# A Balanced Solution

A review of the quality of the care in hospital provided to adults with abnormal levels of blood sodium

**EXTENDED REPORT**

NCEPOD

Improving the quality of healthcare

# A BALANCED SOLUTION

*A review of the quality of care provided to adults in hospital identified as having hyponatraemia (low blood sodium levels) or hypernatraemia (high blood sodium levels)*

A report published by the National Confidential Enquiry into Patient Outcome and Death (2025)

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Cohort: *All patients aged 18 or over who were admitted to hospital between 1st October 2023 and 31st December 2023 and identified as having hypernatraemia or hyponatraemia during their admission by retrospective ICD10 coding*.

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# NOTES FOR READERS

**Normal** blood sodium levels range from **135-145 mmol/L**
**Hypo**natraemia is the term used to describe **low** blood sodium levels (<135 mmol/L)
**Hyper**natraemia is the term used to describe **high** blood sodium levels (>145 mmol/L)
This report relates to adults (18 years and over) only

# LINKS TO ADDITIONAL REPORT SECTIONS


THE GLOSSARY
THE REFERENCES
THE ACKNOWLEDGMENTS
USEFUL RESOURCES ON THIS TOPIC
QI TOOLS FOR THIS STUDY

# INTRODUCTION FROM THE CHAIR

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The measurement of blood sodium levels is one of the most commonly requested pathology tests in the UK and levels outside the reference range are encountered regularly by a wide range of primary and secondary care specialties. The detection of an abnormal blood sodium level is often an incidental finding and may or may not be related to the condition being investigated. Hyponatraemia and hypernatraemia are not diagnoses on their own, and it is vital that the underlying cause of the abnormality is identified and treated. Hyponatraemia in particular can be more challenging as it has several causes, each requiring a different set of investigations and treatment.

The majority of blood sodium measurements are carried out in biochemistry laboratories, but a significant number are now done using point-of-care testing equipment, such as blood gas analysers, close to the patient's bedside. While this has many advantages, it does mean that the results of tests may not always be entered on the patient's laboratory record or filed in the patient's notes. Whichever technique is used, it is important that all results are recorded in the patient's record, and the same method is used when monitoring blood sodium levels, so sequential results are comparable. Thorough assessment and monitoring of patients' fluid status is not easy and may not be done well but is important to help guide both diagnosis and treatment. Accurate fluid balance records should be kept and reviewed regularly. The use of a care bundle, which is easily deliverable in different systems, could help ensure that appropriate ancillary tests are carried out in a timely manner.

There are many causes of abnormal blood sodium levels but one of the most easily identified and corrected is the effect of starting or changing the dose of a medication. An early, thorough medication review should be carried out for all patients, with changes made only after risk assessment and with specialist input if appropriate. It is particularly important that any changes to medications are communicated promptly to the patient's GP and other clinicians, with a rationale for the change, so that appropriate care continues after discharge and in the community.

The diagnosis of the cause of abnormal blood sodium level is not always straight forward, so specialist advice should be available and sought to help inform investigation and treatment decisions. Endocrinologists and clinical biochemists, in particular, have an important role to play in supporting clinicians to investigate and treat electrolyte disturbances such as abnormal blood sodium levels and undertake follow-up after discharge if required.

As always, my grateful thanks go to everyone involved in developing and carrying out this study and those involved in writing the report and its recommendations.

Dr Suzy Lishman CBE, NCEPOD Chair

# TO IMPROVE THE CARE PROVIDED TO PEOPLE WITH ABNORMAL BLOOD SODIUM LEVELS…

## Develop care bundles and training to reduce variation in the assessment and management of abnormal blood sodium levels.

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| **Abnormal blood sodium levels were not always acted on as they should have been, leading to under investigation, inappropriate treatments and poor overall management.** | 116/265 (43.8%) emergency admission hyponatraemia patients should have had further investigations. | Training on hyponatraemia was provided to foundation doctors in most hospitals, but less so for other grades and specialties (37/100; 37.0%). Training on hypernatraemia was only provided in 14/99 (14.1%) hospitals. |

## Improve the clinical assessment of fluid status in all patients.

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| **Patients do not have consistent assessment of their fluid status and monitoring and/or recording of their fluid balance.** | 57/270 (21.1%) patients with hyponatraemia did not have a fluid status assessment documented in their notes during their initial assessment. Furthermore, monitoring and documentation of fluid balance was inadequate in 85/205 (41.5%). | The accuracy of completion of fluid balance charts was only audited in 51/83 (61.4%) hospitals. In 73 hospitals this could not be answered. |

## Integrate test results into patient electronic records to help identify trends in blood sodium levels.

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| **Frequently, results from point-of-care testing are not directly linked into the hospital laboratory electronic reporting system leading to delays in treatment.** | Initial blood sodium results in patients with hyponatraemia (357/386; 92.5%) were from laboratory testing rather than point-of-care testing. | There were delays in the treatment of emergency admission hyponatraemia (64/255; 25.1%) and 17/64 (26.6%) were attributed to the impact of out-of-hours care with reduced staffing. |

## Standardise the use and the dosing of hypertonic saline solution.

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| **Clinical staff are unsure when to use hypertonic saline and the dosage needed. This is hindered further by the variability in the concentrations stocked across all hospitals.** | 55/354 (15.5%) patients received hypertonic saline as part of their treatment. For seven patients, this was not indicated. | Of the 28/55 patients administered with hypertonic saline in an emergency department, only 11 were admitted to a critical care unit. |

## Document and communicate all medication changes to all healthcare providers and patients.

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| **Medication changes were not always communicated which could lead to patients restarting medications that had caused their abnormal blood sodium.** | 225/270 (83.3%) patients admitted on an emergency basis with hyponatraemia were taking one or more medication that could have contributed to their hyponatraemia. | 'Communication' to the GP that a medicine had been stopped, was commonly absent from the patient's medication list at discharge. |

Blood sodium levels is one of the most requested pathology tests and levels outside the reference range are encountered regularly by a wide range of primary and secondary care specialties. The detection of an abnormal blood sodium is often an incidental finding and may or may not be related to the condition being investigated. Hyponatraemia and hypernatraemia are not diagnoses on their own, and it is vital that the underlying cause of the abnormality is identified and treated.

The care of patients in hospital between 1st Oct 2023 and 31st Dec 2023 with a diagnosis code of hyponatraemia or hypernatraemia was reviewed using 428 sets of case notes, 650 clinician questionnaires and 156 organisational questionnaires.

# RECOMMENDATIONS

These recommendations have been formed by a consensus exercise involving all those listed in the acknowledgements. The recommendations have been independently edited by medical editors experienced in developing recommendations for healthcare audiences to act on.

The recommendations in this report support those made previously by other organisations, and for added value should be read alongside:

- Society for Endocrinology: Emergency management of severe and moderately severely symptomatic hyponatraemia in adult patients
- NICE Clinical Knowledge Summary: Hyponatraemia
- European Society of Endocrinology Clinical guideline for the management of hyponatraemia
- NICE Clinical Guideline CG174: Intravenous fluid therapy in adults in hospital

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| 1 | Implement processes to reduce variation in the assessment and management of abnormal blood sodium levels.*<br>• Develop national care bundles.<br>• Develop training for all healthcare professionals to be able to assess and treat patients with abnormal blood sodium levels and recognise when to escalate to specialists.<br>**Promote existing information on hyponatraemia from the Society for Endocrinology and develop it into the care bundle* |
| FOR ACTION BY | Department of Health and Social Care/NHS England, Welsh NHS, Health Department of Northern Ireland, Government of Jersey |
| RATIONALE FOR THE RECOMMENDATION | The care and outcome of patients with an abnormal blood sodium may be improved through timely and appropriate identification and investigation. While there is guidance from the Society of Endocrinology and others on what investigations to do and how to manage hyponatraemia (low sodium), delays and omissions in the appropriate investigations being undertaken for patients were common in this study. Furthermore, patients admitted with conditions that might cause abnormal blood sodium levels should raise a concern and be investigated.<br>There are currently no national guidelines for managing hypernatraemia (high sodium). |
| ASSOCIATED GUIDANCE | Recognition of the patient presenting with severe and moderately severe, symptomatic hyponatraemia<br>Society for Endocrinology: Emergency management of severe and moderately severely symptomatic hyponatraemia in adult patients<br>NICE: Hyponatraemia scenario management |

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| | European Society of Endocrinology Clinical guideline for the management of hyponatraemia |
| ADDITIONAL STAKEHOLDERS | Society for Endocrinology, Royal Colleges of Physicians, Royal College of Emergency Medicine, Royal College of Pathologists, Society for Acute Medicine, Royal College of Surgeons, Association of Surgeons, Royal College of Nursing, Faculty for Intensive Care Medicine, Intensive Care Society, Association for Laboratory Medicine, Royal Pharmaceutical Society, UK Kidney Association |
| IMPLEMENTATION SUGGESTIONS | ▪ Care bundles for **acute kidney injury**, **falls** and **sepsis** have been shown to improve patient care by providing clinicians with clear information on what investigations and treatment need to be undertaken and the timeframe in which this should happen. A clear definition is needed on which staff groups deliver which component of these care bundles, along with 'tick boxes' to indicate completion to improve compliance. In addition, the senior responsible clinician for ensuring delivery of the care bundle should be clearly indicated<br>▪ There would need to be appropriate guidance on determining which investigation(s) should be done to prevent over-investigation<br>▪ These items, including documentation of the time it was done could be considered as part of the care bundle<br>  ▪ Fluid assessment<br>  ▪ Initiation of fluid balance monitoring<br>  ▪ Medication review<br>  ▪ Urine/plasma osmolality<br>  ▪ Urine sodium<br>  ▪ 08:00-09:00 cortisol and other tests as needed such as liver function, thyroid function and NTproBNP<br>▪ Local service level agreements should be put in place specifying turnaround times for urgent investigations and these should be regularly audited<br>▪ Development of eLearning training packages for non-specialist healthcare professionals to assess and treat patients with abnormal blood sodium levels, including 'red flags' for escalation to specialists. |

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| 2 | Develop clear standards and tools for the assessment and recording of fluid status in all patients with abnormal blood sodium levels including, when appropriate, the use of point-of-care ultrasound.*<br>**Point-of-care ultrasound is relatively new so should be considered as further research in its use is published and standards are developed* |

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| FOR ACTION BY | Department of Health and Social Care/NHS England, Welsh NHS, Health Department of Northern Ireland, Government of Jersey |
| RATIONALE FOR THE RECOMMENDATION | Initial and subsequent clinical assessment of fluid status, along with ongoing monitoring of fluid balance after admission were not undertaken well or documented clearly. These assessments should be part of routine clinical care provided by all relevant healthcare professionals. Failure to do these can impact on the appropriateness of the hyponatraemia and hypernatraemia treatment.<br>In addition, there is now interest in the use of point-of-care ultrasound (PoCUS) alongside clinical assessment to improve the quality of the fluid status assessment. Currently this is not widely used due to the lack of availability of technology and appropriately trained clinicians and the most appropriate way to use PoCUS has not been agreed amongst specialists. |
| ASSOCIATED GUIDANCE | NICE Clinical Knowledge Summary: Hyponatraemia scenario management<br>British Medical Ultrasound Society: Focused and Point-of-Care Ultrasound |
| ADDITIONAL STAKEHOLDERS | Royal College of Nursing, Royal Colleges of Physicians, Royal College of Emergency Medicine, Royal College of Pathologists, Society for Acute Medicine, Royal College of Surgeons, Faculty for Intensive Care Medicine, Intensive Care Society, Society for Acute Medicine, Royal College of Radiologists, trusts/health boards, Royal Pharmaceutical Society, UK Kidney Association (clinical)<br>British Society for Echocardiography, Intensive Care Society - Focused Ultrasound in Intensive Care (FUSIC), Consortium for the Accreditation of Sonographic Education and Medical Schools/Universities (training)<br>National Institute for Healthcare Research (NIHR) (research into the use of point-of-care ultrasound) |
| IMPLEMENTATION SUGGESTIONS | ▪ Communications from national bodies to remind healthcare professionals to accurately record fluid balance (all fluid intake and output), and regular local auditing of completeness of fluid balance documentation<br>▪ Electronic patient record procurement criteria should include a requirement for intuitive access to fluid balance data, including ease of integrating it into clinical assessment<br>▪ Improved training for medical students, resident doctors and other clinical staff on how to undertake an appropriate clinical assessment of a patient's fluid status<br>▪ Development of appropriate training and accreditation for clinicians to expand the use of PoCUS alongside clinical assessment of fluid status<br>▪ Consensus agreement on how and when PoCUS should be used to complement clinical fluid status assessment<br>▪ Trusts/health boards to consider business planning to cover any additional technology required to deliver PoCUS testing |

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| | ▪ National improvement programmes to understand the challenges of consistently recording fluid balance, what might help to overcome those challenges, and to understand if there are any acceptable options to fluid balance monitoring (e.g. daily weights)<br>▪ NIHR to consider a themed call around the clinical trials comparing standard (clinical assessment) to PoCUS directed fluid therapy in the management of patients with an abnormal blood sodium. |

| 3 | Integrate point-of-care testing results into patient electronic records. |
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| FOR ACTION BY | Commissioners/integrated care boards with the hospitals in their trusts/health boards |
| RATIONALE FOR THE RECOMMENDATION | Point-of-care analysis, such as blood gas analysers, can enable clinicians to have an initial blood sodium result more rapidly than laboratory results. This allows faster determination if additional investigations and/or specific treatment of hyponatraemia or hypernatraemia is required. Frequently, results from point-of-care testing are not directly linked into the hospital laboratory electronic reporting system and require clinicians to transcribe or include them in the patient's medical records. This may not happen, so they are 'lost', and therefore are not available for review during the current or subsequent admissions, which would allow trends in blood sodium levels to be determined. It is essential that testing done using point of care analysers is validated and quality controlled to ensure the validity and consistency of the reported results. |
| ASSOCIATED GUIDANCE | Integrating in vitro point-of-care diagnostics: guidance for urgent community response and virtual ward services<br>Royal College of Pathologists: The retention and storage of pathological records and specimens (draft 6th edition)<br>Point of Care Testing: National Strategic Guidance for at Point of Need Testing |
| ADDITIONAL STAKEHOLDERS | Royal College of Nursing, Royal Colleges of Physicians, Royal College of Emergency Medicine, Royal College of Pathologists, Society for Acute Medicine, Royal College of Surgeons, Faculty for Intensive Care Medicine, Intensive Care Society, Society for Acute Medicine, Royal College of Radiologists, Association for Laboratory Medicine, Electronic Patient Record providers, |
| IMPLEMENTATION SUGGESTIONS | ▪ Hospital executives, supported by clinical and laboratory staff, should talk to their local business intelligence units (or equivalent) to determine how this integration of point-of-care testing can be achieved. |

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| | ▪ Undertake regular audit of adherence to entering full demographic data on point of care analysers to facilitate linkage to patient's electronic records, and identification of when exemption may be indicated (e.g. identity of patient unknown, mass casualty events). |

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| 4 | Develop a national standard for the use of hypertonic saline in the management of hyponatraemia. This should include:<br>• The indications for its use<br>• The dose, route and location of administration<br>• Monitoring the blood sodium levels, including the rate of correction<br>• Actions to be taken if over-correction occurs<br>• A consensus on the strength of hypertonic saline stocked in hospitals. |
| FOR ACTION BY | Society for Endocrinology |
| RATIONALE FOR THE RECOMMENDATION | Many patients had clinical features of hyponatraemic encephalopathy but only half were administered hypertonic saline, and there were patients with no clinical indication who had it administered. When it was administered, there was variation in the rate, route, strength, and location of administration. Currently there is variability in the strength(s) of hypertonic saline stocked in hospitals, which increases risk as resident doctors rotate between hospitals. Additionally, a fifth of patients administered hypertonic saline had inappropriate subsequent monitoring of their blood sodium levels which increases the risk of too-rapid sodium correction, a risk factor for developing osmotic demyelination syndrome. |
| ASSOCIATED GUIDANCE | Society for Endocrinology: Emergency management of severe and moderately severely symptomatic hyponatraemia in adult patients<br>NICE: Hyponatraemia scenario management<br>European Society of Endocrinology Clinical guideline for the management of hyponatraemia |
| ADDITIONAL STAKEHOLDERS | Royal Colleges of Physicians, Royal College of Emergency Medicine, Society for Acute Medicine, Royal College of Surgeons, Association of Surgeons, Royal College of Nursing, Faculty for Intensive Care Medicine, Intensive Care Society, Royal Pharmaceutical Society |
| IMPLEMENTATION SUGGESTIONS | ▪ Use of hypertonic saline could be improved through localisation of nationally developed guidance, to provide clinicians information on local specialist support for managing hyponatraemia<br>▪ Alongside this, the development of standardised training packages, potentially including multidisciplinary simulation training, would |

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| | improve the appropriate use of hypertonic saline and the assessment of patients with abnormal blood sodium levels<br>▪ Local agreements as to where patients are admitted following administration could be agreed<br>▪ Audits of blood sodium monitoring in patients given hypertonic saline<br>▪ Guidelines could have specific times at which blood sodium levels should be measured and a standardised treatment plan for managing over-correction to reduce the risk of patients developing osmotic demyelination syndrome. |

| 5 | Raise awareness of the importance of documenting and communicating all medication changes made in hospital to primary care as well as the patients and their family/carers. |
|---|---|
| FOR ACTION BY | Royal Colleges of Physicians, Royal College of Emergency Medicine, Society for Acute Medicine, Royal College of Surgeons, Association of Surgeons, Royal College of Nursing, Faculty for Intensive Care Medicine, Intensive Care Society, Royal College of General Practitioners, Royal Pharmaceutical Society |
| RATIONALE FOR THE RECOMMENDATION | Most patients reviewed were taking one or more medicine that could be associated with the development of either hyponatraemia or hypernatraemia. Patients should have a thorough medication review (prescribed, over-the-counter and others) at the time an abnormal blood sodium is identified. As a result, many patients had changes to their prescribed medications during the admission to hospital (for example doses changed, switching to alternative medicines, and/or stopping of medication(s)). These changes were not clearly outlined at the point of discharge to the GP, other healthcare professionals involved in their care, patients and/or their family/carers. |
| ASSOCIATED GUIDANCE | NICE Clinical Knowledge Summary: Hyponatraemia scenario management<br>Professional Record Standard Body: eDischarge Summary Standard<br>Royal College of Physicians: Acute care toolkit 17 Managing multiple medications |
| ADDITIONAL STAKEHOLDERS | Commissioners/integrated care boards, Department of Health and Social Care/NHS England, Welsh NHS, Health Department of Northern Ireland, Government of Jersey |
| IMPLEMENTATION SUGGESTIONS | ▪ Patients admitted with hypo- or hypernatraemia should have a comprehensive medication review at the point of identification of the abnormal blood sodium<br>▪ Hospitals should have protocols and/or a standard operating procedure on how the medication review should be undertaken, and regular auditing that this has been undertaken |

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| | ▪ Chief Executives and others could ensure that discharge letters include a mandatory section on whether any medication changes have occurred, with the rationale for those changes<br>▪ Clinicians should balance changing medicines to reduce the risk of recurrence of further abnormal sodium disorders against the risk of stopping a clinically important drug for an underlying long-term health condition (for example epilepsy)<br>▪ Involve appropriate specialists in outlining the rationale for the changes in communications to the GP and/or other healthcare professionals once made. Failure to do this increases the risk that medicines may be recommenced after discharge, leading to recurrence of the hyponatraemia and associated risks. Conversely, changes undertaken in primary care may not be visible when a patient presents to hospital<br>▪ Local agreements should be in place about who counsels patients, and their family/carers if appropriate, on their medications, including any changes, at the point of discharge<br>▪ The NICE clinical knowledge summary could be updated to strengthen information about communication of medication changes. |

# SUGGESTIONS FOR FUTURE RESEARCH

- Further work is needed to determine whether postoperative fluid protocols should be adjusted for weight and/or size, to reduce the risk of hyponatraemia and other electrolyte disturbances occurring.
- National guidelines or recommendations on how quickly clinicians should act on abnormal blood sodium levels once reported and on the criteria for reporting rapidly dropping sodium results, which may be a more important risk factor for the development of hyponatraemic encephalopathy than the absolute value.
- The use of point-of-care ultrasound in the assessment of blood sodium levels.

