

Sodium Augmentation Prediction Model in Neurocritically Ill Patients

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Research Article

Keywords:

Posted Date: October 4th, 2026

DOI: <https://doi.org/10.21203/rs.3.rs-11052886/v1>

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Abstract

Background/Objective:

Hypertonic saline (HTS) is a common treatment for intracranial hypertension in neurocritically ill patients with cerebral edema, creating an osmotic shift to pull fluid from the intracranial space. Currently, clinicians lack tools to accurately predict the effects of HTS on serum sodium when augmenting sodium beyond eunatremia.

Methods

This was a single center retrospective study of neurocritically ill adults who received ≥ 2 boluses and/or continuous infusion of HTS. Exclusion criteria included vulnerable populations, preexisting hyponatremia (baseline sodium ≤ 135 mEq/L), or no sodium levels obtained following HTS administration. The purpose of this study was the generation of a serum sodium prediction model using a Random Forest Model that was tuned to hyperparameters via grid search and used 5-fold group-vfold cross-validation by patient to prevent leakage. Performance was assessed on a held-out test set of patients using root mean squared error (RMSE), mean absolute error (MAE), R^2 , calibration (slope/intercept), and residual diagnostics. Secondary safety outcomes included rates of hyperchloremia (> 115 mEq/L), acute kidney injury, and the need for renal replacement therapy.

Results

Amongst the 416 patients identified for the study, 7 were excluded for having no serum sodium levels, and a total of 409 patients with 7620 serum sodium levels were included. The first iteration of the model achieved RMSE = 0.573 mmol/L (95% CI: 0.39–0.73), MAE = 0.105 mmol/L (95% CI: 0.08–0.13), $R^2 = 0.994$ (95% CI 0.991–9.997) on the test set. Most recent sodium value, amount of sodium (in mEq) administered over the previous 24 hours, and time since last sodium lab were identified as the parameters with the most effect on the model's outcomes.

Conclusion

This study developed a sodium prediction model to predict the effects of hypertonic saline on serum sodium levels for sodium augmentation in neurocritically ill patients.

Introduction

Cerebral edema is a pathological accumulation of fluid in the brain that can arise secondarily from inflammatory processes, vascular endothelial changes, or ischemia.¹ Cerebral edema can cause elevated intracranial pressure (ICP), which has been identified as a predictor of poor outcomes in acute

brain injury.¹ Maintaining ICP within normal range (7–15 mmHg) helps to promote adequate brain oxygen delivery, avoid further worsening or development of injury, and prevent herniation.¹

The currently available guidelines and literature describe a variety of treatment modalities available to manage cerebral edema and reduce ICP. Hyperosmolar agents, including hypertonic saline (HTS), are used to create an osmotic shift, pulling fluid from the brain to reduce swelling. The 2020 Neurocritical Care Society Guideline for the Acute Treatment of Cerebral Edema recommends the use of HTS as the preferred hyperosmolar agent for ICP management in patients with traumatic brain injury and intracerebral hemorrhage.¹

While HTS is commonly used in neurocritically ill patients, the choice of HTS concentration, formulation, and administration varies significantly. Several commercial formulations of HTS exist, including 3%, 5%, and 23.4%, while additional concentrations (e.g. 2%, 7.5%) can be compounded. Since excessive hyperchloremia is associated with significant metabolic derangements and risk of renal injury^{1,2}, some institutions compound a chloride-sparing solution using sodium acetate (commonly referred to as buffered HTS).^{3,4} The ACETatE Trial (2020) compared 23.4% HTS with buffered HTS in patients with subarachnoid hemorrhages and found that buffered HTS was associated with significantly lower chloride burden and acute kidney injury (AKI).⁴ Lower concentrations of HTS can be administered both as bolus doses and continuous infusions. Wahdan et al. found that 3% HTS boluses produced similar ICP reductions compared to continuous infusions but resulted in more rapid serum sodium elevations at 6–12 hours.⁵ Caution is required when augmenting sodium as excessive hyponatremia (beyond 155–160 mEq/L) is associated with serious adverse effects including seizures, coma, and death.^{1,6}

