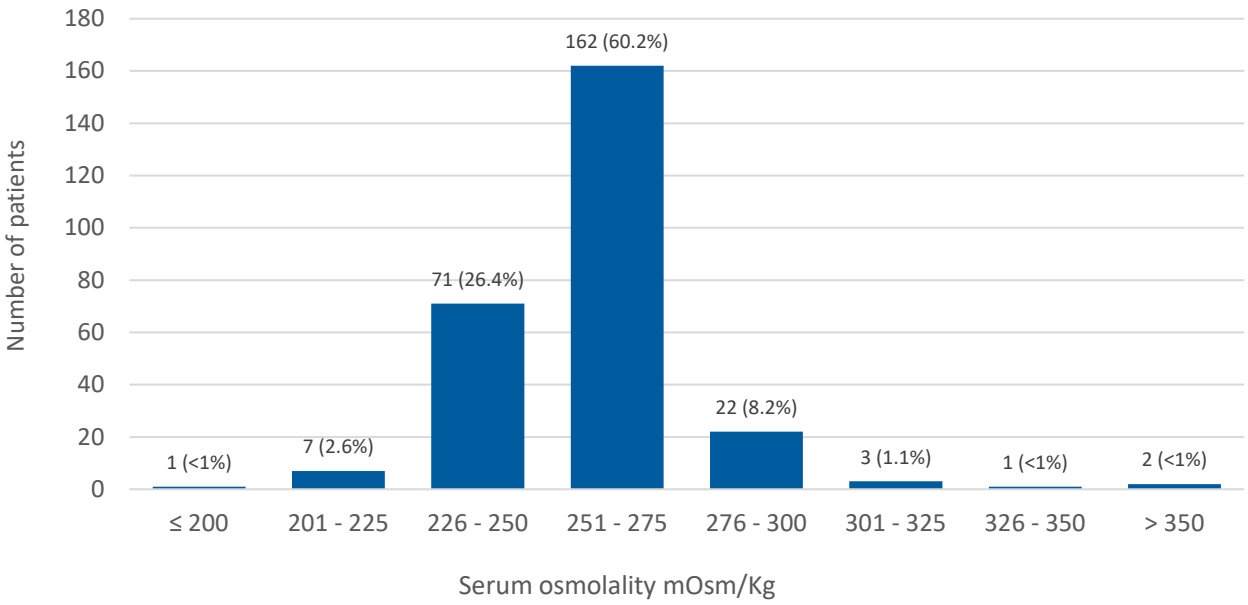

Figure 4.3 Serum/plasma osmolality concentrations in emergency admissions
Clinician questionnaire data (n=269)

There was no strong correlation between the serum and urine osmolality in an individual patient with hyponatraemia (F4.4), which may reflect that urine and serum osmolalities were often not 'paired'; with the urine typically being sent later and so the result may be impacted by any treatment that has been given before the urine is collected. Measurement of urine osmolality remains important as part of work-up to identify the cause of the low sodium in someone with hyponatraemia, however, it is essential that samples are collected at the appropriate time.