# CHAPTER 1: METHODS

## YOU CAN READ MORE ABOUT THIS HERE

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### Study advisory group

A multidisciplinary group of clinicians was convened to steer the study from design to completion, define the objectives of the study and advise on the key questions. The group comprised lay and patient representatives and healthcare professionals in clinical biochemistry, emergency medicine, endocrinology, intensive care medicine, general surgery, neurology, pharmacy, renal medicine and specialist nursing.

### Study aims and objectives

The objectives of the study were to identify and explore the avoidable and modifiable factors in the care of adults with abnormal levels of blood sodium levels in hospital.

### Study population and case ascertainment

**Inclusion criteria**

All patients aged 18 or over were admitted to hospital between 1st October 2023 and 31st December 2023 and identified as having hyponatraemia or hypernatraemia during their admission by retrospective ICD10 coding. Patients who presented as an emergency and those who developed abnormal blood sodium levels after surgery were included.

### Data collection

- A clinician questionnaire was sent to the named consultant for each patient in the sample. To collect data on the care provided throughout the admission, focusing on investigation and treatment of the patient's abnormal blood sodium level.

- An organisational questionnaire was sent to each hospital to collect data on the organisational structures, staffing provision and policies around the assessment and management of abnormalities in blood sodium levels.

- Copies of the case notes were requested for the included admission of each patient for peer review by a multidisciplinary group of case reviewers comprising consultants and trainees from acute medicine, anaesthetics, intensive care medicine, endocrinology, gastroenterology, general medicine, geriatric medicine, renal medicine and clinical biochemistry.

### Data analysis rules

- Small numbers have been suppressed if they risk identifying an individual (usually <5)
- Any percentage under 1% has been presented in the report as <1%
- Percentages were not calculated if the denominator was less than 100 so as not to inflate the findings, unless to compare groups within the same analysis
- There will be variation in the denominator for different data sources and for each individual question as it is based on the number of answers given.

# CHAPTER 2: DATA RETURNED AND THE STUDY POPULATION

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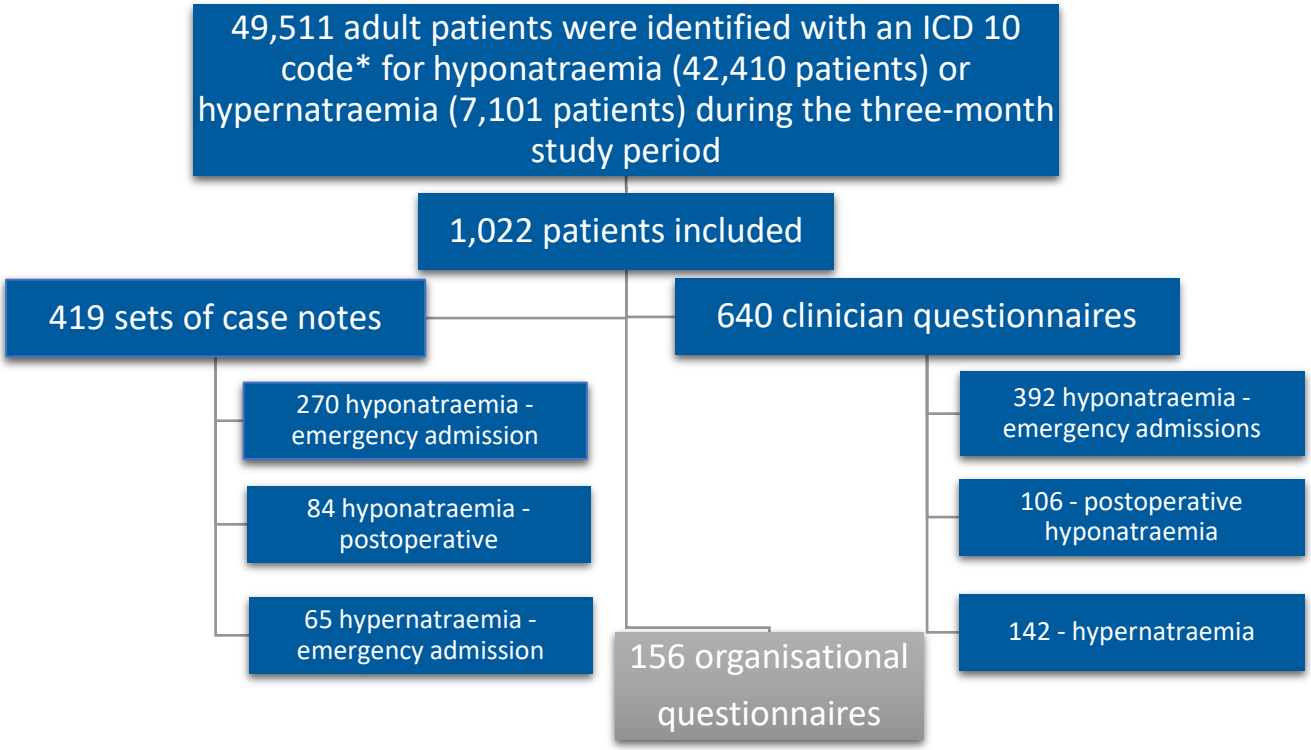
## Data returned




**The presence of an ICD-10 code would only have captured that hyponatraemia and/or hypernatraemia occurred during the admission but would not indicate the cause of the abnormal sodium level.*

In the whole study population, patients with a diagnosis code of hyponatraemia were slightly older (mean 74.0, median 77 years) than patients with a diagnosis code of hypernatraemia (mean 76.9, median 81 years) (F2.1).

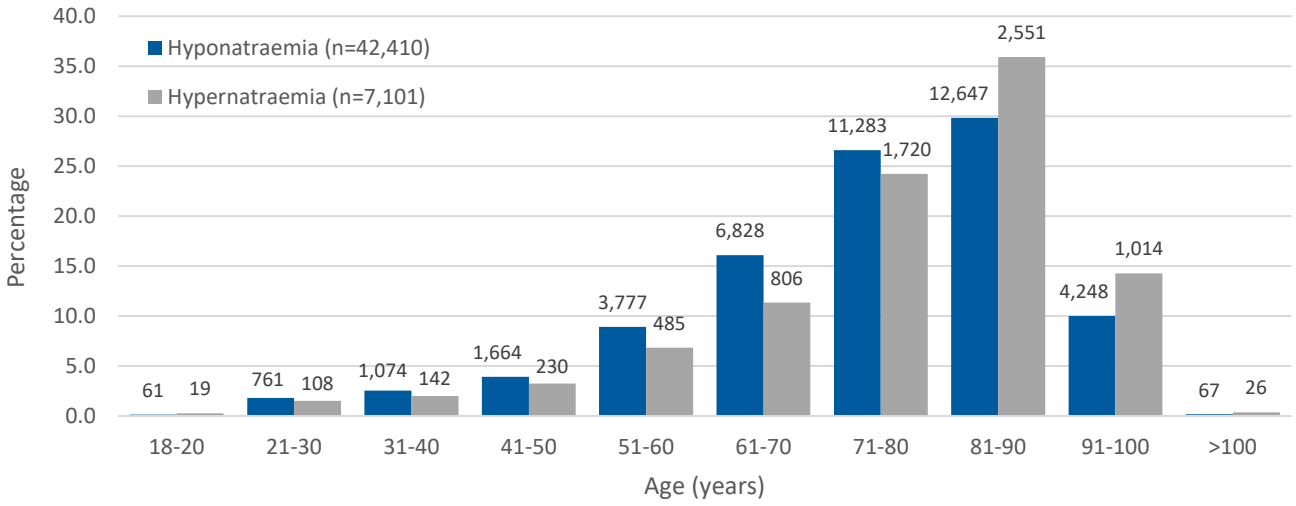



Figure 2.1 Age distribution of patients coded for hyponatraemia or hypernatraemia in the total study population

*Patient identification data*

In the sampled population patients coded with hyponatraemia hypernatraemia were slightly older (mean 69.8, median 73 years) than patients coded for hypernatraemia (mean 66.9, median 71 years) due to the sampling process which avoided over-including patients with hypernatraemia who had been admitted for end-of-life care (F2.2).

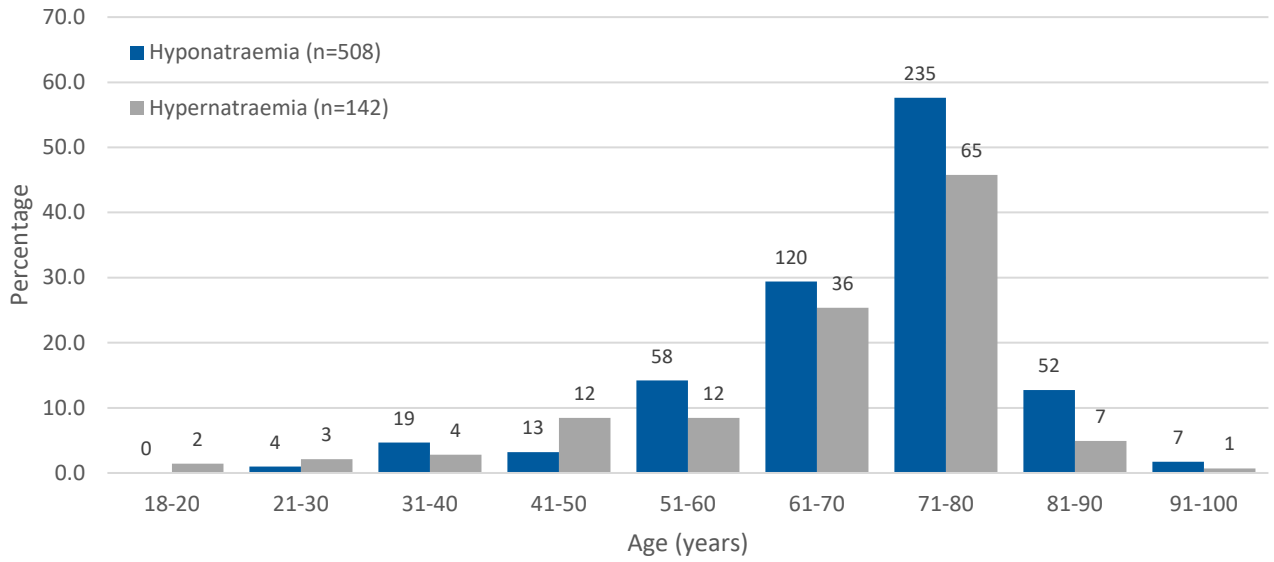



Figure 2.2 Age distribution of patients with hyponatraemia or hypernatraemia in the study sample
*Clinician questionnaire data*

In the sampled population, 281/508 (55.3%) patients with hyponatraemia were women and there was a higher proportion of women in the postoperative hyponatraemia group (72/106; 67.9%) compared to those admitted as an emergency (205/392; 52.3%).

The age distribution by sex for emergency admission-related hyponatraemia and postoperative hyponatraemia is shown in figures 2.3 and 2.4. While some previous reports have suggested that there is a sex difference between the risk of developing hyponatraemia, others have not.[1,2]

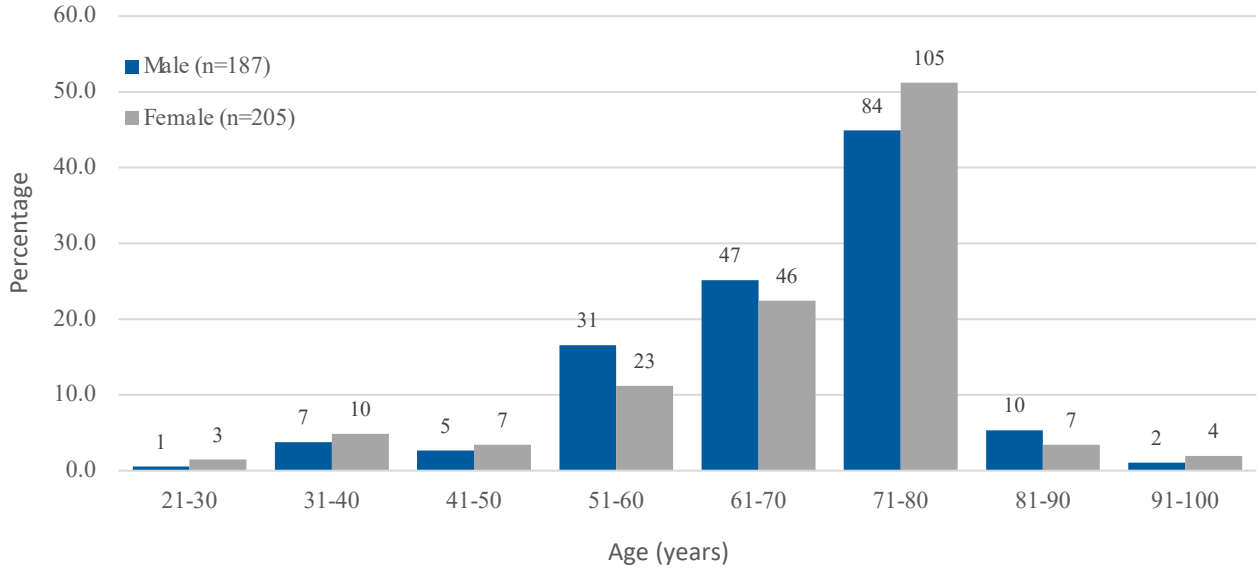



Figure 2.3 Age distribution by sex for emergency admission-related hyponatraemia
*Clinician questionnaire data*

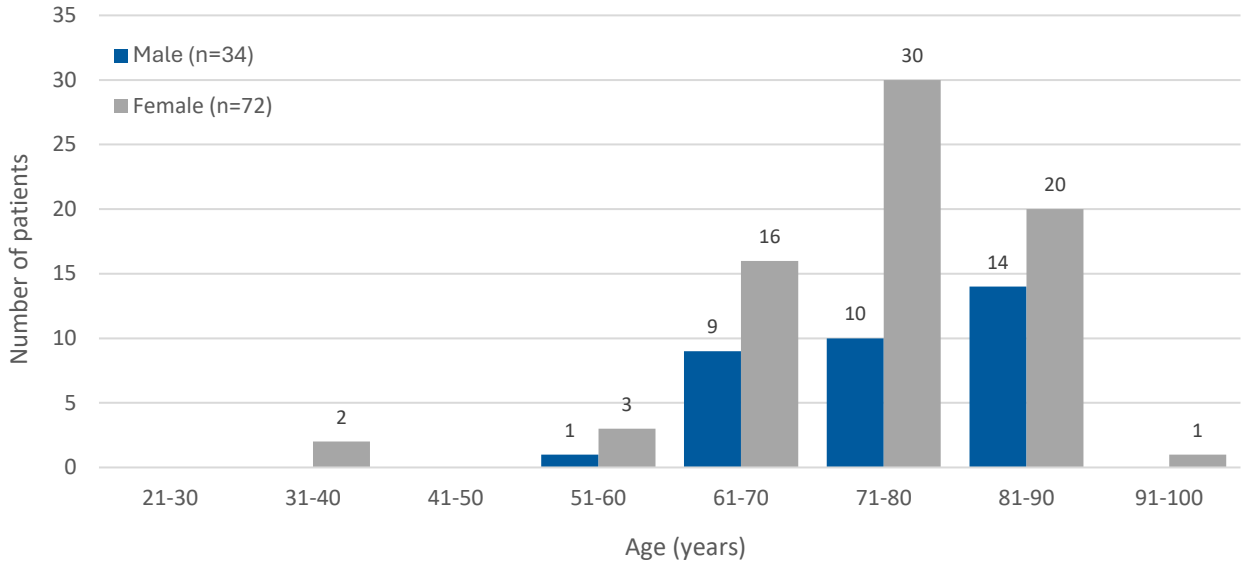



Figure 2.4 Age distribution by sex for postoperative hyponatraemia
*Clinician questionnaire data*

The higher proportion of women in both the emergency admission-related and postoperative hyponatraemia may reflect their greater risk factors. Additionally, the use of 'one size fits all' postoperative fluid protocols may increase the risk of hyponatraemia developing. Further work is needed to determine whether postoperative fluid protocols should be adjusted for weight and/or size, to reduce the risk of hyponatraemia and other electrolyte disturbances occurring.

Most patients with hyponatraemia (38,170/41,272; 92.5%) were emergency admissions; these patients were older than the elective admissions (median 78 years vs 72 years) (F2.5).