While several equations have been proposed to estimate the effect of sodium-containing fluids on serum sodium (Table 1), there is a lack of literature evaluating the effect of HTS administration when augmenting beyond eunatremia. The Edelman equation was developed in 1958 to describe the concentration of sodium in plasma as a reflection of the ratio of total body solutes (sodium and potassium) to total body water.^{7,8} However, this equation utilizes a generalized slope and y-intercept as constants, which can fluctuate in the setting of extreme serum sodium, body weight, and edema.^{8,9} Rose et al. simplified this equation for more clinical applicability but is still limited by the same assumptions.¹⁰ The Adroque-Madias formula is a derivative of the Edelman and Rose equations.¹¹ The formula's algebraic alterations offer a prediction in the change in serum sodium to be expected from administration of 1 L of intravenous fluid, while considering an individual's baseline serum sodium levels.^{11,12} However, the formula is limited to 1 L volumes of fluid; if other volumes are administered, the formula will provide inaccurate estimations on serum sodium levels.^{7,11,12} In addition, the formula treats the body as a closed system, failing to account for renal function, diuresis, and electrolyte losses.^{7,11} Over time, modernized extensions of the Edelman equation were developed, such as the Nguyen-Kurtz and Barsoum-Levine equations, but their clinical applicability remain limited as they omit physiologic shifts in body water and electrolytes.^{13,14,15}

Table 1
Sodium predictive equations

Edelman Equation	$Na_{pw} = 1.11 \times \frac{Na_e}{TBW + 25.6}$
Rose Equation	$Na_{serum} = \frac{Na_e}{TBW + K_e}$
Adroque-Madias Formula	$\Delta Na = \frac{Na_{pw} - K_{inf}}{TBW + 1} - Na_{serum}$
Barsoum-Levine Equation	$Na_2 = \frac{(TBW \times Na_1) + [V_{inf} \times (Na_{inf} + K_{inf})] - [V_0 \times (Na_0 + K_0)]}{TBW + \Delta V}$
Nguyen-Kurtz Equation	$Na_2 = \frac{[(Na_0 + 238) \times TBW] + [1.03 \times (Na_{inf} + K_{inf}) - (Na_0 + K_0)]}{TBW + \Delta V}$
<p>K_e = total body exchangeable potassium; K_{inf} = potassium concentration of infusate; Na_e = total body exchangeable sodium; Na_{inf} = sodium concentration of inufsate; Na_{pw} = plasma water sodium concentration; Na_{serum} = serum sodium concentration; Na_1 = initial serum sodium concentration; Na_2 = predicted serum sodium concentration; TBW = total body water; V_{inf} = volume of infusate; V_0 = volume of fluid output/loss</p>	

The purpose of this study was to develop a predictive sodium augmentation model in neurologically injured patients who require multiple doses of HTS, including bolus doses and continuous infusions. The goal is to ultimately implement the model into a tool for clinicians to guide HTS administration to optimize cerebral edema management while avoiding excessive hyponatremia and renal injury.

Methods

Study Design and Setting

This was a retrospective observational single center study at a large academic medical, level I trauma, and comprehensive stroke center that evaluated patients admitted to the neurocritical care unit. This study was approved by the Brown University Health institutional review board [BUH3-2330813] with waiver for patient consent.

Participant Selection

Study participants included within the study were identified through the electronic medical record (EMR) and were admitted between January 1, 2022, and July 31, 2025. Patients were included if they were neurocritically injured adults (≥ 18 years of age) without baseline hyponatremia (serum sodium < 135 mEq/L) who received ≥ 2 boluses or a continuous infusion of 3% HTS, 23.4% HTS, or buffered HTS. Patients were excluded if no serum sodium levels were obtained following HTS administration or if they were pregnant or incarcerated.

Hypertonic Saline Exposure

Available hypertonic saline formulations include 3% HTS, 23.4% HTS, and buffered HTS. Administration of 3% HTS and buffered HTS could be either an intermittent bolus (250 mL [128 mEq sodium] infused intravenously over 15 minutes) or as a continuous infusion (0.513 mEq/mL). More concentrated 23.4% HTS could be administered as a 30 mL intravenous bolus (120 mEq sodium) over 15 minutes. All HTS boluses and continuous infusions were delivered via an electronic pump with central venous access being the preferred route of administration. Patients' serum sodium levels were drawn every 6 hours to ensure sodium augmentation was within the provider-driven target serum sodium range.