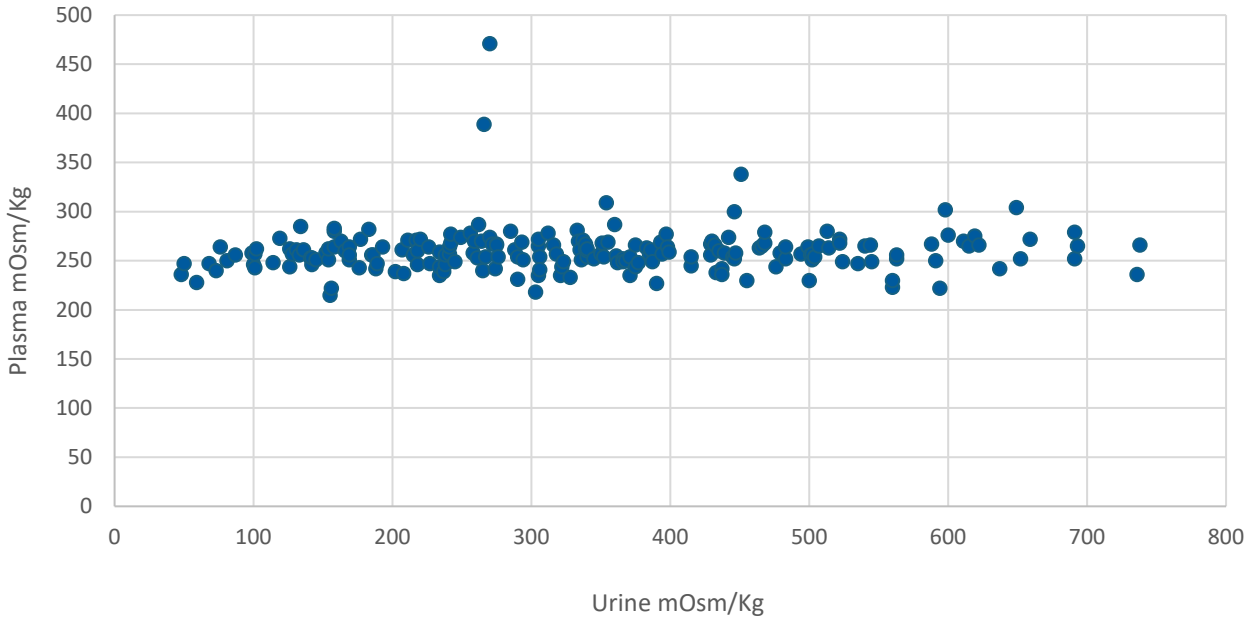

Figure 4.4 Relationship between serum/plasma and urine osmolality

Clinician questionnaire data

The exact times at which samples were collected for plasma/serum osmolality, urine osmolality or urine sodium were not consistently recorded in the medical notes. However, the time the sample was requested by the clinical teams and the time the result was available were reliably available.

There was a delay in obtaining the results from the time of request of a urine osmolality compared to plasma/serum osmolality in emergency admission-related hyponatraemia (F4.5). Obtaining the urine osmolality result rapidly may be helpful in making a diagnosis or determining what treatment is appropriate. For example, a urine osmolality of 100 mOsm/kg or less is indicative of excess fluid intake/administration and treatment with IV fluids would therefore be inappropriate and could worsen hyponatraemia. Of the 12 patients with a urine osmolality of 100 mOsm/kg or less, half were given IV fluids as part of their treatment.

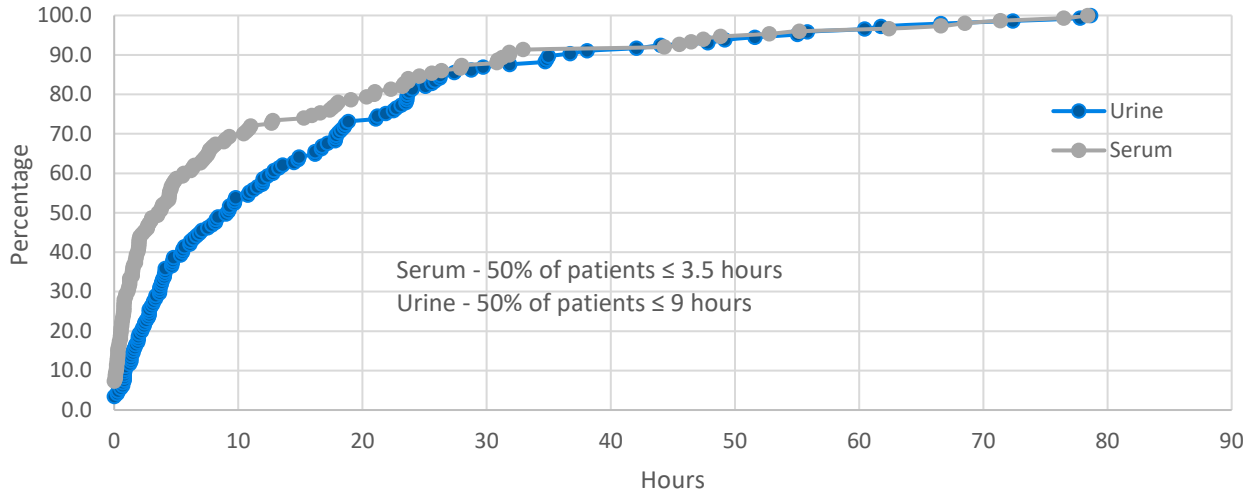

Figure 4.5 Time from osmolality request to result

Clinician questionnaire data

A urine osmolality of greater than 500 mOsm/Kg suggests that treatment with fluid restriction alone will be ineffective. Of the 48 patients with a urine osmolality of 500 mOsm/kg or more, seven were treated with fluid restriction alone. It is worth noting that some of the drugs that can be associated with hyponatraemia may also impact on the validity of the urine osmolality and urine sodium results. Therefore, it is important that there is appropriate specialist advice available to help with determining if any medication a patient is taking may impact on the validity of these results.

This delay in reporting urinary osmolality compared to serum, was multifactorial. Reasons included:

i) A delay in collection of the sample, as it is easier to obtain a blood sample than a urine sample (only approximately 50% of plasma/serum and urine samples were collected within an hour of each other, and nearly a fifth of samples were collected more than 12 hours apart (T4.5);
ii) analysis and reporting of blood samples in the laboratory is usually fully automated whereas the analysis of urine samples may be processed through different pathways; and
iii) measurement of samples for osmolality are manual processes.

This means that overnight when there is reduced staff capacity in the laboratory, osmolality tests may not be prioritised as they divert the limited staff from overseeing and doing a high volume of other tests.

It may not necessarily be appropriate therefore to have all osmolality results available rapidly out of hours due to the pressures on laboratories and laboratory staff. However, the result should be available by first thing the next morning so that it is available to assist decision-making on the morning post-take review or other ward rounds. In some circumstances rapid osmolality results are important – these may relate to the severity of the hyponatraemia, certain patient populations (e.g. very young and older people) or those with suspected polydipsia (as their sodium will rapidly correct with appropriate fluid restriction).