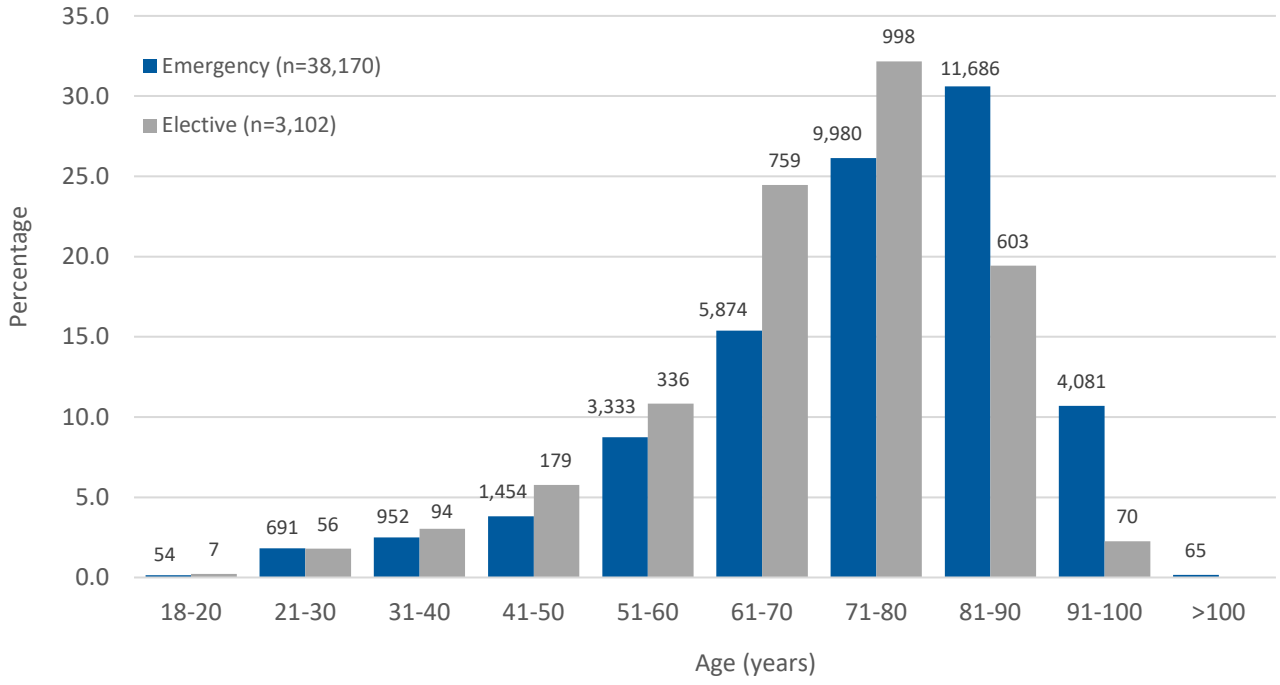



Figure 2.5 Age distribution of emergency and elective hyponatraemia coded admissions
*Patient identification data*

The ethnicity of the study sample was similar to the ethnicity data for England and Wales reported in the 2021 Census for England and Wales (T2.1).[3]

| Table 2.1 Ethnicity of the study population compared with the 2021 England and Wales Census | Study population | | Census data |
|---|---|---|---|
| | Number of patients | % | % |
| White British/White - other | 537 | 82.6 | 81.7 |
| Black/African/Caribbean/Black British | 15 | 2.3 | 4.0 |
| Asian/Asian British (Indian, Pakistani, Bangladeshi, Chinese, other Asian) | 37 | 5.7 | 9.3 |
| Mixed/multiple ethnic groups | 1 | 0.2 | 2.9 |
| Other (specified) | 2 | 0.3 | 2.1 |
| Unknown | 48 | 8.9 | Not applicable |
| **Total** | **640** | | |

*Clinician questionnaire data*

Overall, 26/640 (4.1%) patients with abnormal blood sodium levels were reported by the treating clinician to have a documented learning disability or autism.

Patients admitted as emergencies with hypernatraemia were typically frailer than those admitted with hyponatraemia.

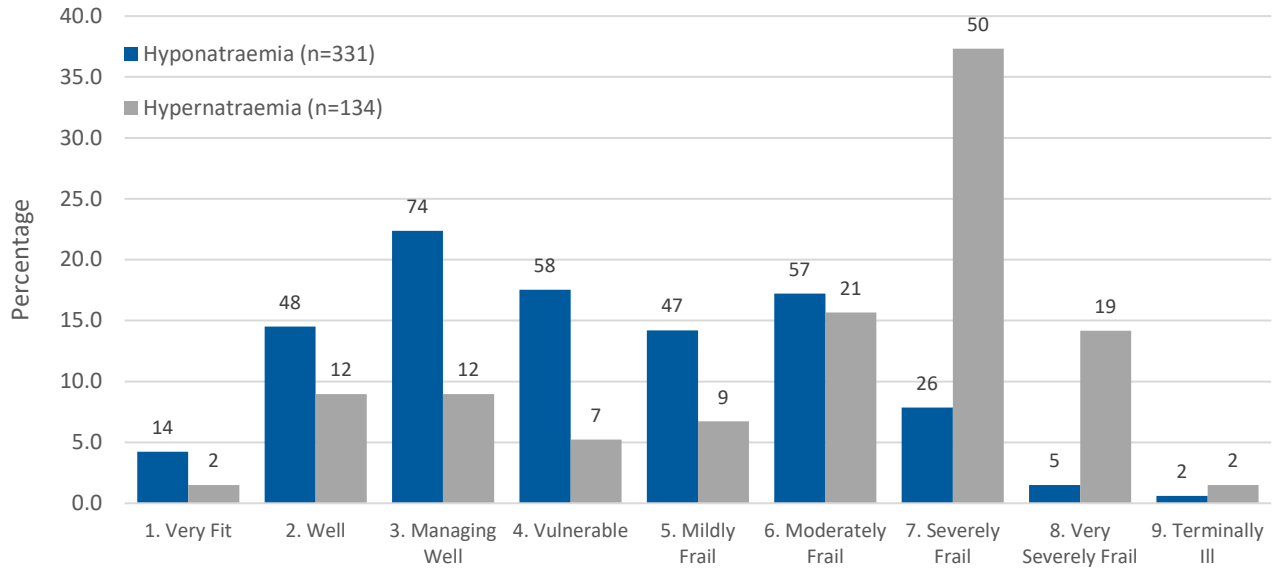



Figure 2.6 Rockwood Frailty Score for emergency admissions with hyponatraemia or hypernatraemia
*Clinician questionnaire data*

There were 136/331 (41.1%) patients with hyponatraemia who had a high functional status (Rockwood Frailty Score 1-3) compared to 26/134 (19.4%) with hypernatraemia (F2.6). This may reflect the greater proportion of patients with hypernatraemia who were admitted to hospital from a residential or nursing home (T2.2) and that they were more likely to have a 'do not attempt cardiopulmonary resuscitation' (DNACPR) decision or treatment escalation plan (TEP) in place (T2.3).

| Table 2.2 Usual place of residence prior to admission | Hyponatraemia | | Hypernatraemia | |
|---|---|---|---|---|
| | Number of patients | % | Number of patients | % |
| Own home | 359 | 93.0 | 75 | 53.2 |
| Residential home | 7 | 1.8 | 15 | 10.6 |
| Nursing home | 8 | 2.1 | 45 | 31.9 |
| Other (specified) | 12 | 3.1 | 6 | 4.3 |
| **Subtotal** | **386** | | **141** | |
| Unknown | 6 | | 1 | |
| **Total** | **392** | | **142** | |

*Clinician questionnaire data*

| Table 2.3 Do not attempt cardiopulmonary resuscitation or treatment escalation plan in place | Hyponatraemia | | Hypernatraemia | |
|---|---|---|---|---|
| | Number of patients | % | Number of patients | % |
| Yes - in place prior to admission | 19 | 7.9 | 22 | 36.1 |
| Yes - during initial clerking | 12 | 5.0 | 6 | 9.8 |
| Yes - during admission | 19 | 7.9 | 11 | 18.0 |
| No | 190 | 79.2 | 22 | 36.1 |
| **Subtotal** | **240** | | **61** | |
| Unknown | 30 | | 4 | |
| **Total** | **270** | | **65** | |

*Reviewer assessment form data*

Patients with hyponatraemia who are discharged without their sodium corrected, are potentially at risk of readmission related to hyponatraemia. Of the patients admitted as an emergency, 93/392 (23.7%) had been an inpatient in the previous 30 days and this was due to hyponatraemia in 31. Therefore, 31/384 (8.1%) patients in this study with hyponatraemia had been in hospital in the previous 30 days for hyponatraemia.

The presence of hyponatraemia during any admission to hospital may be a risk factor for subsequent readmission. At discharge any patient with hyponatraemia or who has had hyponatraemia during the admission should have an appropriate follow-up plan to monitor and adjust risk factors to prevent future admissions. The monitoring and risk factor management can be delivered by either primary or secondary care, or a co-ordinated approach by both. Patients may need more than one admission to understand the cause of their hyponatraemia.[4]

During the study period there were only a small number of patients admitted with Addisonian crisis (512 admissions), diabetes insipidus (419 admissions) or demyelinating disease of the central nervous system (218 patients). This may reflect the rarity of these diseases. Adrenal crisis can occur in patients with underlying adrenal insufficiency; this adrenal insufficiency can be due to rare causes such as Addisonian disease (which affects around ~1 in 10,000 people), or more common causes such as long-term high-dose steroid use. A growing number of experts and international bodies recommend renaming 'diabetes insipidus' to 'arginine vasopressin disorder' to prevent the inappropriate treatment resulting from its confusion with diabetes mellitus.[5,6]

Despite this change in name to reduce potential harm, greater education is needed to ensure that all clinicians understand what arginine vasopressin disorder means, both in terms of pathophysiology and treatment. The current coding system in the UK uses the International Classification of Diseases version 10 (ICD-10), which still uses the term diabetes insipidus (ICD-10 code E23.2), and it is therefore used in this report. Although version 11 of this coding system (ICD-11) was released in 2018, it remains in pilot testing internationally with no agreed switch date for its use in the UK. Even after the switch, the old term will remain in use in coding for either central diabetes insipidus (ICD-11 code 5A61.5) or nephrogenic diabetes insipidus (ICD-11 code GB90.4A).

More patients with hypernatraemia in this study died than those with hyponatraemia; this persisted when a sodium abnormality was the primary reason for their admission (hypernatraemia: 56/374; 14.9% patients died and hyponatraemia 132/5,384; 2.5% patients died) (T2.4 and 2.5). This may reflect the common underlying causes for hypernatraemia, particularly as hypernatraemia is often seen in patients who have poor oral intake as they approaching the end of life.

| Table 2.4 Mortality data – ICD-10 code in any position | Survived to discharge | Died | % mortality |
|---|---|---|---|
| Hyponatraemia E87.1 | 38,423 | 3,888 | 9.2 |
| Hypernatraemia E87.0 | 4,836 | 2,259 | 31.8 |
| **Total** | **43,259** | **6,147** | **12.4** |

*Patient identification spreadsheet data*

| Table 2.5 Mortality data – ICD-10 code in the primary position | Survived to discharge | Died | % mortality |
|---|---|---|---|
| Hyponatraemia E87.1 | 5,252 | 132 | 2.5 |
| Hypernatraemia E87.0 | 319 | 56 | 14.9 |
| **Total** | **5,571** | **188** | **3.4** |

*Patient identification spreadsheet data*

Mortality was lower when the sodium abnormality was coded as the primary reason for admission compared to being coded at any other point in the admission. This may suggest that in the majority of admissions other reasons had a greater impact on the risk of mortality than the sodium abnormality itself, however it was a contributing factor. It is possible that patients with hyponatraemia as a primary reason for admission are identified at the point of admission, and appropriate treatment is delivered in a timely manner reducing the impact of the hyponatraemia on subsequent risk of morbidity and mortality during the admission.

Despite the limitations of the current coding systems in identifying patients with Addisonian crisis or diabetes insipidus, there were recorded deaths in admissions where these conditions were included in the diagnostic codes at any stage (Addisonian crisis: 24 deaths; 4.7%, diabetes insipidus: 40 deaths; 9.5%). However, it should be noted that there were no deaths where either of these conditions was listed as the primary ICD-10 code (primary reason for admission) during the study period.

# CHAPTER 3: IDENTIFICATION OF HYPONATRAEMIA AND HYPERNATRAEMIA

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The blood sodium level for patients presenting to emergency care services, for example emergency departments (EDs), same day emergency care services or medical assessment units, should be available rapidly. Once an abnormal blood sodium level is identified this should then trigger the appropriate clinical assessment and further investigations to determine the cause. In addition, it is important that the clinicians involved in the care of the patient review results of previous blood sodium levels to determine the chronicity of the hyponatraemia or hypernatraemia and any changes from previous results. Chronic hyponatraemia is defined as hyponatraemia that has occurred over more than 48 hours; in addition, any patient who does not have a documented sodium assessment in last 48 hours should be assumed to have chronic hyponatraemia.

There were a small number of patients (7/392; 1.8%) who developed their emergency admission-related hyponatraemia after admission to hospital (i.e. they had a normal blood sodium result on presentation to the emergency department). The majority (258/392; 65.8%) of patients with emergency admission-related hyponatraemia had their lowest sodium level on presentation to hospital (120 (IQR: 116 to 124) mmol/L) (F3.1).

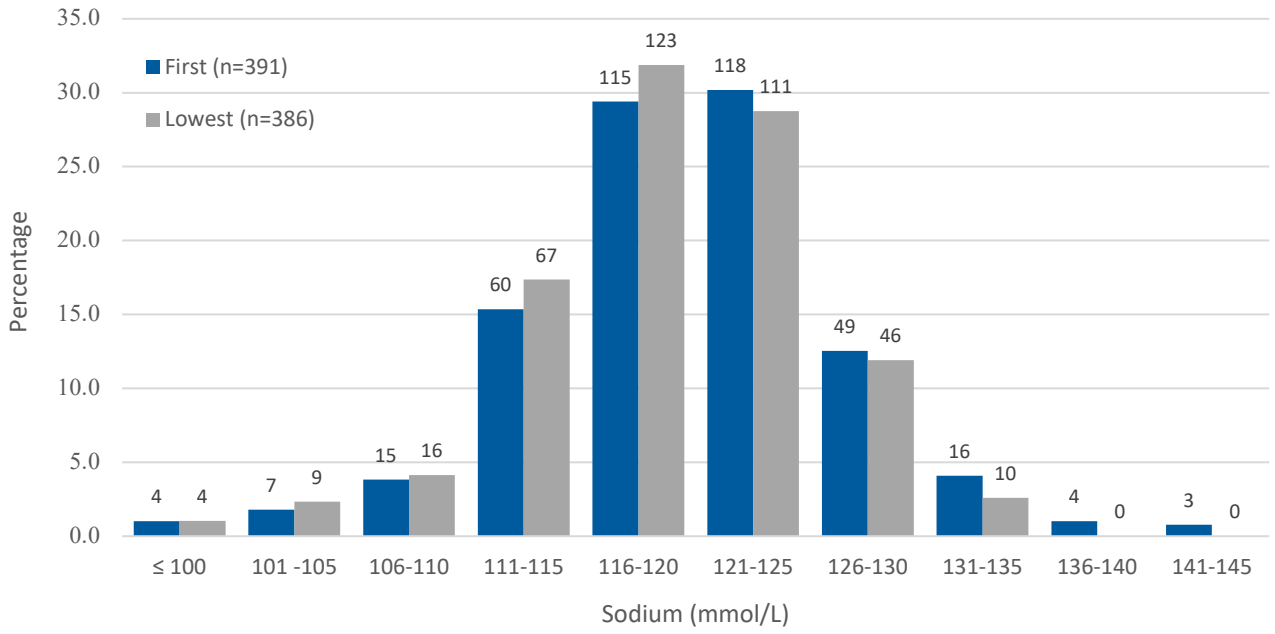


Figure 3.1 The first and lowest blood sodium levels for emergency admission hyponatraemia patients
*Clinician questionnaire data*

Patients who developed hyponatraemia postoperatively had less severe hyponatraemia based on their lowest blood sodium results; median lowest blood sodium was 120 (IQR: 116 to 123) mmol/L for emergency admission-related hyponatraemia and 125 (IQR: 122 to 128) mmol/L for postoperative hyponatraemia (F3.2 and F3.3). It was not possible to determine the impact of postoperative hyponatraemia on outcome due to small numbers.