Data Collection

The primary objective of this study was to develop a predictive model for serum sodium augmentation after administration of ≥ 2 boluses of 3% HTS, 23.4% HTS, or buffered HTS, and a continuous infusion of 3% HTS or buffered HTS. Patient-specific variables were obtained from the EMR, including baseline characteristics, and all recorded serum sodium concentrations from admission through 72 hours following the final HTS administration. All HTS administration data was obtained from the electronic medication administration record. For secondary outcome assessment, patients' baseline and serial serum creatinine and chloride levels were obtained, as well as any renal replacement therapy (RRT) within 7 days of HTS administration.

Predictive Model Development

Feature Engineering

Medication administration records were mapped to sodium exposures by converting ordered volumes and infusion rates into milliequivalents of sodium based on product-specific concentrations. For each observation, sodium exposure was calculated over the 6-, 12-, and 24-hour periods prior to the serum sodium measurement. Both bolus administrations and infusion rates were accounted for in this sodium exposure calculation.

Longitudinal trends were captured for each sodium level measurement, using the two most recent sodium measurements, the change in time from the immediately preceding measurement, and elapsed time since prior laboratory draw were included. Patient-level co-variables included body weight, AKI status, concurrent RRT, sex, race, and ethnicity. Feature construction was restricted to data prior to the prediction time to prevent information leakage.

Model Selection, Implementation, and Development

A random forest regression model was selected as the primary machine learning approach given potential nonlinear relationships between prior sodium measurements, recent sodium exposure, and patient-level factors. A priori specification of interaction terms and functional forms is not required and is suited to real-world, observational, longitudinal clinical data with correlated predictors and irregular measurement timing.

Data was split at the patient level to ensure robust evaluation and avoid within-patient leakage. Patients were randomly assigned in an 80/20 split to training or testing sets, respectively, with all observations from a given patient confined to a single split. Model tuning and internal validation were performed using a five-fold cross-validation grouped by patient identifier. R/R Studio 4.5.2 (Vienna, Austria) was utilized for model implementation using the *ranger* engine within the *tidymodels* framework.

Evaluation Strategy

Model performance was assessed on the held-out test patients using root mean squared error (RSME), mean absolute error (MAE), and coefficient of determination (R^2). Bootstrap resampling stratified by patients was employed to calculate 95% confidence intervals. A linear regression of observed versus predicted sodium values was plotted and evaluated visually to evaluate calibration. Residual distributions and residuals versus fitted values were examined to assess model adequacy. Sub-group analyses were performed to evaluate performance for patients with AKI, on RRT, and across baseline sodium ranges.

Secondary Outcomes

The secondary safety outcomes included incidence of hyperchloremia (> 115 mEq/L), rates of AKI based on the KDIGO 2012 definition (increase in SCr by ≥ 0.3 mg/dL within 48 hours, increase in SCr by ≥ 1.5 times baseline, and a mean urine volume of < 0.5 mL/kg/h based on ideal body weight for ≥ 6 hours), and the rates of RRT within 7 days of HTS administration.¹⁶ These safety outcomes were selected based on previously reported electrolyte and renal safety concerns associated with hypertonic saline.

Results

Study Cohort

A total of 416 neurocritically ill adult patients who received HTS were identified. Of these, 7 patients were excluded due to the absence of post-administration serum sodium measurements, resulting in 409 patients included for analysis (Fig. 1). Amongst the included cohort, a total of 3,240 HTS administrations were recorded, and 7,620 serum sodium measurements were collected.

Baseline characteristics of the cohort are included in Table 2. The study population had a mean age of 60 years old with 57% being male. The most common admission diagnoses were non-traumatic

intracerebral hemorrhage, acute ischemic stroke, and traumatic brain injury. The most frequently administered HTS was a continuous infusion of 3%.

Table 2
Baseline characteristics

Characteristic	Study Cohort (N = 409)
Age [yr], mean (SD)	60 (17.26)
Male Sex, n (%)	233 (57.0)
Weight [kg], mean (SD)	80.05 (21.16)
Race, n (%)	
White or Caucasian	291 (71.1)
Black or African American	29 (7.1)
Asian	17 (4.2)
American Indian or Alaska Native	2 (0.5)
Native Hawaiian or Other Pacific Islander	1 (0.2)
Other	62 (15.2)
Unknown	4 (1.0)
Patient Refused	3 (0.7)
Ethnicity, n (%)	
Hispanic or Latino	69 (16.9)
Unknown	6 (1.5)
Patient Refused	6 (1.5)
Admission Diagnosis, n (%)	
Non-Traumatic Intracerebral Hemorrhage	136 (33.3)
Acute Ischemic Stroke	122 (30.0)
TBI	75 (18.3)
Aneurysmal Subarachnoid Hemorrhage	33 (8.1)
Brain Mass	30 (7.3)
Cerebral Venous Sinus Thrombosis	4 (1.0)
Meningitis	4 (1.0)
Other	5 (1.2)
Total Administrations, mean (SD)	7.58 (7.24)
Hyperosmolar Agent, n (%)	