Table 4.5 Time between collection of plasma/serum and urine samples	Number of patients	%
0	58	38.2
>0 – 1	19	12.5
>1 – 2	6	3.9
>2 – 3	7	4.6
>3 – 4	6	3.9
>4 – 5	7	4.6
>5 – 6	2	1.3
>6 – 7	6	3.9
>7 – 8	6	3.9
>9 – 10	2	1.3
>10 – 11	2	1.3
>11 – 12	3	2.0
>12 – 24	15	9.9
> 24	13	8.6
Total	**152**	

Clinician questionnaire data

Most hospitals had agreed turnaround times for urine osmolality (93/114; 81.6%), urine sodium (95/118; (80.5%) and serum/plasma osmolality (99/118; 83.9%) (T4.6). However, the reported service level agreements for these turnaround times in a high proportion of hospitals exceed what the reviewers considered to be clinically acceptable (T4.7). In addition, regular audit of the turnaround times for these tests occurred in only 30/73 (41.1%) hospitals where it was known (T4.8).

Table 4.6 Agreed turnaround times	Urine osmolality		Urine sodium		Serum osmolality	
	Number of hospitals	%	Number of hospitals	%	Number of hospitals	%
Yes	93	81.6	95	80.5	99	83.9
No	21	18.4	23	19.5	19	16.1
Subtotal	**114**		**118**		**118**	
Unknown	42		38		38	
Total	**156**		**156**		**156**	

Organisational questionnaire data

Table 4.7 Reported turnaround times for urine and serum osmolalities and urine sodium			
Time (hours)	Urine osmolality	Urine sodium	Serum osmolality
1	23	21	24
2	4	1	6
3	2	2	2
4	10	14	12
6	2	6	0
8	1	0	2
12	4	3	4
24	46	46	48
48	1	2	1

Organisational questionnaire data

Table 4.8 Auditing of turnaround times for urine and plasma/serum osmolalities and urine sodium	Number of hospitals	%
Yes	30	41.1
No	43	58.9
Subtotal	**73**	
Unknown	26	
Total	**99**	

Organisational questionnaire data

In addition to considering any potential delays in the analysing and reporting of investigations, there may be delays in the correct response and action by clinical staff to a blood sodium level that is abnormal. Any delays may be greater out of hours, especially overnight, when the abnormal results are being reviewed and actioned by resident doctors and specialist support may not be readily available.

Serum cortisol

Measurement of serum cortisol in patients with hyponatraemia should be undertaken if the suspected cause of the hyponatraemia is thought to be adrenal insufficiency. Ideally, the serum cortisol should be measured between 8:00am and 9:00am to facilitate the interpretation of the result, as there is variation in cortisol with higher levels in the morning and lower levels in the evening. Although, outside of these hours a low serum cortisol in patients with severe hyponatraemia may alert clinicians to suspect adrenal insufficiency. Cortisol testing should not be routinely undertaken in patients on external corticosteroids equivalent to more than 5mg prednisolone per day. If measurement of cortisol is required, then the steroids should be stopped, and specialist advice may be required to determine the time after stopping steroids before the cortisol can be measured. As shown previously in Table 4.9, there were patients who should have had a cortisol measurement and in patients with suspected SIADH a cortisol level is an essential investigation and failure to undertake it is an 'incomplete work-up'.

Cortisol levels between 8:00am and 9:00am of less than 150 nmol/L indicate possible adrenal insufficiency while levels above 300 nmol/L suggest it is unlikely. Levels between 150 and 300 nmol/L require further investigation, potentially with a short Synacthen test.[24]

Results of cortisol testing undertaken at other times or in patients being treated with corticosteroids are more difficult to interpret. The range of times that serum cortisol was measured is shown in Figure 4.6, and the range of cortisol results split between those undertaken between 8:00am and 10:00am and at other times is shown in Figure 4.7. Only 25/150 (16.7%) patients had cortisol samples collected between 8:00am and 9:00am. The presence of an abnormal cortisol outside of 8:00am and 10:00am, should lead clinicians to repeat the test utilising additional resources.