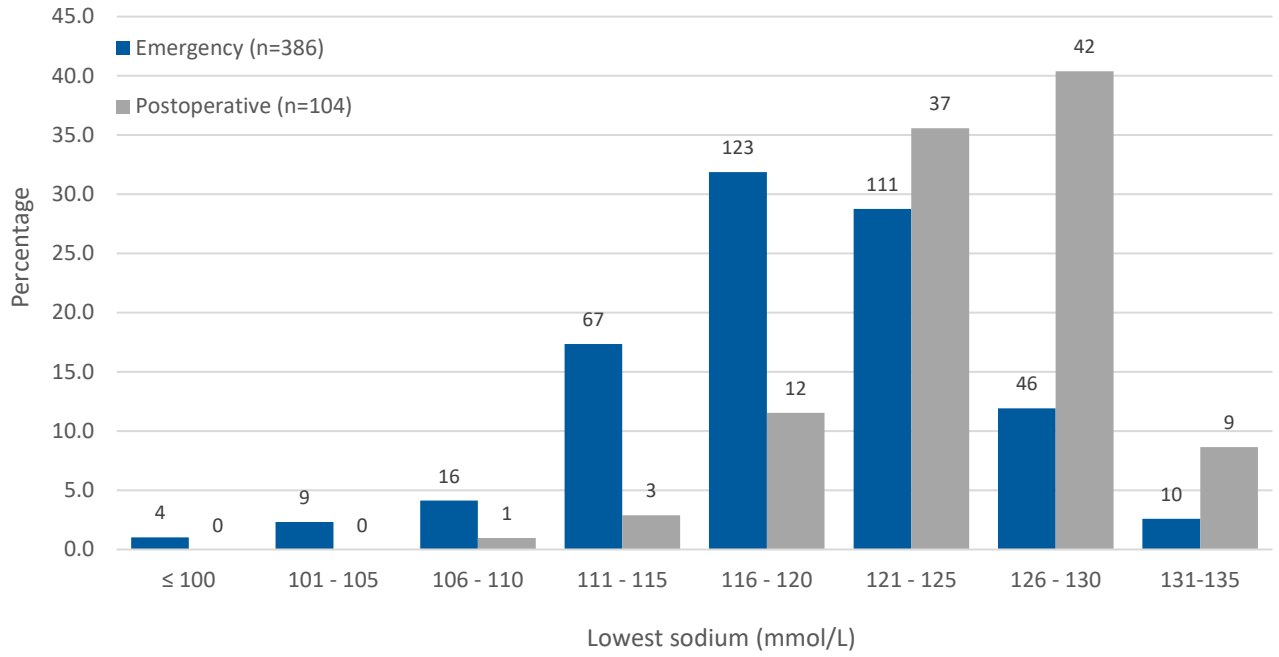


Figure 3.2 The lowest blood sodium levels for emergency admission and postoperative hyponatraemia patients by blood sodium concentration

*Clinician questionnaire data*

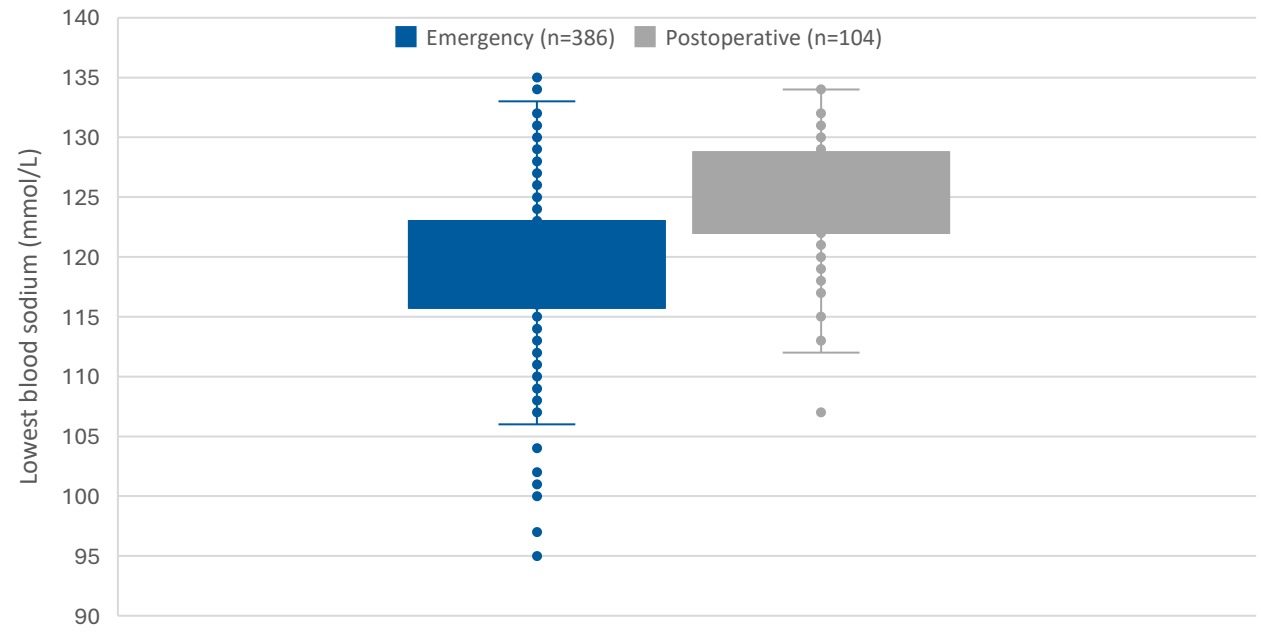


Figure 3.3 Comparison of lowest blood sodium levels between emergency admission and postoperative hyponatraemia

*Clinician questionnaire data*

The blood sodium level on admission (first) and highest blood sodium level in patients with hypernatraemia are shown in Figure 3.4. The median (IQR) blood sodium concentration on admission (first) was 150 (142 to 159) mmol/L and the median (IQR) highest blood sodium concentration during the admission was 156 (153 to 164) mmol/L (F3.5). Patients who had hypernatraemia were more likely to develop worsening of their hypernatraemia during their admission, as only 38/142 (26.7%) had their highest blood sodium level at the time of admission.

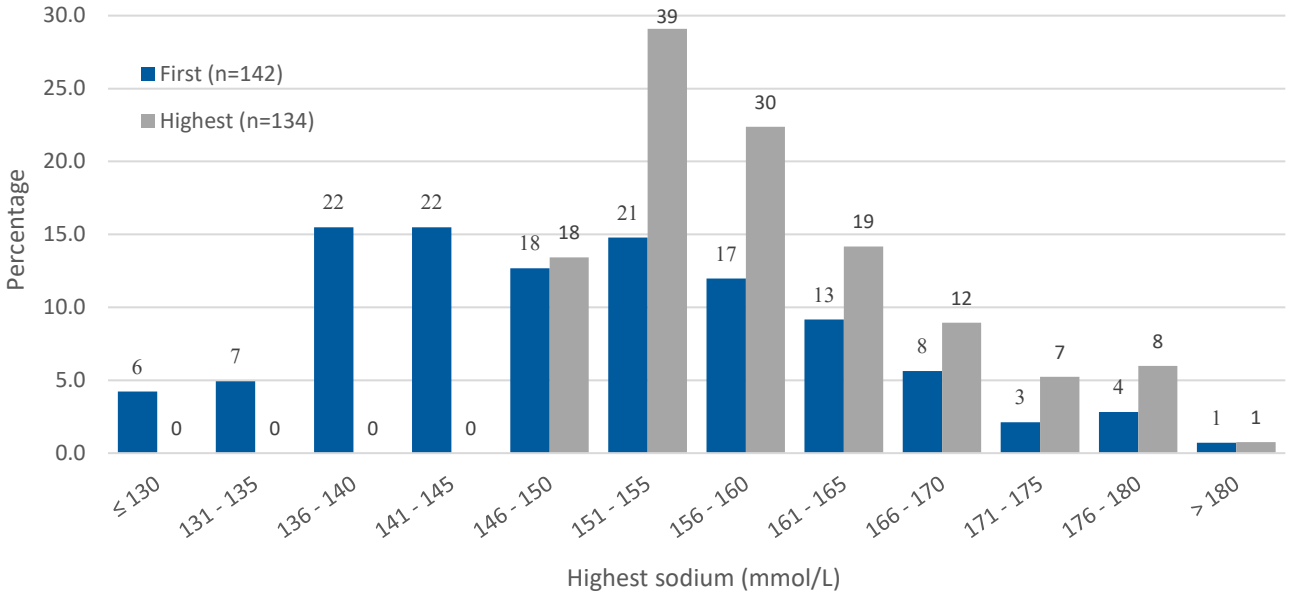


Figure 3.4 First and highest blood sodium levels in patients with hypernatraemia
*Clinician questionnaire data*

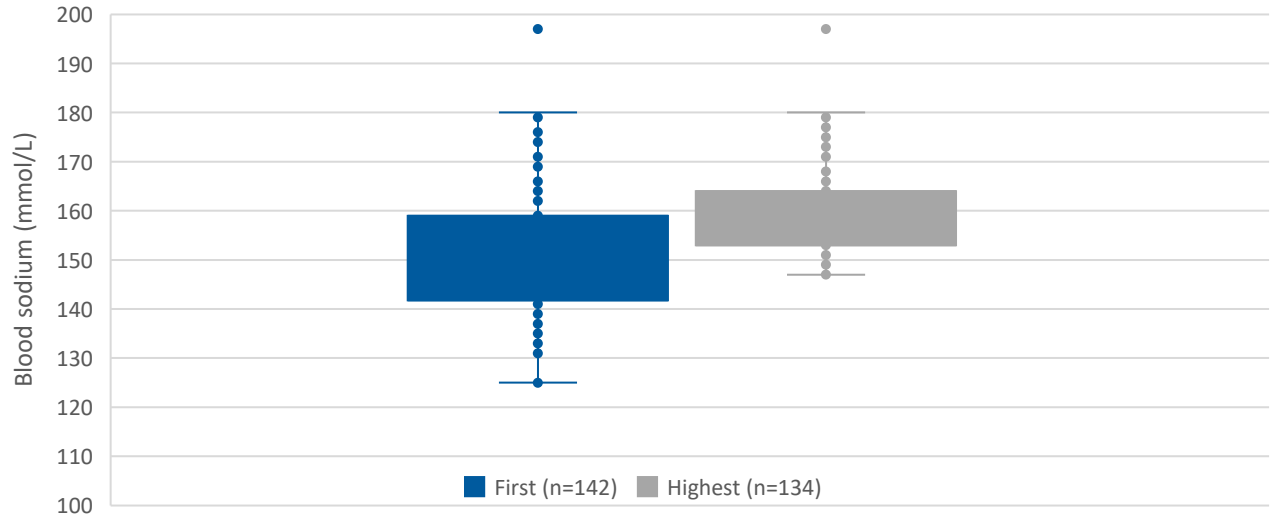


Figure 3.5 Comparison of admission (first) and highest blood sodium concentration in patients with hypernatraemia
*Clinician questionnaire data*

Royal College of Pathologists guidance in 2013 provided a key performance indicator on the turnaround times for specific blood testing from emergency department presentations, stating that *"90% of renal function and urea and electrolytes (U&Es) (both of which would include measurement of sodium) tests from A&E should be completed within one hour of sample collection,"* although it did note that the laboratory often may not be aware of the time of collection.[7]

This key performance indicator forms the basis of the subsequent 2019 Royal College of Pathologists *'Key Assurance Indicators for Pathology Services'* guidance and the 2nd Edition of the NHS England and NHS Improvement *'Pathology Quality Assurance Dashboard'*, which while not specifying turnaround times for laboratory tests, both stated that there needs to be local agreements between laboratory services and requesters on the anticipated times for all relevant laboratory investigations

around collection of the sample, receipt of the sample in the laboratory and the reporting of the result to the requester.[8,9]

Delays to the processing and analysis of the blood sample within the laboratory can impact on agreed times from collection to reporting of results, delays can also occur at various other points in the pathway. These include: i) time from presentation to blood collection; ii) time from blood collection to arrival in the laboratory; and iii) time for clinicians to act on the abnormal results.

There were 90/183 (49.1%) first sodium results available for patients with hyponatraemia within an hour of time of arrival at hospital. This increased to 137/183 (74.9%) within 2.5 hours (F3.6).

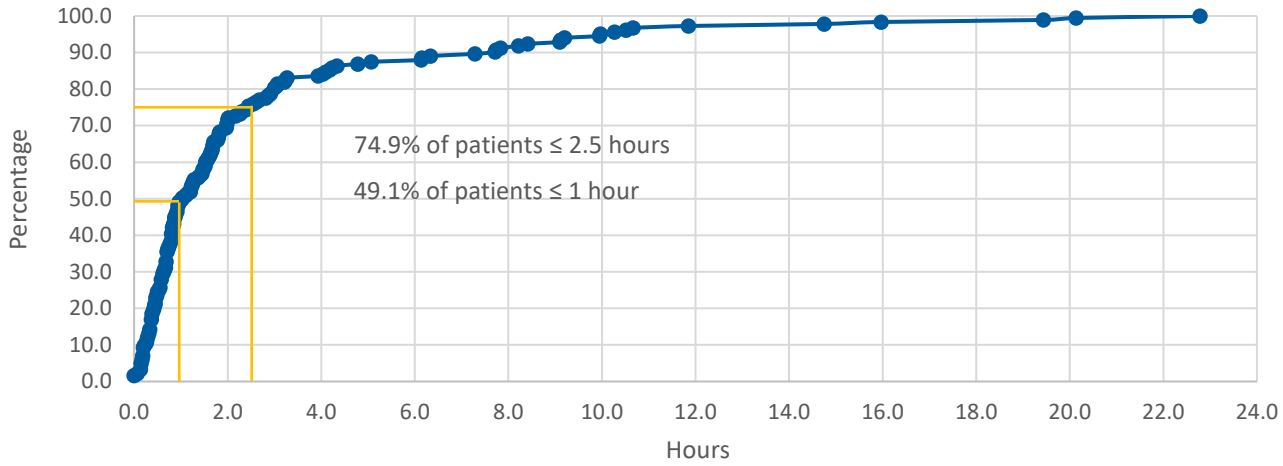


Figure 3.6 Time to first sodium measurement from arrival
*Reviewer assessment form data (n=183)*

Most of the initial sodium results in patients with hyponatraemia recorded in the clinician questionnaires (357/386; 92.5%) (unknown for 6) and reviewer assessment forms (169/263; 64.3%) (unknown for 7) were from laboratory testing rather than point-of-care testing (e.g. blood gas analyses).

More initial sodium results from point-of-care testing may have been available to the treating clinicians than indicated in the questionnaire responses. This discrepancy could arise because such results are not always fully integrated into laboratory electronic reporting systems, or clinicians using blood gas analysers might not have entered the patient identifiers needed to enable linkage. In these circumstances, the blood gas or other point-of-care results are typically printed and then once reviewed may not be filed in the patient's medical records.

During any single admission, it is important that clinicians follow the trend in results from one type of analysis (point-of-care or laboratory) as there is the potential for differences between different analytical methods.

Clinicians may act with caution on blood gas or other point-of-care results, concerned that electrolyte measurements on blood gas machines may be inaccurate. Where point-of-care testing machines are maintained, then the sodium results on these machines are reliable and can be used to guide treatment without the need to wait for confirmation from a laboratory processed sample.[10-12] Even where treatment is started prior to laboratory confirmation, the initial point-of-care test result should be subsequently confirmed by a laboratory-processed sample.

Royal College of Pathologists guidelines state that *"sodium values below 120 mmol/L in adults and 130 mmol/L in children should be escalated to clinical teams, ideally by telephone, within two hours."*[13] When the laboratory staff are unable to contact someone, staff should follow a local escalation procedure for managing the abnormal blood result. This guideline is being revised and does not have any changes to the sodium values that require alerting clinical teams, but there is greater emphasis on the use of non-telephone-based alerting systems. As most of the alerting is through telephone, this can lead to laboratory staff spending long times waiting to speak to clinical staff, meaning they cannot undertake other essential activities.

The majority of hospitals (123/156; 78.8%) had guidelines for laboratory staff to escalate abnormal results and set values to trigger an alert (T3.1).

| Table 3.1 Hyponatraemia values for escalation to clinical teams | Number of hospitals | % |
|---|---|---|
| Sodium ≤120 mmol/L | 98 | 79.7 |
| Sodium ≤125 mmol/L | 18 | 14.6 |
| Sodium ≤130 mmol/L | 7 | 5.7 |
| **Total** | **123** | |

*Organisational questionnaire data; n=156*

All hospitals reported that a sodium level of 120 mmol/L or less would be escalated to clinicians; interestingly, 25/123 (20.3%) hospitals reported that they alerted clinicians to sodium values higher (121 to 130 mmol/L) than the Royal College of Pathologist recommendations. This study did not provide the data to determine if, or how soon the treating clinical teams were alerted to a blood sodium level of less than 120 mmol/L by laboratory staff.

Currently there are no national guidelines or recommendations on how quickly clinicians should act on abnormal blood sodium levels once reported. And there are no recommendations on the criteria for reporting rapidly dropping sodium results, which may be a more important risk factor for the development of hyponatraemic encephalopathy than the absolute value.

Only 60/156 (38.5%) organisations had local guidelines to assist clinicians in the management of hypernatraemia. Despite this, 119/156 (76.3%) had criteria for laboratory staff for escalation of elevated blood sodium levels.

All of the local guidelines available met the Royal College of Pathologists guidance for rapid (within two hours) escalation to clinical staff of blood sodium levels above 160 mmol/L criteria, and 77/119 (64.7%) recommended escalation for blood sodium levels between 150 to 159 mmol/L (T3.2).

| Table 3.2 Blood sodium level for escalation by laboratory staff to clinical teams | Number of hospitals | % |
|---|---|---|
| Sodium ≥ 160 mmol/L | 40 | 33.6 |
| Sodium ≥155 mmol/L | 32 | 26.9 |
| Sodium ≥150 mmol/L | 45 | 37.8 |
| Other | 2 | 1.7 |
| **Total** | **119** | |

*Organisational questionnaire data; n=156, unknown for 37*

# CHAPTER 4: ASSESSMENT AND INVESTIGATION OF THE CAUSE OF HYPONATRAEMIA

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## 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).

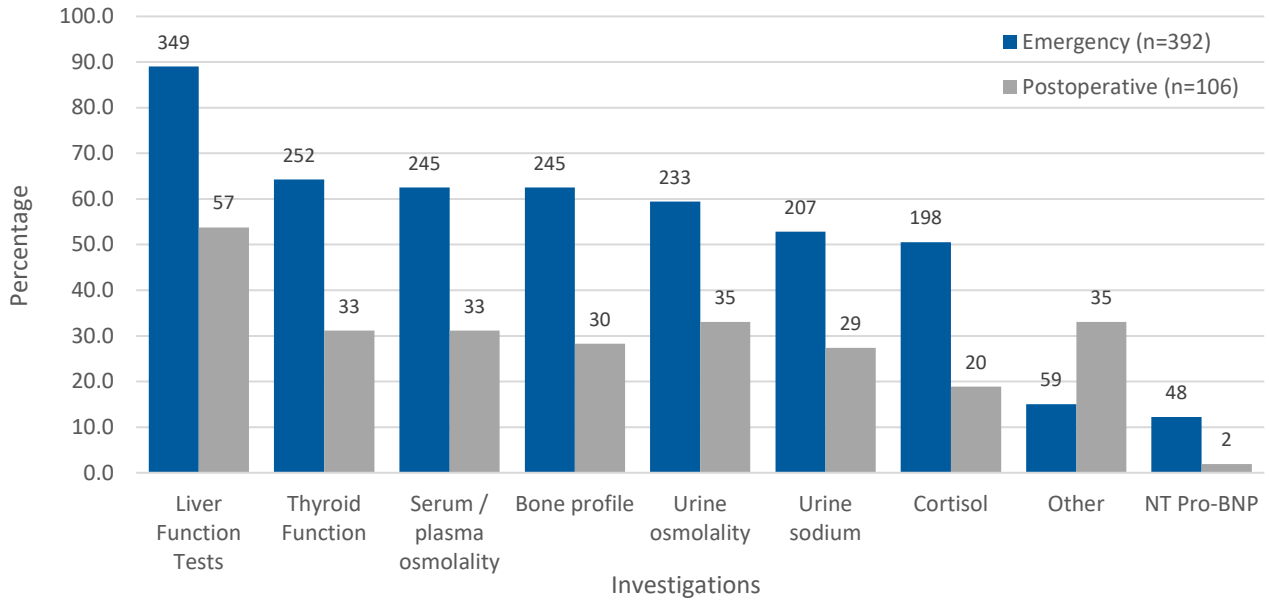


Figure 4.1 Investigations undertaken in emergency and postoperative hyponatraemia patients
*Clinician questionnaire data*

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.