Characteristic	Study Cohort (N = 409)
Hypertonic Saline 23.4%, Bolus	140 (34.2)
Hypertonic Saline 3%, Bolus	190 (46.5)
Hypertonic Saline 3%, Continuous Infusion	284 (69.4)
Sodium Chloride 1.5% + Sodium Acetate 1.5%, Bolus	10 (2.4)
Sodium Chloride 1.5% + Sodium Acetate 1.5%, Continuous Infusion	36 (8.8)

Predictive Model

The first iteration of the model achieved a root mean squared error (RMSE) of 0.573 mEq/L (95% CI: 0.39–0.73), a mean absolute error (MAE) of 0.105 mEq/L (95% CI: 0.08–0.13) and an R^2 of 0.994 (95% CI: 0.991–0.997) on the test set. Figures 2 and 3 illustrate the linear plot of observed versus predicted sodium values and residual distributions (defined as the per patient difference between actual and predicted sodium values), respectively. Model performance assessed with the held-out test patients is demonstrated in Fig. 4.

Most recent sodium value, amount of sodium (in mEq) administered over the previous 24 hours, and time since last sodium lab were identified as the parameters with the most effect on the model's outcomes.

Secondary Outcomes

Secondary endpoints included incidence rates of hyperchloremia, AKI, and delayed RRT. Hyperchloremia, defined as a serum chloride > 115 mEq/L, occurred in 74.1% of patients, and AKI was observed in 70.9% of patients. Delayed RRT was required in 2.9% of the cohort.

Discussion

This is the first predictive model that aims to quantify the effects of sodium augmentation with various formulations of HTS on serum sodium levels in neurologically injured patients. Our model focuses on neurocritically ill patients without baseline hyponatremia requiring sodium augmentation for treatment of ICP caused by cerebral edema. Although there are other available equations that assess the effect of hypertonic solutions and normal saline on serum sodium, they may not adequately account for the multifactorial factors influencing the serum sodium response in neurocritically ill patients. The Edelman equation and the Adroque-Madias formula were studied in a variety of dysnatremias, however, their studied patient baseline characteristics varied.^{7,9,11} The Edelman equation assessed patients with disease states affecting body water and sodium homeostasis, such as diseases of the heart, liver, or central nervous system.¹¹ While the Adroque-Madias formula was validated in patients with both hyponatremia and hypernatremia experiencing fluid and electrolyte abnormalities within different

disease states,¹¹ whereas our patient population were baseline eunatremic. In comparison to the Adrogue-Madias formula, our model does not limit serum sodium assessment to a specific volume of infused fluids, allowing for a more variable assessment, lending to its clinical applicability. In addition, our predictive model was generated using random forest regression, compared to the linear regression model used by the Edelman equation and Adrogue-Madias formula.

Our predictive model showed an overall high accuracy, demonstrated through a MAE of 0.105 mEq/L. The RSME score of 0.573 mEq/L was much larger than the MAE, which is evidenced by sparsity within the data set that can be attributed to the higher variation in sodium prediction values once the serum sodium is > 165 mEq/L. However, the clinical impact of these errors is likely limited as these sodium values are significantly higher than utilized in practice for ICP management. The R^2 of 99.4% depicts strong model performance, though it may partially reflect potential overfitting of the model to the internal patient population assessed and should be interpreted cautiously pending external validation.

Evaluating secondary safety outcomes, the incidence of AKI and hyperchloremia were identified in > 70% of the patients included within the study. The rate of AKI was significantly higher amongst our patient population when compared to the COBI trial where AKI occurred in 2.2–4.4% of patients.¹⁷ Some of this difference can be attributed to the different definitions of AKI utilized – the COBI trial utilized a more restrictive definition of KDIGO stage ≥ 2 (serum creatinine 2-2.9 times baseline and/or urine output < 0.5 mL/kg/h for ≥ 12 hours), while our study had a broader criteria that likely captured more fluctuations in serum creatinine. It is also unclear the duration and impact of the serum creatinine elevations, as the rate of renal replacement therapy was significantly lower at 2.9%.