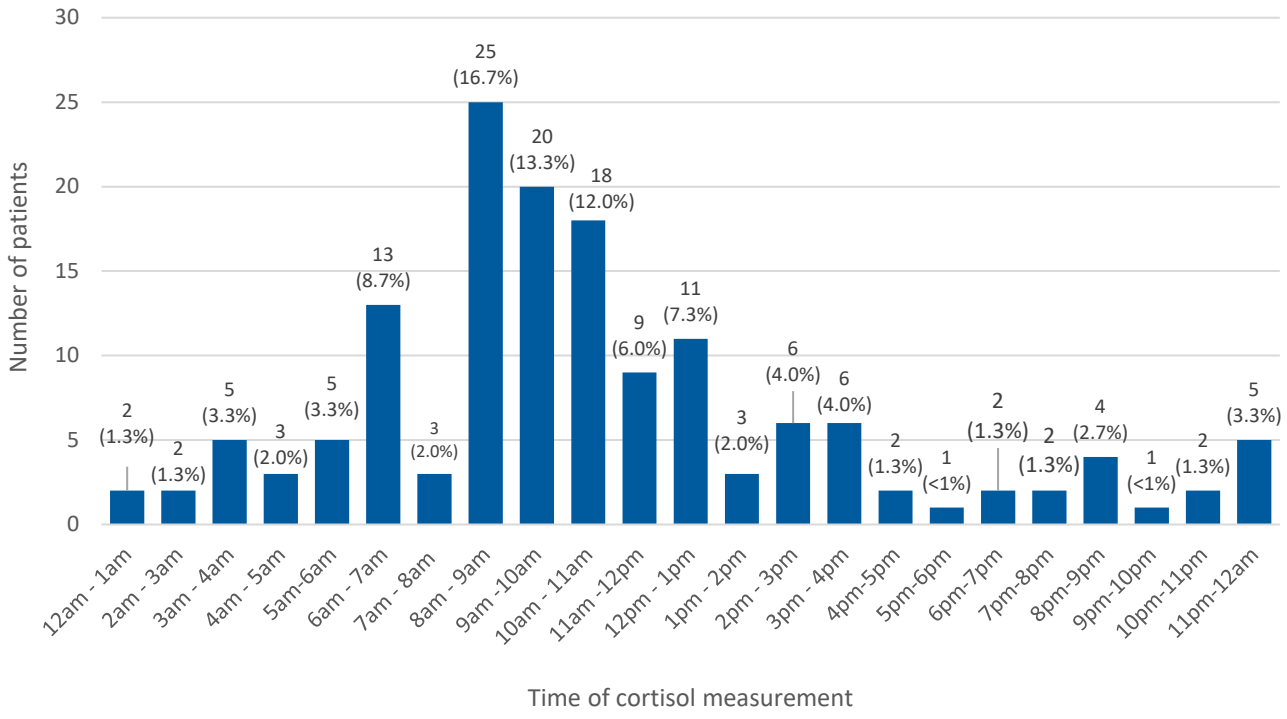

Figure 4.6 Times cortisol measurements were undertaken

Clinician questionnaire data (n=150)

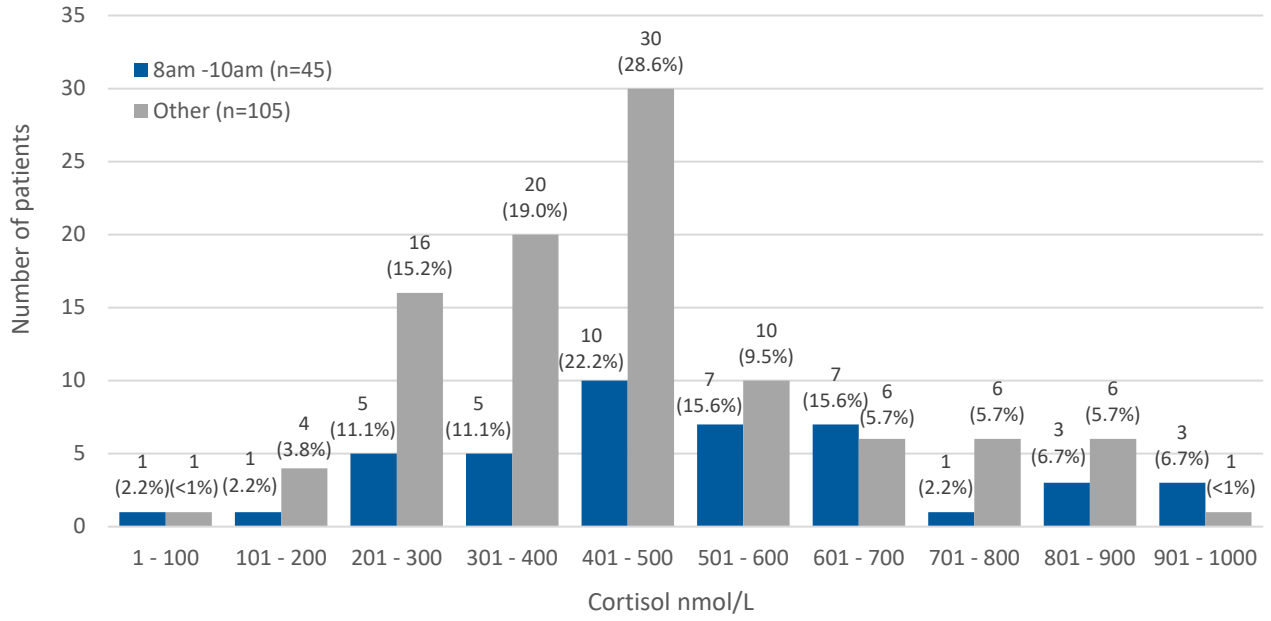

Figure 4.7 Range of cortisol results based on time test undertaken

Clinician questionnaire data

Blood glucose

The range of blood glucose concentrations at the time of the lowest sodium result is shown in Figure 4.8. The lowest sodium results in those with a blood glucose of greater than 10 mmol/L, which is likely to have an impact on the blood sodium analysis and reporting were 111 – 115 mmol/L in four patients; 116 – 120 mmol/L in eight patients; 121 – 125 mmol/L in six patients; and 126 – 130 mmol/l in three patients. This distribution of blood sodium concentrations was broadly similar to those patients with a blood glucose of 10 mmol/L or less.