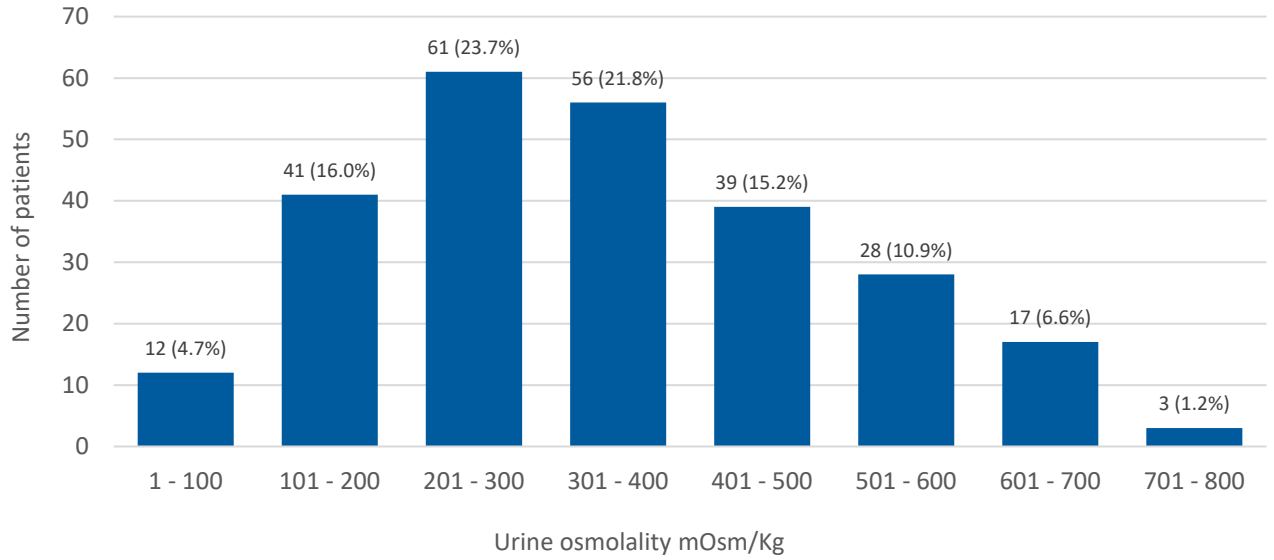


Figure 4.2 Urine osmolality concentrations in emergency admissions
*Clinician questionnaire data (n=257)*

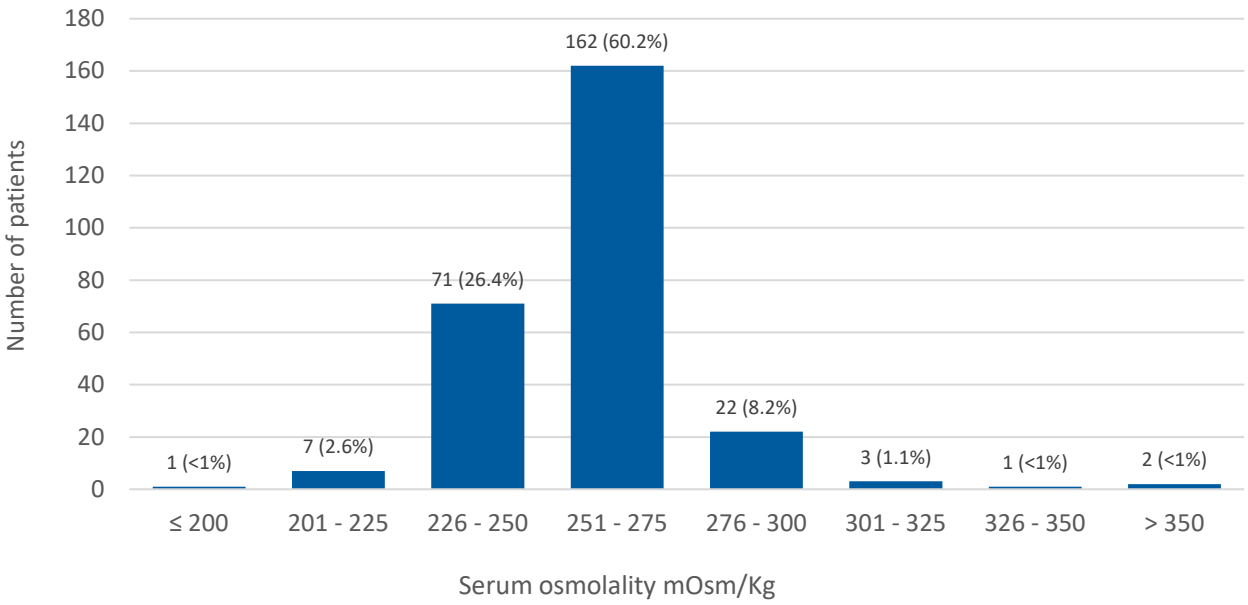


Figure 4.3 Serum/plasma osmolality concentrations in emergency admissions
*Clinician questionnaire data (n=269)*

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.

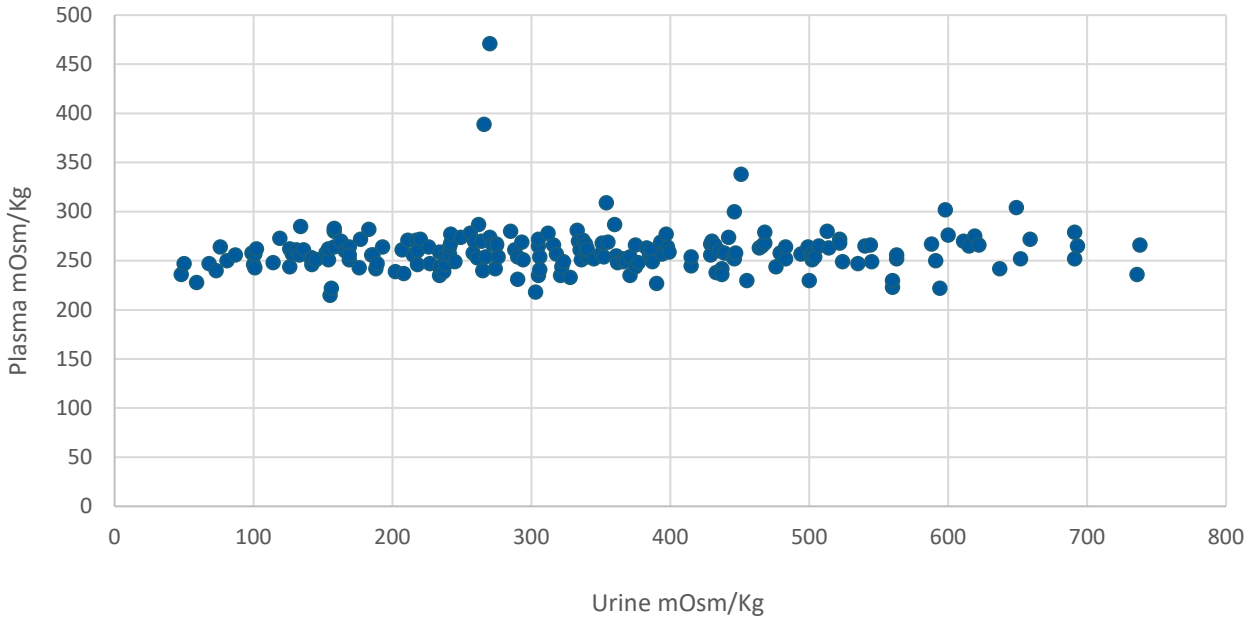


Figure 4.4 Relationship between serum/plasma and urine osmolality

*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.

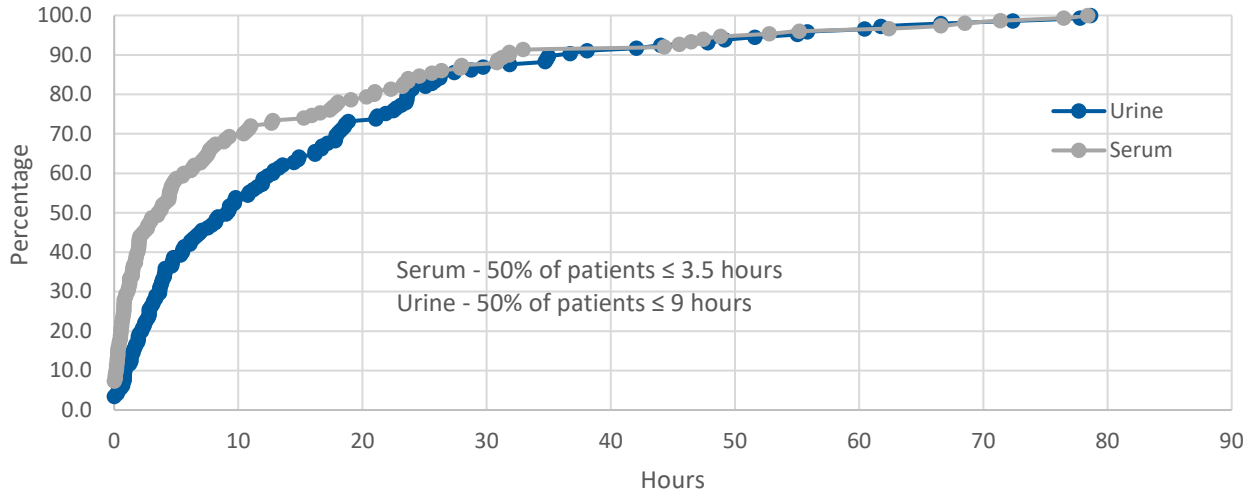


Figure 4.5 Time from osmolality request to result

*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 9:00am 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.

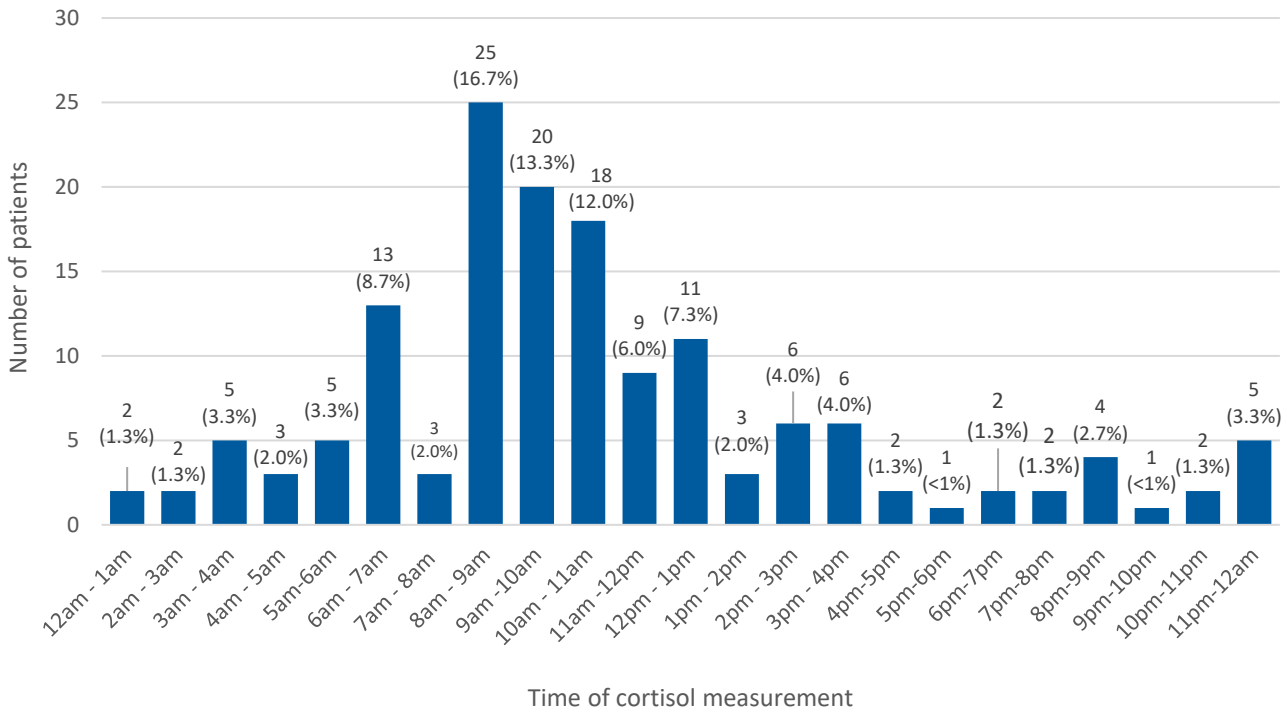


Figure 4.6 Times cortisol measurements were undertaken

*Clinician questionnaire data (n=150)*

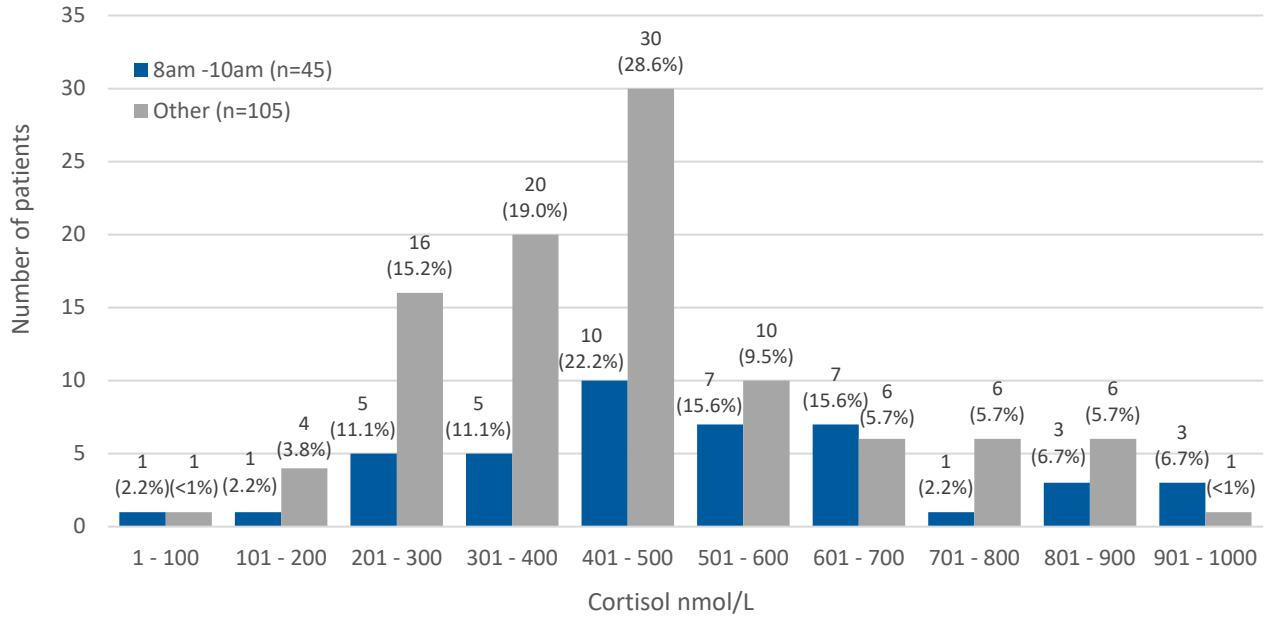


Figure 4.7 Range of cortisol results based on time test undertaken

*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.

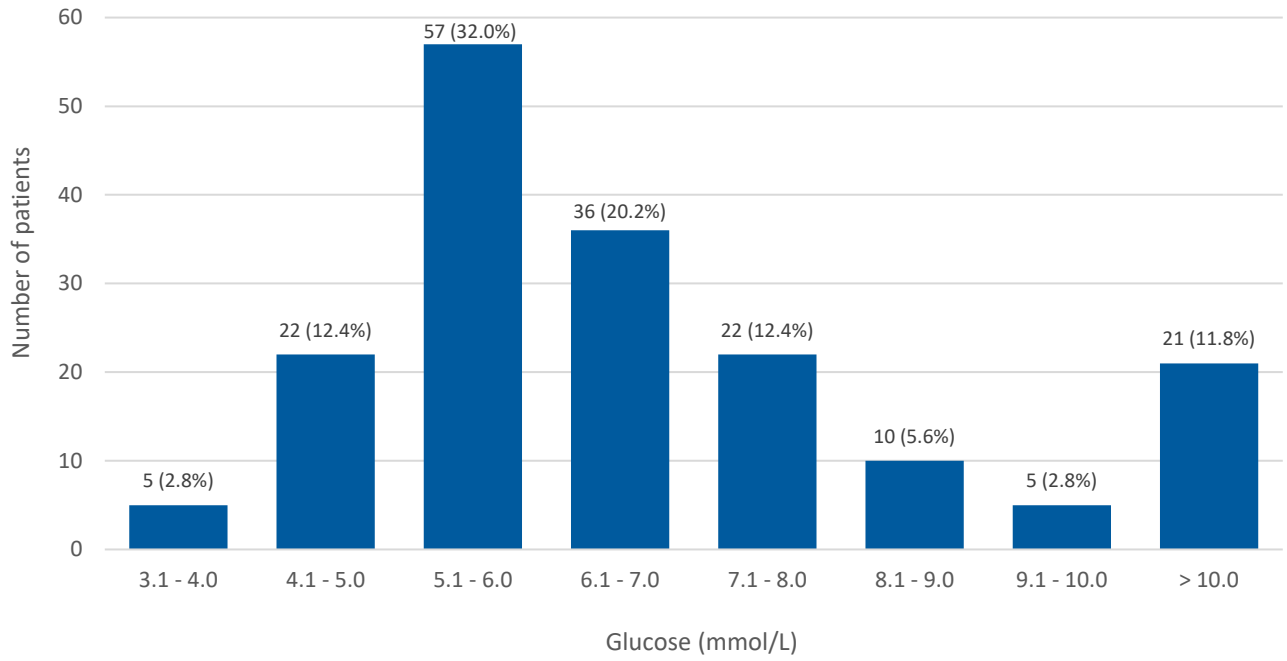


Figure 4.8 Range of glucose measurements at the time of the lowest sodium

*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.