Several studies outline the relationship between HTS and hyperchloremia. Rhia et al. (2018) assessed hyperchloremia effects on in-hospital mortality in intracranial hemorrhage patients who received a continuous infusion of 3% HTS, finding that rates of hyperchloremia increased rates of mortality.¹⁸ The ACETatE trial identified that the rates of AKI when using a buffered HTS fell from 53.3% down to 11.8%.⁴

This study used real-world data to create a predictive model in a high-acuity patient population, enhancing clinical utility. It incorporated a broad range of clinically relevant variables known to impact serum sodium (e.g. most recent sodium value, time since last sodium level, and the amount of sodium administered over the previous 24-hour period), allowing for a more comprehensive assessment of contributing factors. However, we were unable to account for all potential confounding variables, such as medications prepared in sodium chloride, diuresis, hyperglycemia, or other disease states that may alter serum sodium levels. In addition, data collection relied on the accuracy and consistency of documentation in the EMR, which may be variable in practice.

This model was only assessed within one institution; to account for potential overfitting of the data, external validation is still required to ensure our model is applicable across larger populations. Lastly, the use of HTS, dosing strategies, formulation selection, and serum sodium monitoring were non-protocolized and provider-specific and can vary from institution to institution.

Future Directions

Future studies should assess the external validity of this sodium augmentation prediction model in a broader neurocritically ill patient population to account for varying treatment practices. In addition, the model should be evaluated in specific patient populations with altered sodium physiology, such as pediatrics, pregnancy, and pre-existing renal replacement therapy. The ultimate goal is to implement this model as a clinical decision support tool for providers to predict sodium changes during augmentation to optimize hyperosmolar therapy and reduce adverse events.

Conclusion

This sodium augmentation predictive model has an overall high accuracy in predicting serum sodium levels after ≥ 2 doses and/or continuous infusion of HTS. It can serve as a clinical decision support tool for providers weighing the risks and benefits of giving HTS in neurocritically injured patients.

Declarations

This manuscript complies with all instructions per the *Neurocritical Care* Instructions for Authors. All authorship requirements have been met, and all authors have reviewed and approved the final manuscript. Individual author contributions are listed below. This manuscript has not been published elsewhere and is not under consideration with any other journal. This study was approved by the Brown University Health Institutional Review Board [BUH3-2330813] with waiver for patient consent and all requirements set forth and maintained proper protocol procedures required for retrospective study designs. All authors have no conflicts of interest or sources of funding to disclose. The EQUATOR Checklist has been completed and submitted. Artificial intelligence was not used in any aspect of the production of this study.

Author Contributions:

AF: Methodology, data curation, formal analysis, writing – original draft, writing – review & editing

DT: Methodology, data curation, formal analysis, software, resources, validation, visualization, writing – original draft, writing – review & editing