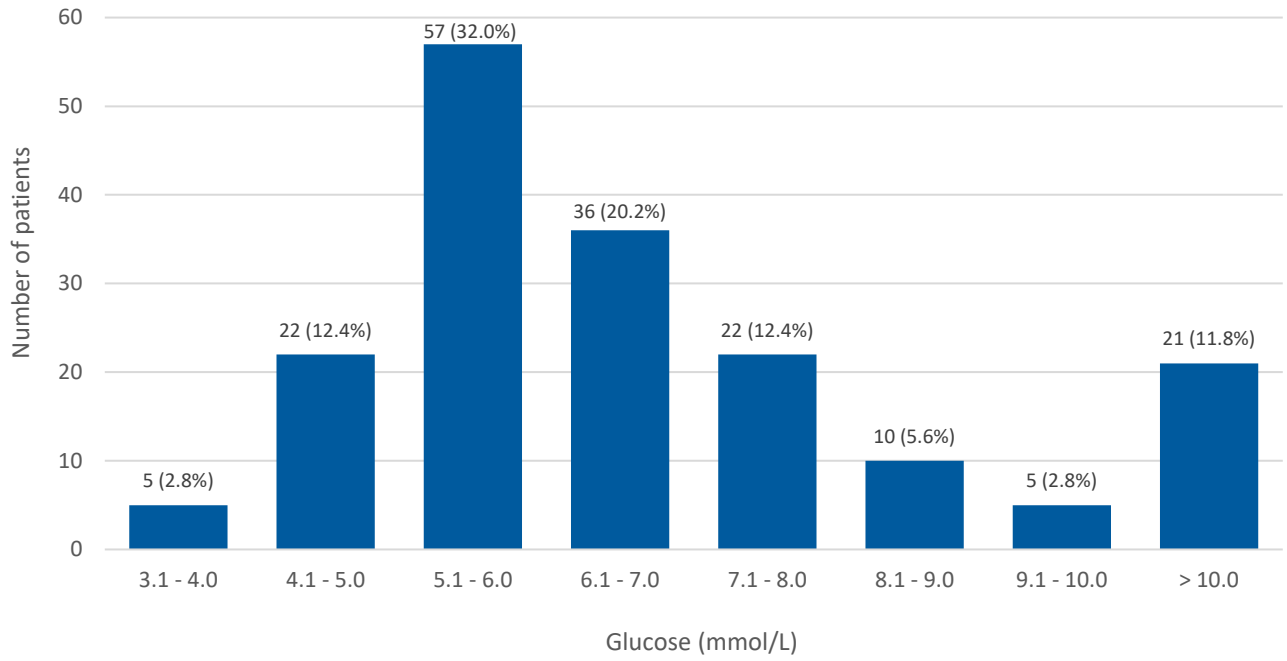

Figure 4.8 Range of glucose measurements at the time of the lowest sodium

Clinician questionnaire data (n=178)

Duration and severity of emergency admission-related hyponatraemia

Acute hyponatraemia is defined as occurring in the previous 48 hours. Where the time of onset could be determined, 184/306 (60.1%) patients admitted as an emergency had acute hyponatraemia (T4.9). This differed from routine clinical practice, where chronic hyponatraemia is much more frequent than acute hyponatraemia.[10,25] Our sampling methodology which aimed to review more patients with moderate or severe hyponatraemia, may have biased our sampling towards those with acute hyponatraemia.

Table 4.9 Acute or chronic hyponatraemia – emergency admissions	Number of patients	%
Acute	184	60.1
Chronic	122	39.9
Subtotal	**306**	
Unknown	86	
Total	**392**	

Clinician questionnaire data

When patients present with hyponatraemia, they may have a previous results of blood sodium levels, but this is often not within the previous 48 hours. Due to the potential risks associated with rapid over-correction of hyponatraemia in patients with longer-term hyponatraemia (where compensation for the hyponatraemia has occurred), they are typically treated as having 'chronic hyponatraemia'.[25]

For 55 patients the clinician who treated the patient was unable to determine retrospectively from the notes whether the hyponatraemia was acute or chronic, and it is possible that the majority may have chronic hyponatraemia. However, the uncertainty noted by the reviewers suggests poor documentation of the timeframe of the hyponatraemia at the time of admission, although where no previous blood results are available it may not be possible to determine the chronicity.

The severity of hyponatraemia was determined by the local treating clinician. However, as the severity gradings were not defined for the clinicians, their assessment could have been made based on biochemical severity, clinical severity or a combination of both (T4.10).