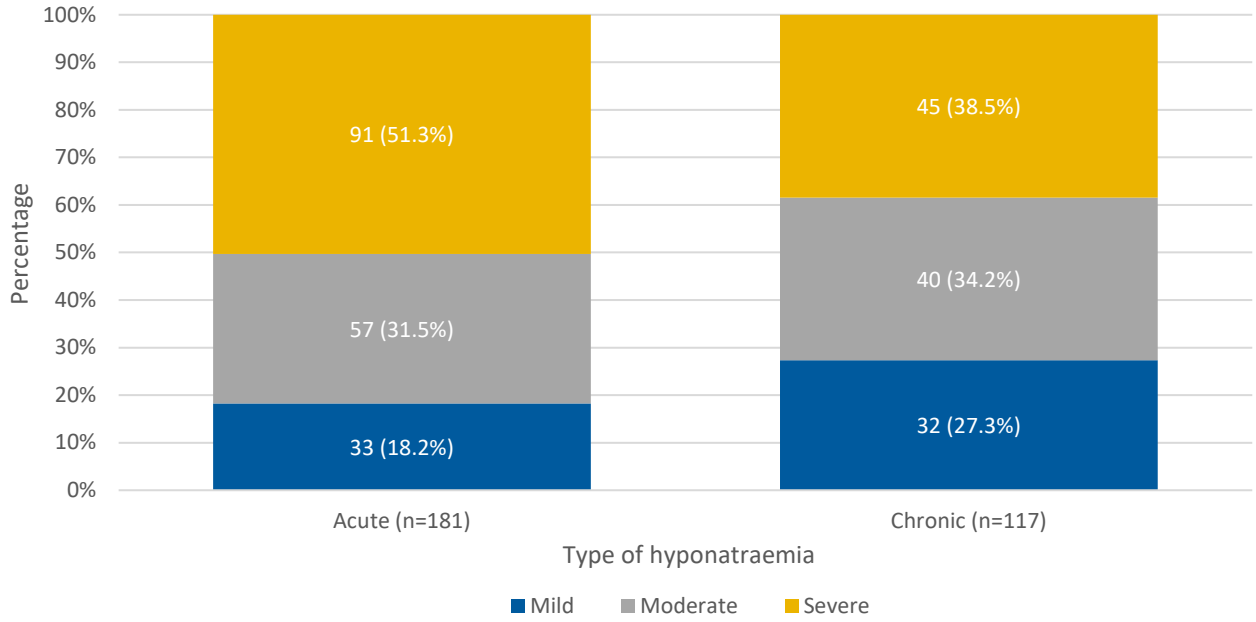


Figure 4.9 Comparison of hyponatraemia severity in acute or chronic hyponatraemia cases
*Clinician questionnaire data*

**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*

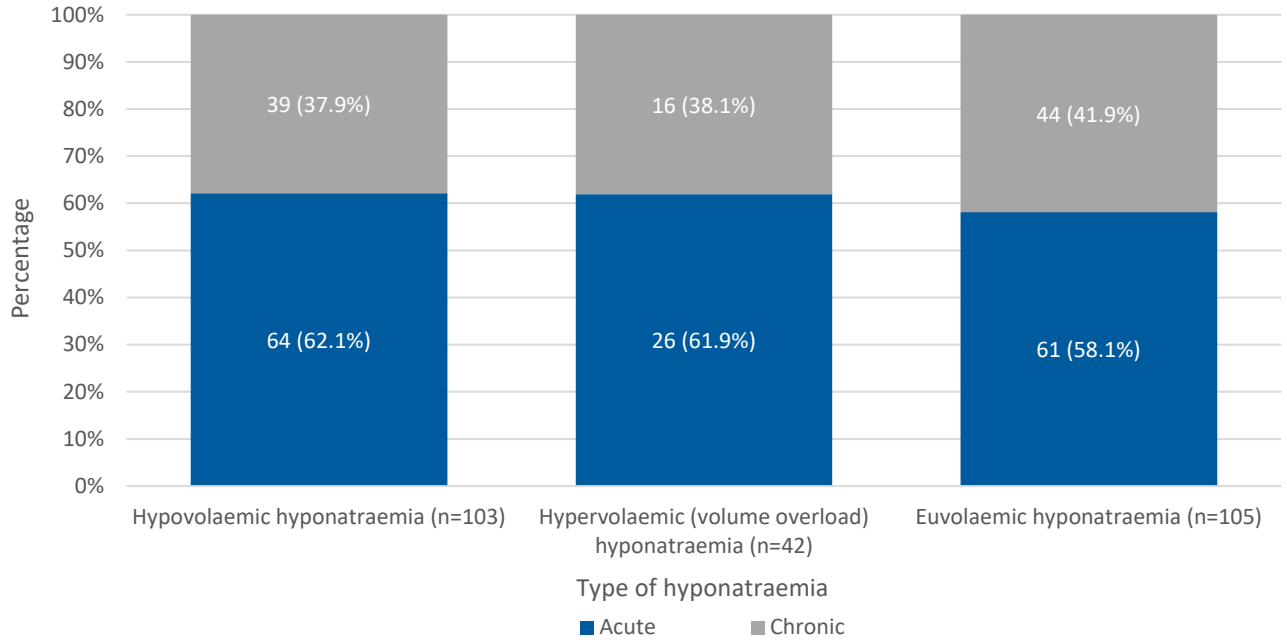


Figure 4.10 Comparison of frequency of acute compared to chronic hyponatraemia in the three most common types of hyponatraemia

*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).

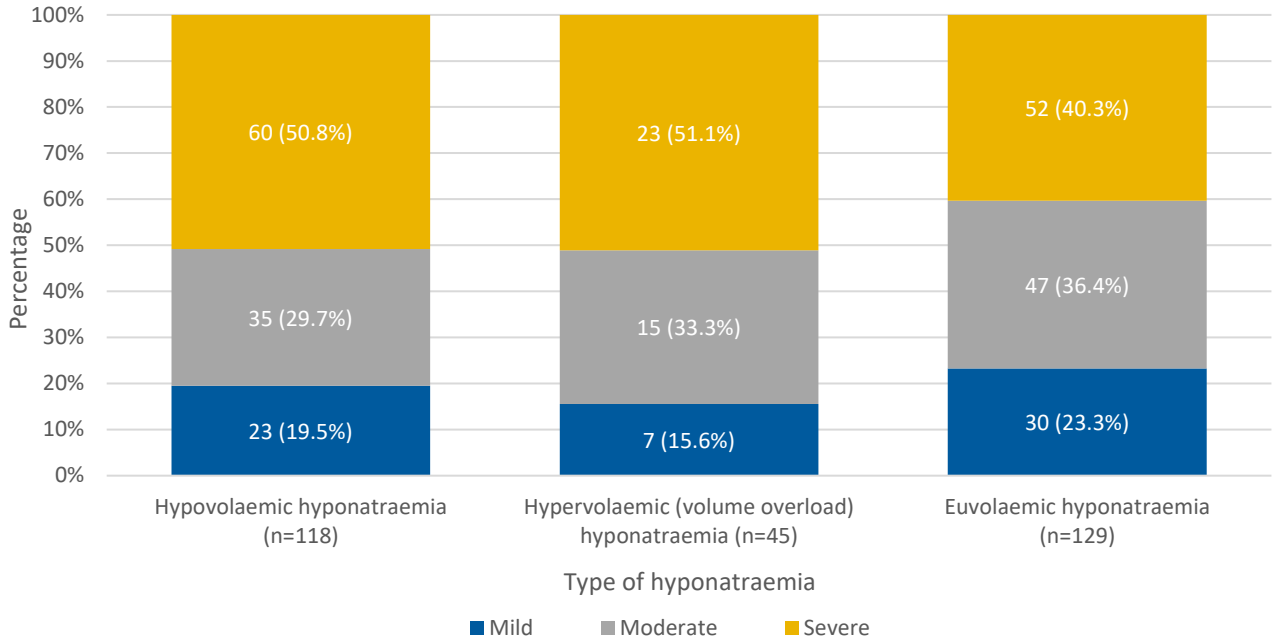


Figure 4.11 Comparison of degree of severity of the hyponatraemia for the three most common types of hyponatraemia

*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%) 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.

# CHAPTER 5: MANAGEMENT OF HYPONATRAEMIA

(BACK TO CONTENTS)

**Location of admission**

In general, the reviewers stated that the ward the patient was first admitted to from the emergency department (ED) was appropriate (247/262; 94.3%), although eight patients should have been admitted directly to a critical care area, rather than to a general ward. It is possible that the wide availability of critical care outreach teams supported the management of hyponatraemia in both the ED and after admission to general wards, meaning that subsequent admission to a critical care area was not required. In addition, patients with severe clinical or biochemical hyponatraemia may have had the appropriate initial treatment in the emergency department (56% of hypertonic saline was administered in the ED) and so may not have required an admission to a critical care area. Due to the potential for over-correction of blood sodium there is a need for close monitoring for the 24 to 48 hours after hypertonic saline is admitted; this level of monitoring may not be possible outside of critical care.

In total, 219/270 (81.1%) patients admitted as an emergency were admitted to an acute/general medicine/elderly care ward area and 31/270 (11.5%) were admitted to a critical care (level 2 or 3) area (T5.1). Three patients were admitted to an endocrinology ward, reflecting that endocrinology care is typically delivered as a specialist consultation service to patients admitted to other ward areas. The majority of hospitals do not have specialist endocrine inpatient beds.

| Table 5.1 Ward patient was first admitted to from the emergency department | Number of patients | % |
|---|---|---|
| Acute medical unit | 171 | 63.3 |
| General medical | 40 | 14.8 |
| Critical care | 31 | 11.5 |
| Care of the elderly | 8 | 3.0 |
| Cardiology | 4 | 1.5 |
| Endocrinology | 3 | 1.1 |
| General surgical | 2 | <1 |
| Neurology | 2 | <1 |
| Oncology | 1 | <1 |
| Renal | 1 | <1 |
| Unknown | 7 | 2.6 |
| **Total** | **270** | |

*Reviewer assessment form data*

**Treatment of hyponatraemia**

It was reported that most hospitals had clinical guidelines on the assessment and management of hyponatraemia 130/156 (83.3%), and 75/130 (57.7%) guidelines stated who should be responsible for the oversight and care of patients with severe hyponatraemia.

The treatments provided in both the emergency admission-related and postoperative hyponatraemia groups are shown in Table 5.2. The vaptans are selective vasopressin V2-receptor antagonists, which are licenced for use in patients with autosomal dominant polycystic kidney disease and associated rapidly progressing kidney disease. Additionally, tolvaptan is licenced in the UK for the management of hyponatremia secondary to the syndrome of inappropriate antidiuretic hormone secretion (SIADH), although typically this use is limited to specific groups of patients.

| Table 5.2 Treatments received for hyponatraemia | Emergency admissions | | Postoperative hyponatraemia | |
|---|---|---|---|---|
| | Number of patients | % of all patients (n=270) | Number of patients | % of all patients (n=84) |
| 0.9% sodium chloride solution | 166 | 61.5 | 41 | 48.8 |
| Fluid restriction | 107 | 39.6 | 27 | 32.1 |
| Hypertonic saline | 49 | 18.1 | 6 | 7.1 |
| Medications stopped | 38 | 14.1 | 5 | 6.0 |
| Other intravenous fluids | 25 | 9.3 | 8 | 9.5 |
| Diuretics | 18 | 6.7 | 4 | 4.8 |
| Oral sodium | 16 | 5.9 | 8 | 9.5 |
| None | 10 | 3.7 | 13 | 15.5 |
| V2 receptor antagonists (vaptans) | 8 | 3.0 | 0 | 0.0 |
| Demeclocycline | 3 | 1.1 | 1 | 1.2 |
| Enteral urea | 0 | 0.0 | 0 | 0.0 |

*Reviewer assessment form data*

There was very low use of vaptans; this reflect either low availability of these drugs in hospitals, a lack of awareness by non-specialist clinicians of their potential use in the management of hyponatraemia or a limitation on their use due to local medicines management. Overall, the choice of treatment was deemed to be inappropriate for a quarter of both emergency admission patients (63/256; 24.6%) and postoperative hyponatraemia (22/74; 29.7%) (T5.3).

| Table 5.3 Appropriateness of treatment choices for hyponatraemia | Emergency admissions | | Postoperative hyponatraemia | |
|---|---|---|---|---|
| | Number of patients | % | Number of patients | % |
| Yes | 193 | 75.4 | 52 | 70.3 |
| No | 63 | 24.6 | 22 | 29.7 |
| **Subtotal** | **256** | | **74** | |
| Unknown | 14 | | 10 | |
| **Total** | **270** | | **84** | |

*Reviewer assessment form data*

Eight patients with a blood sodium level of 100 mmol/L were administered IV fluids; this was 0.9% sodium chloride in five patients, hypertonic saline in two patients and a combination of hypertonic saline and 0.9% sodium chloride in one patient. None of the patients with a sodium of less than 100

mmol/L died, and therefore the decision to administer intravenous (IV) fluids and the choice of IV fluids was not associated with an adverse outcome.

The issues with the treatment(s) undertaken in both groups were broadly similar (T5.4). A total of 45 emergency admission-related hyponatraemia patients had both 0.9% sodium chloride and fluid restriction. This was inappropriate in only 10 patients; this reflects not only the challenge in the diagnosis of the cause of hyponatraemia, but also that on subsequent clinical assessment it may be appropriate to consider fluid restriction after a period of intravenous fluids or vice versa.

| Table 5.4 Summary of issues identified with the treatments provided | Emergency admissions | Postoperative hyponatraemia |
|---|---|---|
| Hypertonic saline indicated | 11 | 2 |
| Fluids and restriction | 10 | 3 |
| Hypertonic saline NOT indicated | 5 | 2 |
| No indication for 0.9% Saline | 3 | N/A |
| No treatment given | 2 | 5 |
| Slower rate required | 2 | N/A |
| Fluid restriction not indicated | 2 | 3 |
| Not enough fluid given | 2 | N/A |
| Tolvaptan not indicated | 2 | N/A |
| Medications not reviewed/stopped | N/A | 3 |
| Further treatment required | N/A | 1 |
| Fluids not indicated | N/A | 1 |

*Reviewer assessment form data*

Hyponatraemia in both the emergency admissions and postoperative groups had issues with use of hypertonic saline. There were 55 patients (49 emergency admissions, six postoperative) who received this as part of their treatment. The reviewers believed that hypertonic saline solution was not indicated for seven patients. In all seven this was because the severity of the hyponatraemia did not warrant the use of hypertonic saline solution, as the patient was asymptomatic or the reviewer's felt that there was no clinical evidence of hyponatraemic encephalopathy documented in the patient's notes.

A 2.7% sodium chloride solution was the most administered hypertonic saline (2.7% alone in 38 patients or combined 1.8% and 2.7% sodium chloride solution in 12 patients) (T5.5).

| Table 5.5 Type of hypertonic saline solution administered | Number of patients |
|---|---|
| 2.7 % | 38 |
| 1.8 % | 12 |
| Both 2.7% and 1.8% | 2 |
| **Subtotal** | **52** |
| Unknown | 3 |
| **Total** | **55** |

*Reviewer assessment form data*

International guidelines recommend the use of 3% hypertonic saline solution, although due to availability clinicians typically use 2.7% hypertonic saline.[26] The administration of 1.8% hypertonic saline will require larger volumes to deliver the same amount of sodium, this additional volume has a potential impact on baroregulated vasopressin secretion. It was more commonly administered by peripheral cannula rather than via central venous access.

Previous work has shown that administration of boluses of hypertonic saline is associated with better clinical outcomes.[27] Bolus administration occurred in 33 patients (bolus alone in 31, combined boluses and IV infusion in two) (T5.6). Typically, hypertonic saline was administered in a critical care area for 44 patients (ED resuscitation or level 2 or 3 critical care) (T5.7).

| Table 5.6 Frequency of administration by IV bolus and/or IV infusion | Number of patients |
|---|---|
| IV boluses | **31** |
| IV infusion | **22** |
| IV boluses and infusion | **2** |
| **Total** | **55** |

*Reviewer assessment form data*

| Table 5.7 Location where hypertonic saline solution was administered | Number of patients |
|---|---|
| Emergency department resuscitation | 28 |
| Critical care (level 2 or level 3) | 16 |
| Endocrinology | 2 |
| General medicine | 4 |
| **Subtotal** | **50** |
| Unknown | 5 |
| **Total** | **55** |

*Reviewer assessment form data*

Overall, where hypertonic saline was administered, in 17/55 patients there were issues with the decision to use, volume administered, rate of administration and/or duration of administration. While not captured in this study, the seniority and experience of the clinician involved in managing hyponatraemia is likely to impact on whether to treat with hypertonic saline, and where it is used, the appropriateness of how the hypertonic saline is prescribed and administered.

For 39/55 patients who were administered hypertonic saline outside of critical care, the grade of doctor who determined that hypertonic sodium could be identified in 22 sets of case notes. Ten were consultants, 11 were specialist trainees or specialty doctor and one was a foundation doctor. Of the six patients who were administered hypertonic saline where the reviewers felt it was not indicated, the decision was made by a consultant in two patients and by non-consultant doctors in four patients.

Due to the risk of over-correction patients who are administered hypertonic saline outside of critical care should be admitted to critical care for at least 24-48 hours of monitoring (including regular blood sodium measurements).[22,28,29] Of the 28 patients administered with hypertonic saline in an emergency department, only 11 were admitted to a critical care unit. The reviewers felt that five other patients were inappropriately admitted to a general ward area rather than critical care.

**Monitoring**

Blood sodium levels were monitored appropriately in 185/234 (79.1%) patients admitted as an emergency and 61/75 (81.3%) patients with postoperative hyponatraemia (T5.8). When hypertonic saline was administered, blood sodium levels were not monitored appropriately in 9/44 (20.5%) patients (T5.9).

The issues with monitoring were due to blood sodium levels not being rechecked soon enough and/or inappropriate frequency of monitoring after administration of hypertonic saline solution. The use of indwelling venous lines (for example midline catheters) or arterial lines may assist with the increased frequency of blood tests required in patients with severe hyponatraemia and especially following the administration of hypertonic saline.

| Table 5.8 Appropriateness of blood sodium monitoring for emergency admission and postoperative hyponatraemia | Emergency admissions | | Postoperative hyponatraemia | |
|---|---|---|---|---|
| | Number of patients | % | Number of patients | % |
| Yes | 185 | 79.1 | 61 | 81.3 |
| No | 49 | 20.9 | 14 | 18.7 |
| **Subtotal** | **234** | | **75** | |
| Unknown | 36 | | 9 | |
| **Total** | **270** | | **84** | |

*Reviewer assessment form data*

| Table 5.9 Appropriateness of blood sodium monitoring when hypertonic saline administered | Number of patients |
|---|---|
| Yes | 35 |
| No | 9 |
| **Subtotal** | **44** |
| Unknown | 11 |
| **Total** | **55** |

*Reviewer assessment form data*

When monitoring blood sodium levels, particularly after the administration of hypertonic saline, it is important that the same analytical method (either point-of-care or laboratory) is used to prevent differences in results between the analytical methodology impacting on the 'reported' rise in blood sodium levels. Therefore, it is essential that if the repeat testing is done using point-of-care analyses, the results are integrated into the laboratory electronic reporting systems, or they are accurately documented in the patient's medical notes, so that the trends in blood sodium levels are easily visible to anyone involved in the patient's care.