SW: Conceptualization, resource

MP: Conceptualization, methodology, supervision, writing – review & editing

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Figures

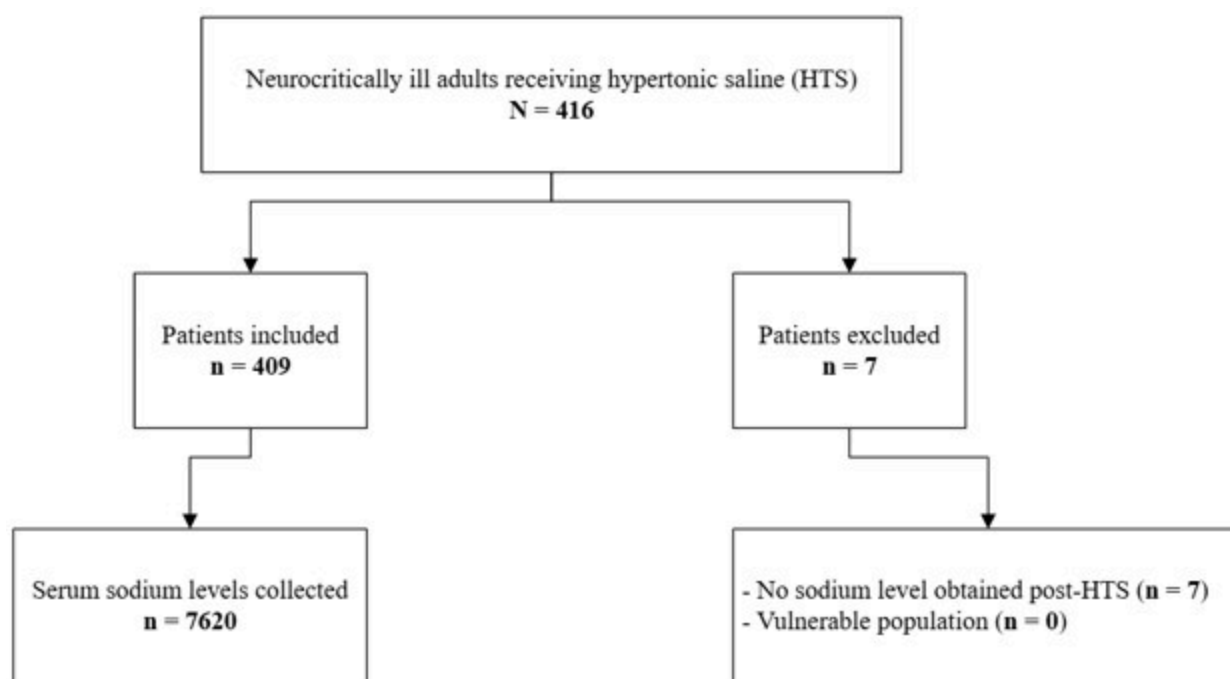


Figure 1

Flowchart of study inclusion and exclusion criteria and patient selection.

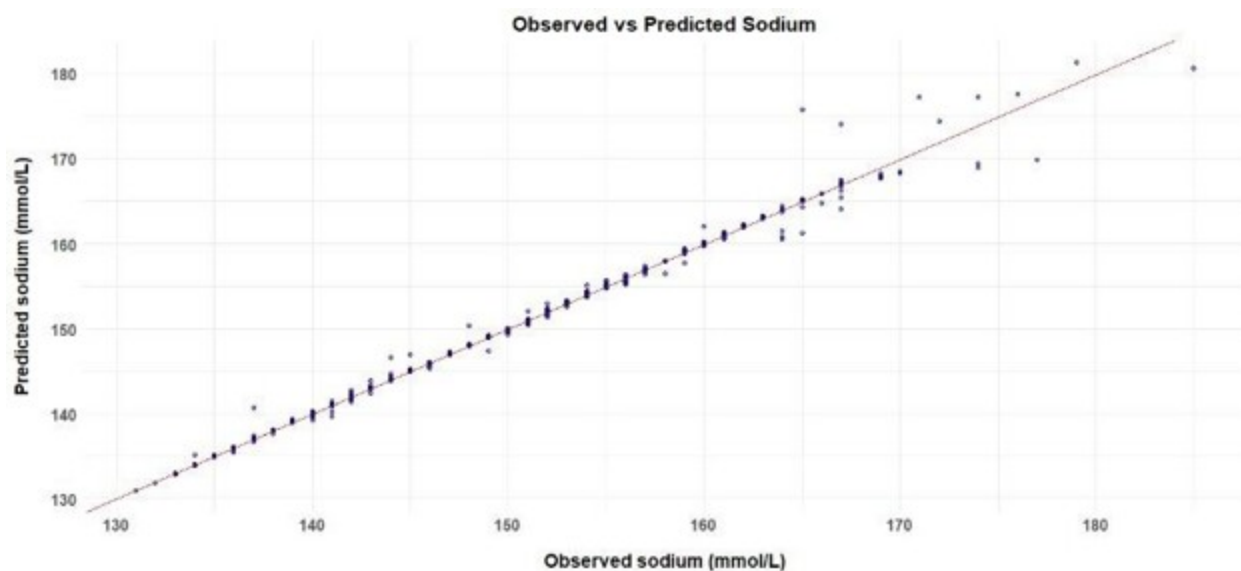


Figure 2

Linear plot of predicted serum sodium and observed serum sodium, with line of best fit ($R^2 = 0.994$, 95% CI: 0.991-9.997).