Table 4.10 Severity of hyponatraemia – emergency admissions	Number of patients	%
Mild	75	21.3
Moderate	118	33.5
Severe	159	45.2
Subtotal	**352**	
Unknown	40	
Total	**392**	

Clinician questionnaire data

A greater proportion of acute hyponatraemia emergency presentations were classified by the treating clinician as 'severe' compared to chronic hyponatraemia presentations (91/181; 50.3% compared with 47/117; 38.5% respectively) (F4.9). While we did not provide guidance on how to

grade the severity of hyponatraemia it should be noted that severe biochemical hyponatraemia and symptomatic hyponatraemia can cause confusion for clinicians.

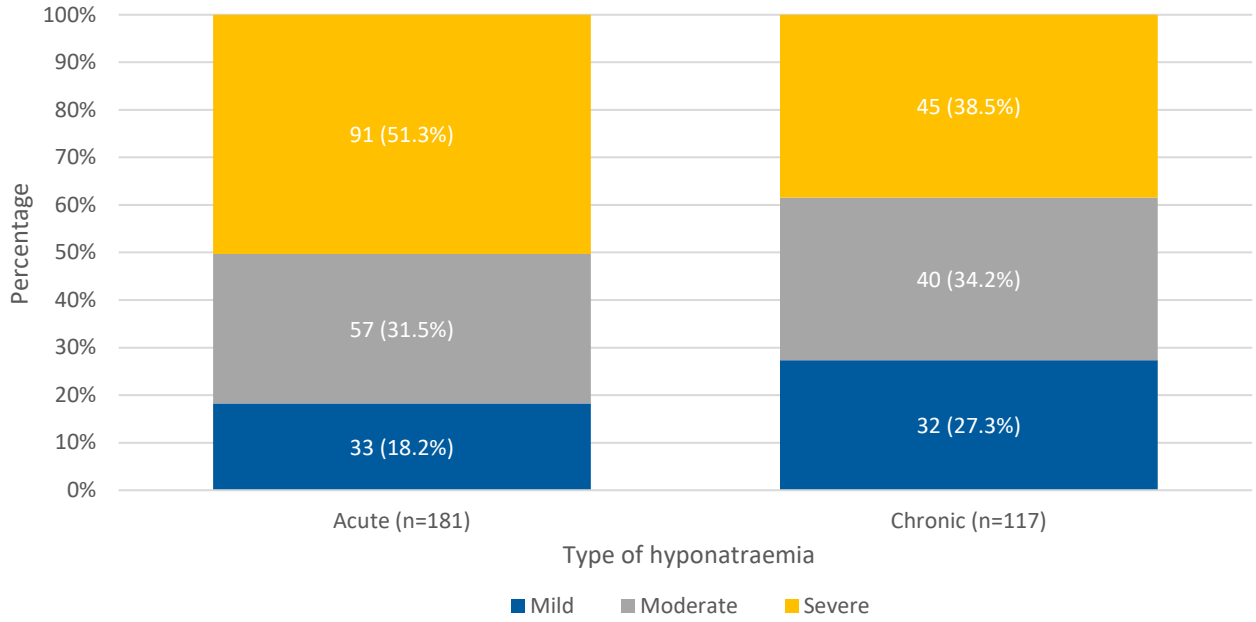

Figure 4.9 Comparison of hyponatraemia severity in acute or chronic hyponatraemia cases
Clinician questionnaire data

Classification and the causes of hyponatraemia for patients admitted as an emergency

Hyponatraemia can be classified as hypotonic, hypertonic or pseudo-hyponatraemia (T4.11 and F4.10).

Table 4.11 Type of hyponatraemia as defined by the local treating clinician		Number of patients	%
Hypotonic	Euvolaemic hyponatraemia: Total body water increases without causing oedema (swelling); total body sodium remains unchanged.	132	42.4
	Hypovolaemic hyponatraemia: Total body water decreases, but total body sodium decreases even more.	129	41.4
	Hypervolaemic (volume overload) hyponatraemia: Both total body water and sodium increase, with a significant rise in total body water causing oedema.	46	14.8
Hypertonic	Hypertonic (hyperosmolar) hyponatraemia: an increase in osmotic pressure in the extracellular compartment, causing water to move from the intracellular to the extracellular compartment thereby diluting extracellular sodium. A common cause is significant hyperglycaemia.	3	1.0
Pseudo-hyponatraemia	Can be seen in patients with very high serum lipids or proteins, which result in a false reduction in blood sodium levels during analysis.	1	0.3
	Subtotal	**311**	
	Unknown	81	
	Total	**392**	