Over-correction of blood sodium levels, such as too-rapid increase in blood sodium concentrations, occurred in 22 patients and this was thought to have been avoidable in nine patients. The reviewers considered that more frequent sodium checks (four patients), excessive hypertonic saline solution (two patients) and failure to recognise a rapid rise in sodium (two patients) made the over-correction avoidable. In 12 patients, an attempt was made to re-lower the sodium with 5% dextrose

in ten patients and desmopressin in two. There was no harm reported in relation to those patients who had over-correction of the hyponatraemia.

As illustrated in the case below, rapid recognition of hyponatraemia in the emergency department and use of a local hyponatraemia management proforma can ensure appropriate administration and monitoring of hypertonic saline solution and recognition of the risk of blood sodium over-correction.

**CASE STUDY**

A 35-year-old patient with bulimia, alcohol excess, vomiting and history of a GI bleed presented to hospital and had a seizure in ED. Their sodium level was 115 mmol/L having been normal three days earlier. The initial seizure due to hyponatremia was recognised, and the patient received IV hypertonic saline in the ED. There was clear documentation of use of the local guideline, which included a completed 'hyponatraemia proforma' that gave clear instructions about the sodium levels over the first 48 hours, plus advice about the rate of correction/what to do in case of over-correction.

*The reviewers considered that this demonstrated a good assessment in the ED with rapid appropriate treatment of clinically and biochemically severe hyponatraemia. The completion of the hyponatraemia proforma showed a clear treatment/investigation plan had been put in place for the patient.*

The range of blood sodium concentrations on discharge for patients admitted with hyponatraemia is shown in Figure 5.1. The median (inter-quartile range)) blood sodium for emergency admission-related hyponatraemia had increased from the lowest blood sodium of 120 (116 to 123) mmol/L to 131 (128 to 134) mmol/L on discharge (F5.2). Similarly, for postoperative hyponatraemia it had increased from 125 (122 to 128.5) mmol/L to 132 (129 to 134) mmol/L on discharge (F5.3).

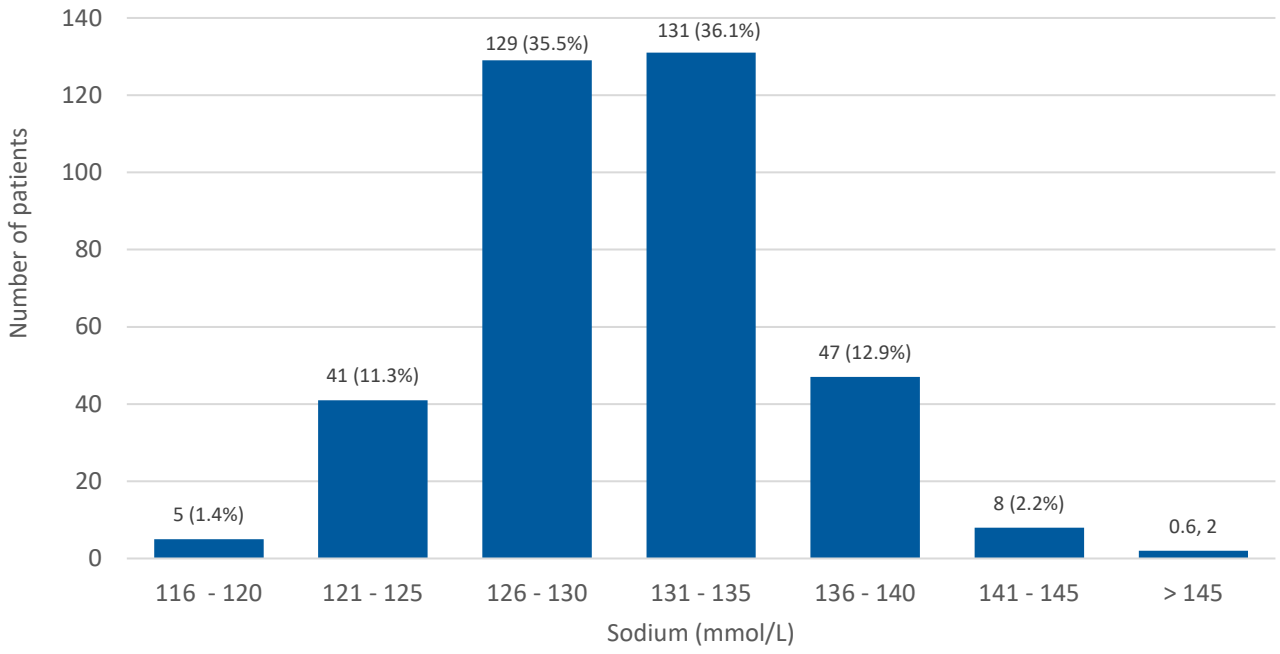


Figure 5.1 Post-discharge sodium concentrations

*Clinician questionnaire data (n=363, unknown for 29)*

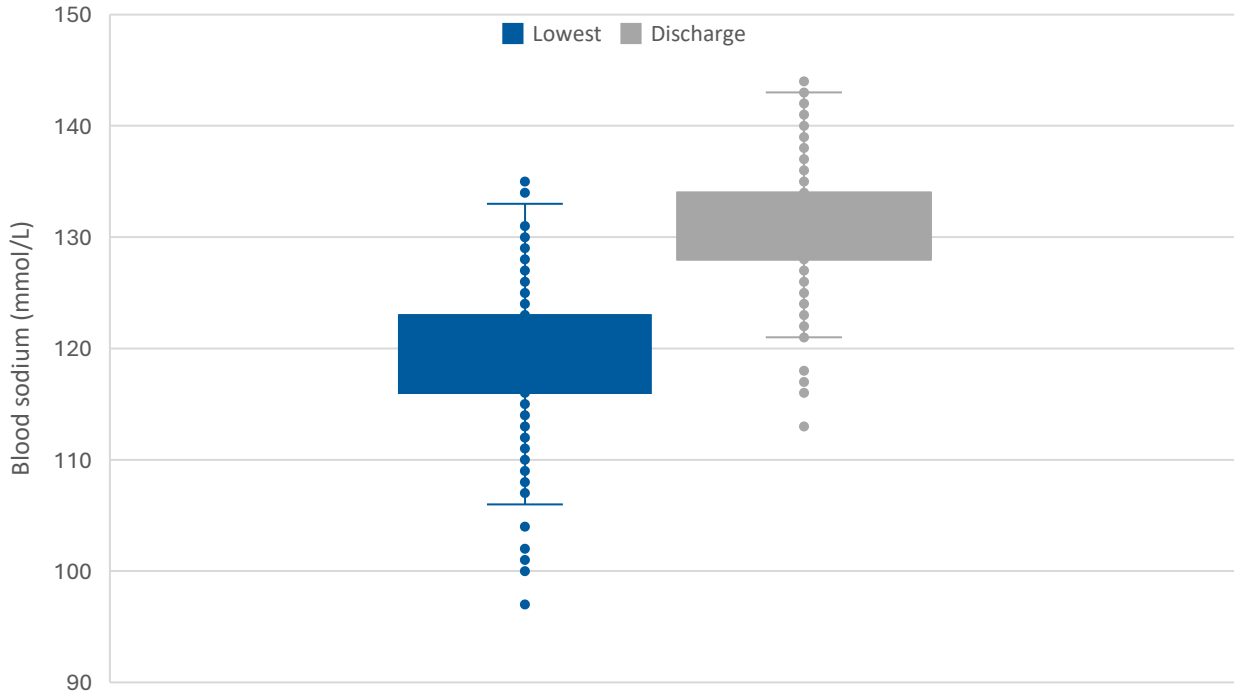


Figure 5.2 Comparison of change from lowest blood sodium levels to discharge blood sodium levels in patients with emergency admission-related hyponatraemia

*Clinician questionnaire data (n=362; unknown for 30)*

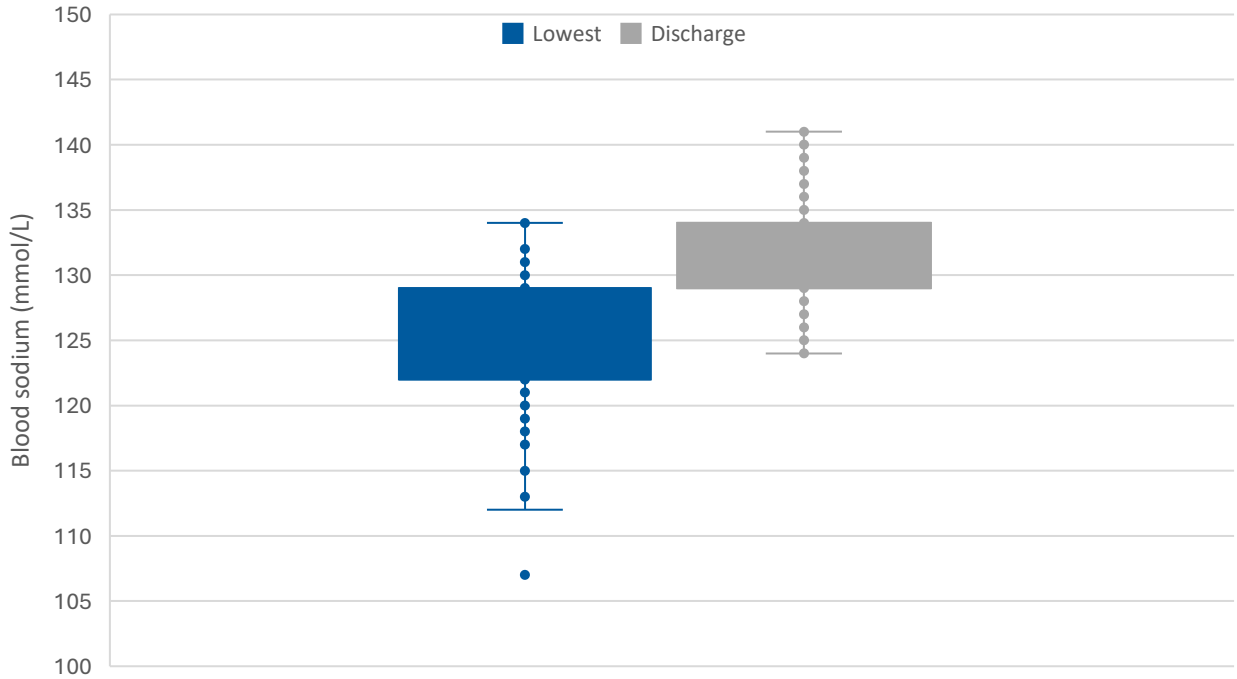


Figure 5.3 Comparison of change from lowest blood sodium to discharge blood sodium in patients with postoperative hyponatraemia.

*Clinician questionnaire data (n=106)*

There were 46/362 (12.7%) patients who were discharged with a sodium of 125 mmol/L or less. While this may be appropriate in some patients with known chronic hyponatraemia, others would require appropriate specialist follow-up rather than being discharged to the GP to monitor the sodium and 'treat', and this has to happen in an appropriate timeframe.

**Medication interactions**

Most patients admitted on an emergency basis with hyponatraemia were taking one or more medications prior to admission that could have contributed to their hyponatraemia (225/270; 83.3%) (T5.10).

| Table 5.10 Medicines potentially associated with hyponatraemia used prior to admission to hospital | Number of patients | % |
|---|---|---|
| Antihypertensive agents | 109 | 40.5 |
| Protein pump inhibitors | 106 | 39.4 |
| Diuretics | 76 | 28.3 |
| Antidepressants | 75 | 27.9 |
| Antiseizure medication | 29 | 10.8 |
| Antipsychotic drugs | 23 | 8.6 |
| Steroids – oral* | 19 | 7.1 |
| Opioid drugs | 17 | 6.3 |
| Other (specified) | 17 | 6.3 |
| Steroids – inhaled | 9 | 3.3 |
| Anticancer agents | 8 | 3.0 |
| Antibiotics | 6 | 2.2 |
| Non-steroidal anti-inflammatory drugs (NSAIDS) | 3 | 1.1 |

*Reviewer assessment form data. Answers may be multiple; n=269*

**Failure to take oral steroids can lead to hyponatraemia if stopping use can lead to adrenal insufficiency*

Of the 76 patients taking diuretics, 27 used thiazides, 19 used loop diuretics, and in 30 patients the type of diuretic was not specified. The median admission blood sodium concentration was similar for patients taking thiazide diuretics (117.6 mmol/L) and those taking loop diuretics (117.4 mmol/L).

Our sampling methodology, which focused on patients with more severe hyponatraemia, may have prevented us from showing an association between thiazide diuretic use and lower blood sodium concentrations at admission. Often the initiation of a medicine and/or a change in its dose can precipitate the development of hyponatraemia. The case reviews did not provide information on the timing of starting any medicines or dose changes in relation to the presentation with hyponatraemia.

Given the potential relationship between certain medicines and the development of hyponatraemia 157/270 (58.1%) patients with emergency admission-related hyponatraemia had one or more changes to the medications they were taking on admission that may have contributed to the development of the condition. These changes may have occurred at the time of admission, at any point during the admission, or at the point of discharge.

It is important to undertake a risk-benefit assessment before stopping a medication. For example, if the evidence for the development of hyponatraemia related to an anticonvulsant is weak, the benefit of stopping in relation to the hyponatraemia maybe minimal, while the risk of developing seizures from stopping may be significant.

The most common changes made were discontinuation of a medication likely to cause hyponatraemia (141 patients) or dose adjustments to reduce risk of recurrence of the hyponatraemia (12). Of note, 17 patients had a change from use of a proton pump inhibitor to famotidine (a gastric acid suppressant which is not associated with hyponatraemia) during admission or on discharge.

In terms of other specific treatments related to hyponatraemia, ten patients had slow sodium commenced, four patients were started/had an increased dose of steroid treatment, two were started on tolvaptan and one had a change in their previous dose of desmopressin.

Where medications were changed, most changes were communicated to the GP on discharge (140/151; 92.7%). Commonly the 'communication' to the GP that a medicine has been stopped is its absence from the patient's medication list at discharge. It can therefore be unclear whether this is an intentional discontinuation or an omission on discharge prescribing.

It is vital that there is clear documentation on discharge that the medicine has been actively discontinued and the underlying reasons for this decision should be included. Additionally, there should be guidance or details of who to contact about restarting the medication if that is needed.

**CASE STUDY**

A 36-year-old patient was referred to hospital by their GP with a low sodium and increased lethargy over the last few days. The patient had complex medical issues and was taking multiple medications. The patient received a timely medication review which included neurology input. The neurology review led to the stopping of the anti-seizure medication that could be associated with hyponatraemia, with an alternative started. This was clearly communicated to the GP and an outpatient neurology follow-up was booked for the patient.

*The reviewers considered that this demonstrated very good communication between hospital clinical teams and with primary care in a complex medical case.*

In those patients where no changes were made to medications during their hospital admission, reviewers identified that changes should have been made in 14/67 patients. These changes primarily involved stopping medicines associated with hyponatraemia (seven patients) and wider longer-term medication/disease management reviews (four patients). It was not possible to determine whether changes in medication were explained to the patient at the time of discharge. This is essential, as if patients are not aware of the changes, they might restart the stopped medicines using pre-dispensed supplies at home or contact the GP to request a repeat prescription.

**Delays in treatment**

Delays occurred in the investigation or management of hyponatraemia in 17/64 (26.6%) emergency presentations and 5/18 (27.8%) postoperative hyponatraemia patients (T5.11). These delays were attributed to the impact of out-of-hours care where typically there was reduced medical, nursing and laboratory staff. Some of these delays could have been mitigated if clinicians had confidence to start treatment based on point-of-care testing (e.g. blood gas results) rather than waiting for laboratory results.

| Table 5.11 Frequency of delays in investigation and/or management of hyponatraemia | Emergency admissions | | Postoperative hyponatraemia | |
|---|---|---|---|---|
| | Number of patients | % | Number of patients | % |
| Yes | 64 | 25.1 | 18 | 22.8 |
| No | 191 | 74.9 | 61 | 77.2 |
| **Subtotal** | **255** | | **79** | |
| Unknown | 15 | | 5 | |
| **Total** | **270** | | **84** | |

*Reviewer assessment form data*

Reviewers identified issues with responsibility for the initial and then ongoing management of postoperative hyponatraemia. While the on-call general medicine team will provide advice on the management of hyponatraemia, they tend not to assume responsibility for the care of a postoperative surgical patient with hyponatraemia. This may be because the hyponatraemia could resolve quickly than the postoperative needs of the patient, which would be better managed by the surgical team.

In orthopaedic surgery, many hospitals have addressed this by providing proactive care for older patients undergoing surgery (POPS) teams. These teams support the orthopaedic surgeons and manage the hyponatraemia while the orthopaedic surgeons manage the orthopaedic issues in parallel,[29] Given the benefits that this model of care has delivered, there is increasing interest in it being delivered in other surgical specialties.[30]

Complications related to the hyponatraemia occurred in 30/270 (11.1%) emergency admissions and 17/84 (20.2%) postoperative hyponatraemia cases. Most commonly seizures which occurred in 20 patients and confusion in three. While there were no reported cases of cerebral vasospasm or acute cerebral oedema, it is possible that some of the seizures may have been related to these complications of hyponatraemia but were not detected by the treating clinical teams.

The development of postoperative hyponatraemia was more likely to lead to a longer length of stay than seen in those admitted with hyponatraemia (15/84; 17.9% vs 9/270; 3.3%).

There were 12 deaths in patients with hyponatraemia. Seven deaths were discussed at local morbidity and mortality (or similar) meetings; reviewers did not identify any remediable factors related to the hyponatraemia in any of the deaths.

**Quality of care assessment and areas for improvement**

According to the clinicians who treated the patients there were areas regarding the management of the patient's hyponatraemia that could have been improved in 121/392 (30.9%) emergency patients and 25/106 (23.6%) postoperative patients.

Themes for improvement identified by the local clinicians mirrored those areas highlighted by the case reviewers and are summarised in Table 5.12. Further investigation was the biggest area for improvement for both emergency admissions and postoperative hyponatraemia.

| Table 5.12 Themes of areas for improvement in the care provided | Emergency admissions | Postoperative hyponatraemia |
|---|---|---|
| | Number of patients | Number of patients |
| Further investigation | 47 | 12 |
| Sodium checks | 22 | 7 |
| Treatment choice | 21 | 3 |
| Specialist input | 12 | 2 |
| Fluid status | 12 | 5 |
| Medicines review | 10 | 3 |

*Clinician questionnaire data*

The quality-of-care grading by the reviewers for emergency admission and postoperative hyponatraemia is shown in Figure 5.4. For both groups of patients, the main areas for improvement related to clinical issues rather than organisational issues (emergency admission: 133/265; 50.2% and postoperative: 51/83; 61.4%) (unknown for 5 and 1).