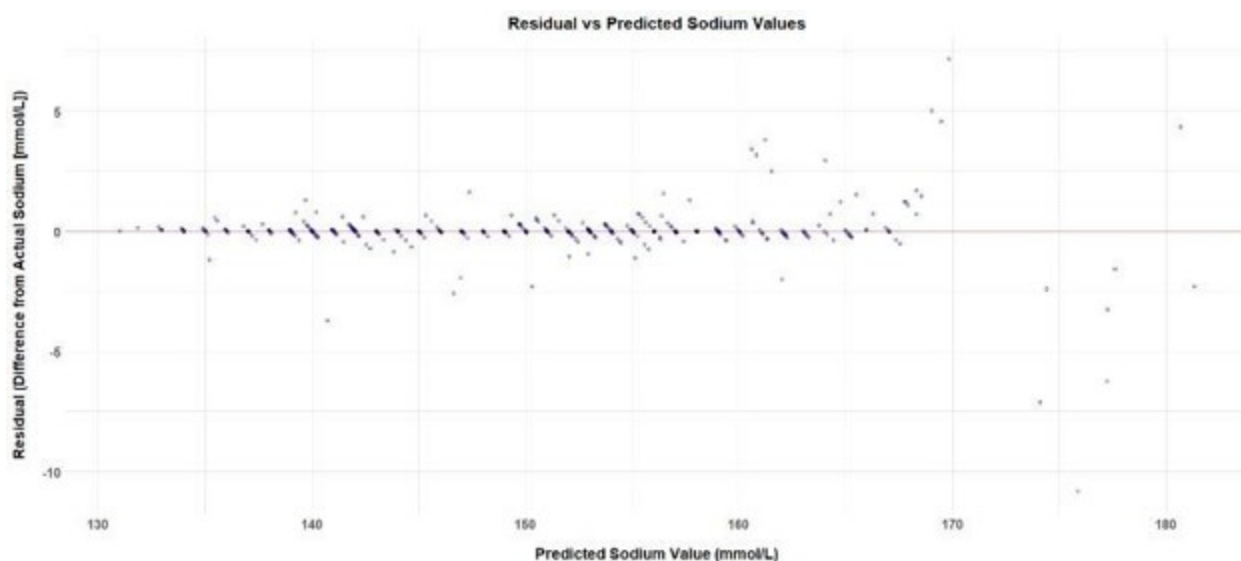


Figure 3

Plot of predicted serum sodium and residual (defined as difference from actual serum sodium value).

Sodium Over Time: Observed vs Predicted

Faceted by patient encounter (de-identified)

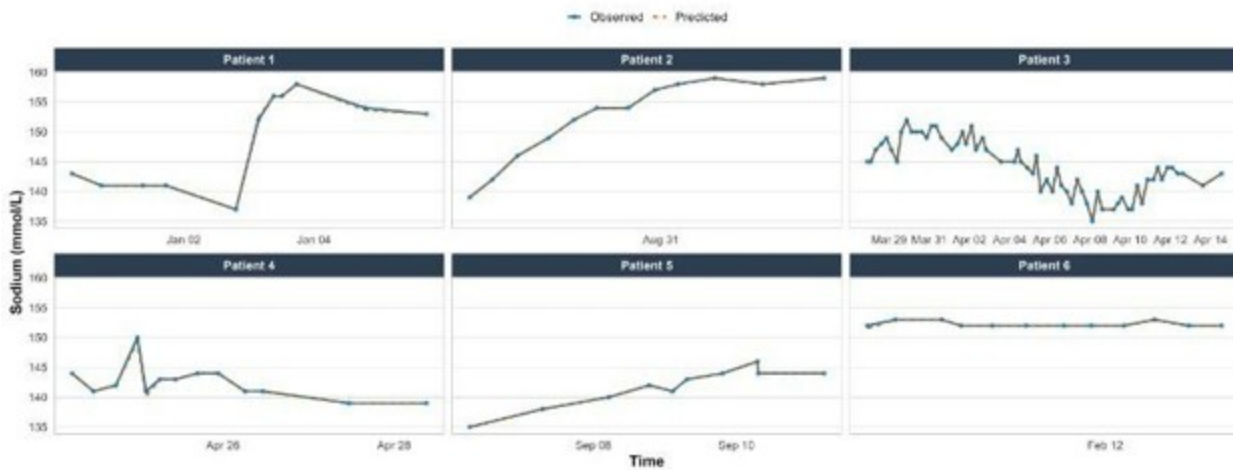


Figure 4

Model performance assessment with six held-out test patients. Dotted line indicates predicted sodium values and solid line indicates observed sodium values.

Supplementary Files

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- [EQUATORChecklist.docx](#)