Reviewer assessment form data

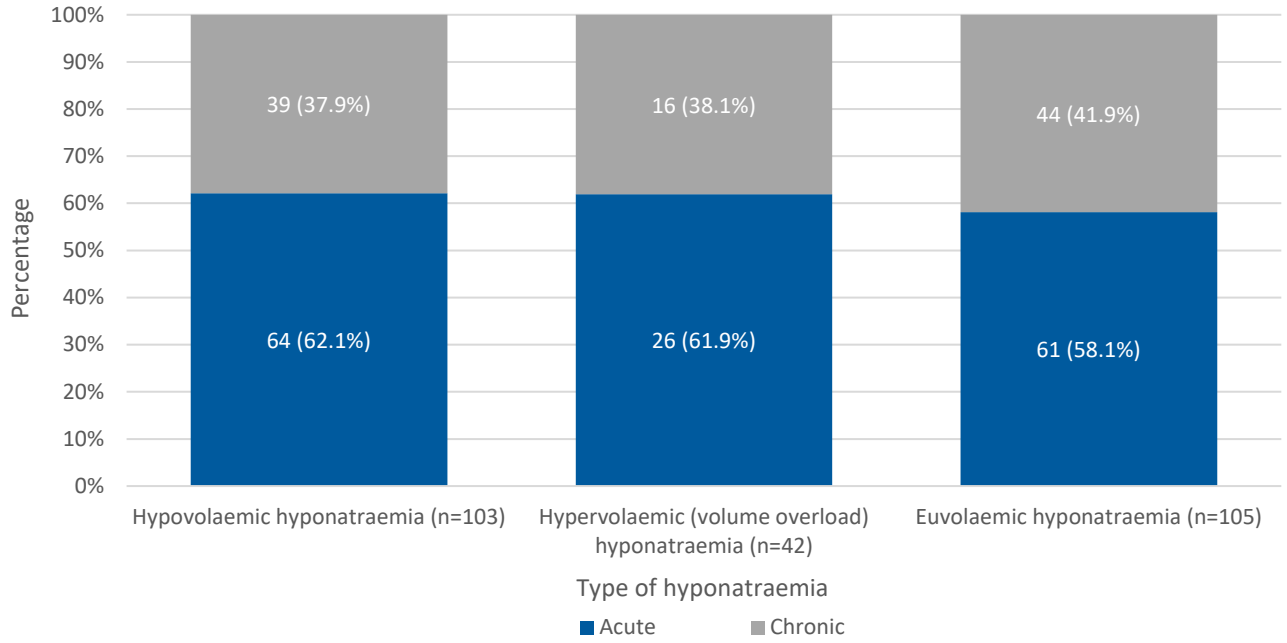

Figure 4.10 Comparison of frequency of acute compared to chronic hyponatraemia in the three most common types of hyponatraemia

Clinician questionnaire data

Severe hyponatraemia was more common in patients with hypotonic (true) hyponatraemia (60/118; 50.8%) and hypervolaemic (volume overload) hyponatraemia (23/45; 51.1%) than in those with euvolaemic hyponatraemia (52/129; 40.3%) (F4.11).

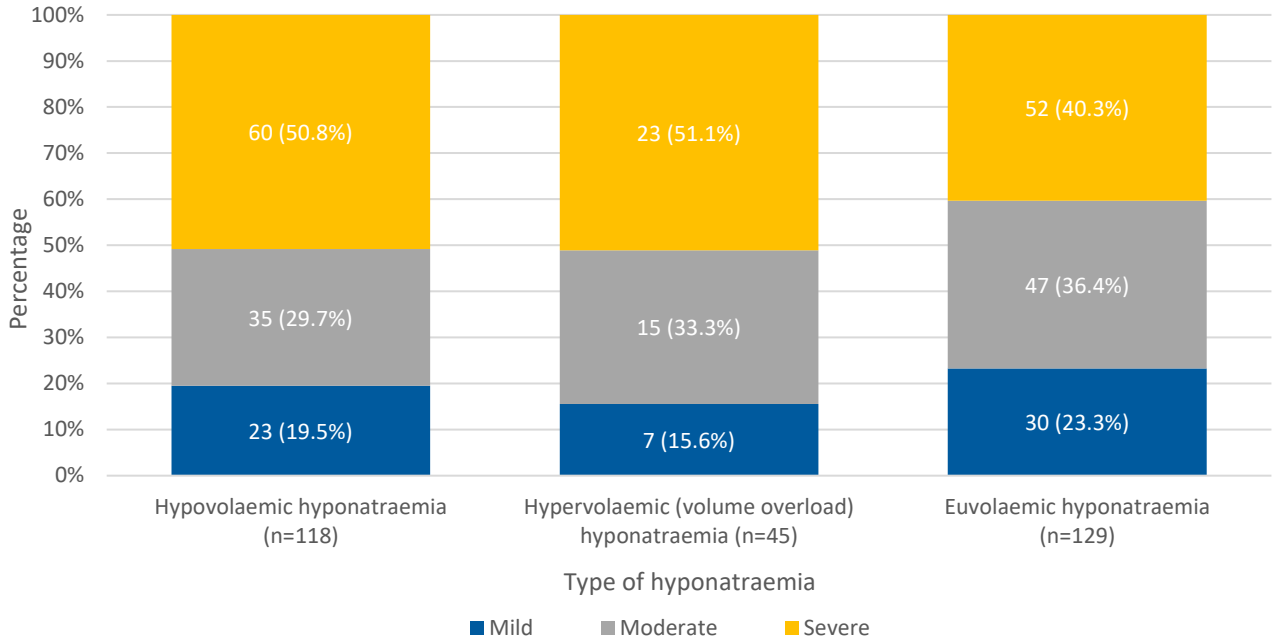

Figure 4.11 Comparison of degree of severity of the hyponatraemia for the three most common types of hyponatraemia