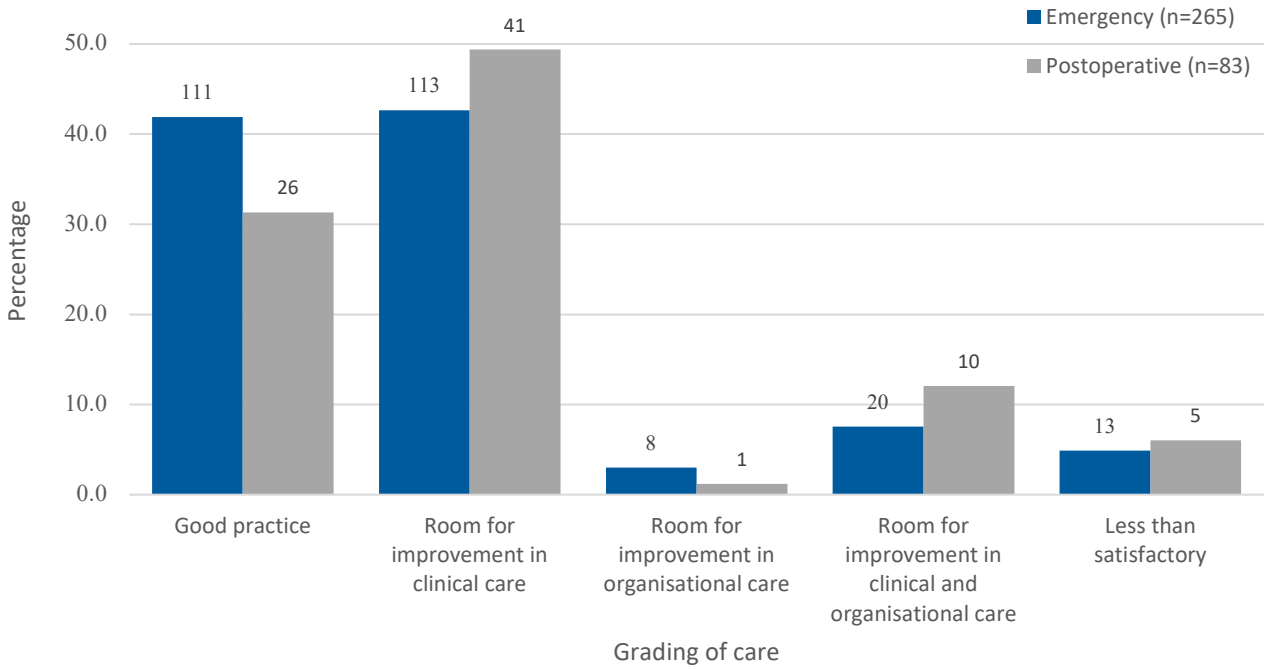


Figure 5.4 Assessment of overall quality of care for emergency admission and postoperative related hyponatraemia

*Reviewer assessment form data*

One area for improvement involves ensuring that follow-up on discharge is adequate and timely. In 35/270 (13.0) emergency admission-related hyponatraemia, the follow-up arranged on discharge was felt to be inappropriate. Themes identified included inadequate follow-up, inadequate instructions to the GP regarding repeat sodium measurements, and further investigation as outpatient required.

# CHAPTER 6: ASSESSMENT AND MANAGEMENT OF HYPERNATRAEMIA

(BACK TO CONTENTS)

Timely identification of poor oral intake may allow interventions to prevent the development and/or worsening of hypernatraemia.[4] The most common cause of hypernatraemia was poor oral intake (77/142 (54.2%) (T6.1). Notably, there were no patients in our study who had undergone pituitary surgery, which can be associated with acute onset arginine vasopressin deficiency (diabetes insipidus) and subsequent hypernatraemia.

| Table 6.1 Diagnoses associated with the hypernatraemia | Number of patients | % |
|---|---|---|
| Poor oral intake | 77 | 54.2 |
| Dementia/cognitive impairment | 44 | 31.0 |
| Acute kidney injury | 43 | 30.3 |
| Recent diarrhoea and/or vomiting | 18 | 12.7 |
| Hyperglycaemic hyperosmolar state | 17 | 12.0 |
| Mental health diagnosis | 13 | 9.2 |
| Significant brain injury | 11 | 7.7 |
| Other (specified) | 6 | 4.2 |
| Previous diagnosis of vasopressin related polyuria - diabetes insipidus | 4 | 2.8 |
| None documented | 19 | 13.4 |

*Clinician questionnaire data. Answers may be multiple; n=142*

In addition to fluid balance, it is essential that regular assessment of fluid status is undertaken to determine if the patient is becoming dehydrated. This would enable steps, such as increased fluid intake or stopping medicines that may worsen dehydration, to prevent hypernatraemia developing.

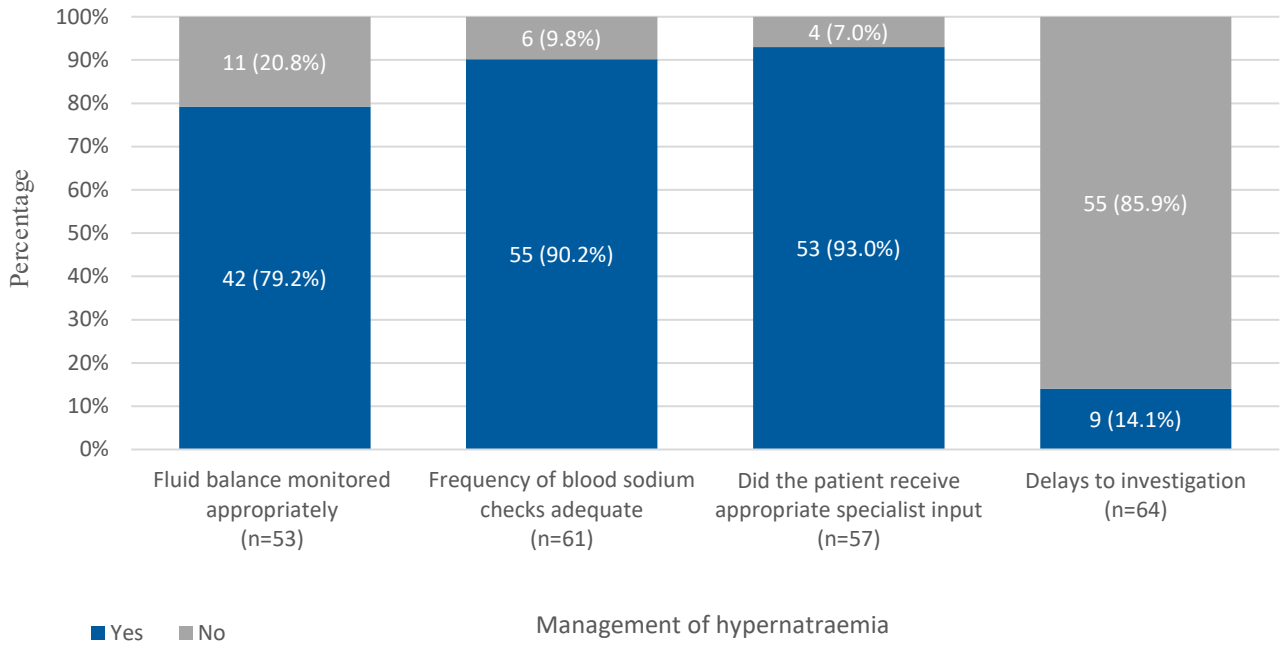


Figure 6.1 Frequency of appropriateness of key stages of management of hypernatraemia

*Reviewer assessment form data*

There were 11/53 (unknown in 12) patients with hypernatraemia where appropriate monitoring of fluid balance was not undertaken which if improved could have detected ongoing poor oral intake (F6.1). In addition to monitoring fluid balance, it is essential that patients who have or are at risk of hypernatraemia have regular assessment of their fluid status to determine if they are becoming dehydrated. This would enable steps, such as increased oral or intravenous fluid intake or stopping medicines that may worsen dehydration, to prevent hypernatraemia developing or worsening.

Even though appropriate monitoring of blood sodium levels was undertaken in 55/61 (unknown in 4) patients, there were delays in investigations in 55/64 (unknown in 1) (F6.1). Only 39/52 patients had both an appropriate fluid balance and sodium monitoring undertaken. If both documentation of fluid balance and sodium monitoring were improved, this could detect those patients at risk of developing or worsening hypernatraemia, allowing for earlier intervention and prevention.

Although this study focused on the care provided following admission to hospital, given the high proportion of patients with hypernatraemia admitted from a supported living environment (residential or care home) (T2.2) education on monitoring fluid intake in these settings could enable interventions to be delivered outside of hospital, potentially preventing hospital admissions due to hypernatraemia.

In total, 4/142 (2.8%) patients admitted with hypernatraemia had a previous diagnosis of arginine vasopressin disorder (diabetes insipidus). Not all were taking desmopressin, yet it is essential that patients take desmopressin on a regular basis, otherwise there is the risk that they will develop hypernatraemia due to an inability to maintain sufficient fluid intake.

Occasionally patients treated with desmopressin are advised to omit one dose of desmopressin a week to prevent over-treatment; this is on an individual patient basis and the day it is omitted can be varied to prevent the patient being inconvenienced by the increased urine output and associated thirst. This may be more difficult to do in a patient who does not have a sensation of thirst, due to brain injury (surgical or trauma related) or has a neurocognitive impairment where they do not remember to drink. Where patients do omit doses, they will be given advice by their endocrine team on what to do if they are passing large amounts of urine, have excessive thirst or there is an increase in ambient air temperature (for example during a heatwave).

The majority of hypernatraemia treatment involved rehydration (intravenous: 105 patients; oral/nasogastric rehydration: nine patients and combined oral/intravenous treatment: seven patients) (T6.2). In a small proportion (10: 7.0%) of patients there was no active treatment provided; the highest sodium values in these patients were 146–150 mmol/L: four patients; 151–155 mmol/L: three patients; 156–160 mmol/L: two patients; greater than 160 mmol/L: one patient and seven patients who had no active treatment died. For most patients, the treatment(s) administered were appropriate (61/65). Overall, the themes for improvement included not fluid restricting in hypernatraemia and appropriateness of fluid choice for IV rehydration.

| Table 6.2 Fluid used for intravenous rehydration in patients with hypernatraemia | Number of patients | % |
|---|---|---|
| Intravenous 0.9% sodium chloride | 65 | 45.8 |
| Intravenous 5% dextrose | 61 | 43.0 |
| Oral water | 18 | 12.7 |
| Nasogastric water | 17 | 12.0 |
| Not actively treated | 10 | 7.0 |
| Intravenous 0.45% sodium chloride | 7 | 4.9 |
| Hartmann's solution | 5 | 3.5 |
| Desmopressin | 2 | 1.4 |

*Clinician questionnaire data*

There were 44 patients with hypernatraemia who died, seven deaths were indirectly related. Five deaths were discussed at local morbidity and mortality (or similar) meetings and the themes identified were around failure to monitor sodium levels, renal function, and oral intake (appropriate fluid balance) and lack of senior review over a number of days during the admission.

Despite the lack of organisational focus on the assessment and management of hypernatraemia, 38/65 (58.5%) patients with hypernatraemia had their overall care graded as 'good practice' (F6.2). This was better than for those patients with emergency admission hyponatraemia (111/265; 41.9%) or postoperative hyponatraemia (26/183; 31.3%). This higher grading of 'good practice' may reflect that the diagnosis of the cause is usually less complex than hyponatraemia and does not require the interpretation of blood and urine osmolalities, urine sodium, other blood test or investigations.

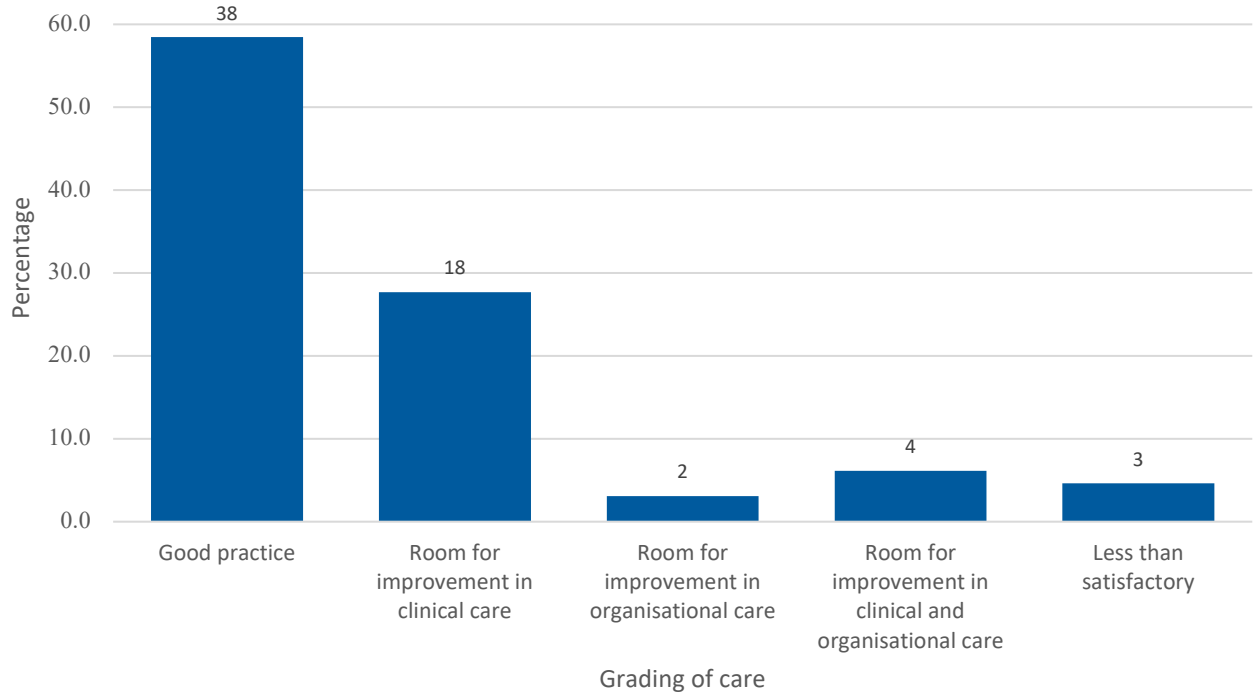


Figure 6.2 Overall quality of care for patients with hypernatraemia

*Reviewer assessment form data (n=65)*

# CHAPTER 7: SUPPORT FOR CLINICIANS TREATING PATIENTS WITH ABNORMAL BLOOD SODIUM LEVELS

(BACK TO CONTENTS)

**Training for clinical staff**

Training in hospitals on hyponatraemia and/or fluid management was more commonly provided to foundation doctors (97/115; 84.3% unknown in 41), although it was only part of mandatory training in 30/90 hospitals (unknown in 66). Training for other grades and specialties was less common (37/100; 37.0% unknown in 56). Training for staff on the management of hypernatraemia was only provided in 14/99 (14.1%) hospitals (unknown in 57). The high number of 'unknowns' suggests that the percentages may be a generous assessment of training provided.

Where training was provided it typically involved a single session with no assessment of its efficacy or impact. It is likely to have a greater impact on resident doctors, as well as other staff, when senior staff such as consultants, ward managers and matrons actively assess patients with hyponatraemia and manage treatment accordingly. It is important that, where appropriate, consultants and senior nursing staff are provided with access to regular training to maintain their own knowledge and skills in managing hyponatraemia and fluid management. Additionally, inter-professional simulation-based training may improve retention of the information and lead to overall improved patient care.

**Audit and quality improvement projects**

Regular audit of the management of hyponatraemia or hypernatraemia will determine where improvements are required, which can be addressed through quality improvement projects.

Quality improvement projects on hyponatraemia been undertaken in only 46/103 (44.7%) hospitals (unknown in 53), and only eight in hypernatraemia, in the previous five years. Where undertaken positive actions included dedicated training for resident doctors, hyponatraemia investigation order sets/bundles, hyponatraemia assessment and management guidelines and protocols, guidance on use of hypertonic saline solution and development of electronic referral systems to specialist services for advice/clinical reviews and updating local guidelines on hypernatraemia management.

**Specialist input and support**

The reviewers found that 203/248 (75.2%) patients with hyponatraemia either received appropriate specialist input or did not need it. The type of specialist input the patient received e.g. endocrinology, renal, critical care or other specialist input was not specified. Specialist advice for clinicians treating patients with hyponatraemia was available in 140/156 (89.7%) hospitals and was largely provided by services within the hospital or with a network.

The advice could be provided by more than one specialty; endocrinology provided most of the advice (126/140; 90.0%) while clinical biochemistry only provided advice in 24/140 (17.1%) hospitals (T7.1). This advice was available 24 hours a day in 71/140 (50.7%) hospitals; it was available in normal working hours (08:00 to 18:00) seven days a week in ten hospitals and in normal working hours only on weekdays in 49. However, in clinical practice, patients' hyponatraemia was often managed by

emergency medicine and acute/general physicians, rather than specifically by endocrinology or critical care teams.

| Table 7.1 Specialities providing advice on hyponatraemia | Number of hospitals | % |
|---|---|---|
| Endocrinology | 126 | 90.0 |
| Critical care | 49 | 35.0 |
| General medicine | 36 | 25.7 |
| Clinical biochemistry | 24 | 17.1 |
| Renal medicine | 15 | 10.7 |
| Other | 10 | 7.1 |

*Organisational questionnaire data. Answers may be multiple; n=140*

There are many physicians in other specialities who have developed expertise/specialist interests in the assessment and management of blood sodium abnormalities; it is important that each hospital provides guidance on who to contact locally to facilitate accessing this specialist advice. Timely specialist input can help with diagnosing the cause of the hyponatraemia and ensuring the appropriate and timely treatment is administered in the correct clinical environment.

There is generally less need for specialist input in patients with hypernatraemia as diagnosing the underlying cause and determining the appropriate treatment option(s) is less challenging than in patients with hyponatraemia. In this study 53/65 (81.5%) patients had appropriate specialist input or did not require it.

Specialist advice for clinicians treating patients with hypernatraemia was available in 126/156 (80.8%) hospitals and largely provided by services within the hospital. This advice was only available 24 hours a day in 67/126 (53.2%) hospitals, and it was available in normal working hours (08:00 to 18:00) seven days a week in seven and in normal working hours only on weekdays in 44. Like hyponatraemia, specialist advice on hypernatraemia was predominately provided by endocrinologists.