Clinician questionnaire data

Hyponatraemia is a descriptive term indicating that the patient has a low blood sodium concentration; it does not provide an indication of the actual cause of the sodium abnormality. Of note, 22/251 (8.8%) of emergency admission patients with a low blood sodium level had only

'hyponatraemia' listed a cause of the low blood sodium in their notes, without any further clarification on the potential cause(s) for this (T4.12). In addition, 66/251 (26.3%) had an initial or working diagnosis of SIADH, a common cause of euvolaemic hyponatraemia. It should be noted that there are many different causes of SIADH and once this working diagnosis is confirmed then further investigation maybe required to determine the cause of the SIADH.

Table 4.12 Most common working diagnosis of the cause of hyponatraemia that was documented in the patient's medical records	Number of patients
Syndrome of inappropriate antidiuretic hormone secretion (SIADH)	66
Medication-related hyponatraemia	43
Diarrhoea and vomiting	41
Alcohol abuse	29
Just recorded as hyponatraemia	22
Acute or chronic heart failure	20
Malnutrition/dehydration	20
Acute cerebral event/ head injury	15
Renal disease	12
Beer potomania	11
Excess fluid intake	11
Infection	9
Ascites	8
Dementia/acute confusional state	6
Hyperglycaemia	5
Epilepsy	5
Adrenal insufficiency	5

Reviewer assessment form data; n=251 (answers may be multiple)

The reviewers agreed with the working diagnosis in 200/270 (74.1%) cases reviewed. In the cases where the reviewer did not agree, their reason was either that the clinicians had only documented 'low sodium' or 'hyponatraemia' as the diagnosis, or there were insufficient investigations undertaken for the reviewer to be able to support the clinical teams working diagnosis.

Osmotic demyelination syndrome

Rapid increases in blood sodium levels in patients with chronic hyponatraemia can result in a rare condition called osmotic demyelination syndrome (ODS). These rapid changes in sodium levels lead to changes in brain fluid balance, which results in damage to the myelin covering brain nerve cells.

Patients with ODS can develop a range of symptoms including confusion, delirium, hallucinations, tremor, poor balance, drowsiness, lethargy, slurred speech (dysarthria) and generalised or focal weakness. There is no specific treatment for ODS when it develops, and the focus is to prevent its occurrence by limiting the rate of blood sodium rise when treating patients with suspected or known chronic hyponatraemia. ODS typically only occurs in patients who have one or more other risk factor, in addition to the presence of hyponatraemia. The greatest risk factor for ODS is having a blood sodium level of less than 120 mmol/L.

Just under half of those patients admitted with hyponatraemia had one or more other risk factor(s) for the development of ODS (109/270; 40.4%) as shown in table 4.13.

Table 4.13 Risk factors present for osmotic demyelination syndrome	Number of patients	%
Alcohol excess	66	24.4
Smoking history	36	13.3
Nutrition	38	14.1

Reviewer assessment form data. Answers may be multiple; n=270

Despite the relatively high proportion of patients with one or more risk factor and 219/392 (55.9%) of those with emergency admission-related hyponatraemia having a lowest blood sodium level of less than 120 mmol/L, none of the patients reviewed by either the clinicians at the hospital or the case reviewers developed ODS during their admission. This may reflect that there was a high proportion of patients in our study with acute hyponatraemia, when more rapid increases in blood sodium can be tolerated with a lower risk of developing ODS.

Hyponatraemic encephalopathy

There were 63/270 (23.3%) patients with hyponatraemia who had a diagnosis of hyponatraemic encephalopathy documented in their notes. On review, a further 43 patients had clinical features consistent with encephalopathy that not been recognised/documented by the treating clinical team, and one patient who they thought did not have despite it being recorded.

The reviewers determined that 105/260 (39.5%) patients should have had a diagnosis of hyponatraemic encephalopathy based on their symptoms (unknown for 10) (T4.14).

Table 4.14 Symptoms present consistent with hyponatraemic encephalopathy diagnosis	Number of patients
Confusion/headaches/visual disturbance	24
Seizures	23
Nausea/vomiting	22
Fatigue	16
Attention deficit	15
Gait problems	13
Falls	11
Loss of consciousness	11

Reviewer assessment form data

Of the 63 patients who the treating clinicians documented as having a diagnosis of hyponatraemic encephalopathy, 38 were treated with hypertonic saline. In the additional 43 patients the reviewers believed should have been diagnosed with hyponatraemic encephalopathy, 11 were given hypertonic saline, suggesting that some patients were treated without the treating clinical team documenting that the patient had encephalopathy related to the hyponatraemia. Uncertainty around using hypertonic saline, even when it is thought to be required, may result in clinicians deferring using it until more senior input is available, which can further contribute to delays and risk of complications developing.