

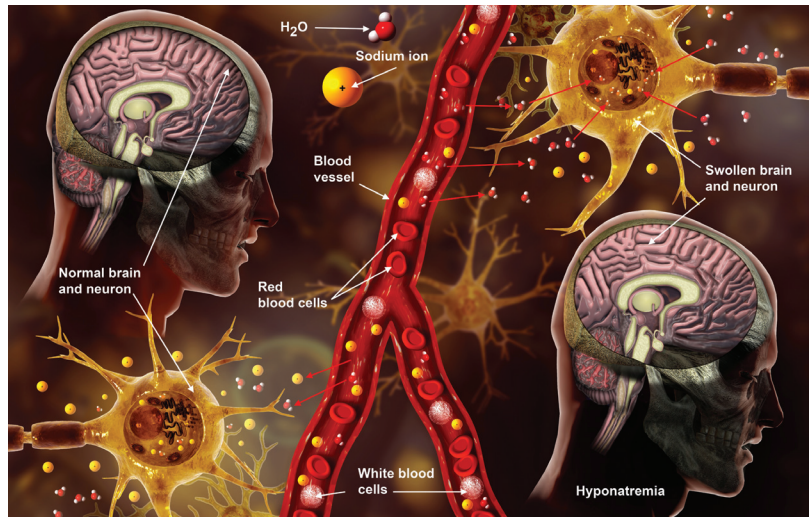
Hyponatremia: Beer Potomania and Overcorrection

Q Recently, we had a patient admitted for hyponatremia with a serum sodium level of 117 mEq/L. One of the hospitalists mentioned “beer potomania” in the differential. Not wanting to look dumb, I just agreed. What is beer potomania, and how is it related to low serum sodium?

Potomania is the excessive consumption of alcoholic beverages; *beer potomania* is used to refer to a dilutional hyponatremia caused by excessive consumption of beer.¹ First recognized in 1971, this cause of hyponatremia is not the most common but should be in the differential if the patient is a heavy alcohol imbiber who presents with encephalopathy and low serum sodium.

When considering this diagnosis, keep in mind that hyponatremia is common among chronic alcoholics and can be due to conditions such as cirrhosis, congestive heart failure, syndrome of inappropriate antidiuretic hormone (SIADH) secretion, and hypovolemia. Less common but still belonging in the differential are pseudohyponatremia secondary to alcohol-induced severe hypertriglyceridemia and cerebral salt wasting syndrome.^{2,3}

Beer potomania usually manifests as altered mental status,



Hyponatremia is generally defined as a serum sodium level of less than 135 mEq/L. When it occurs, water is drawn into the cells, causing the brain to swell (shown at right, in contrast to a normal brain at left).

weakness, and gait disturbance with an average serum sodium concentration of 108 mEq/L.³ Other abnormal lab results consistent with this diagnosis include hypokalemia (mean potassium, 3 mEq/L) and low blood urea nitrogen and urine sodium levels.^{2,3} Another fairly consistent finding is a recent personal history of binge drinking (more than about 5 L, or 14 cans of beer, in 24 hours) and/or history of illness (vomiting, diarrhea) that predisposed the patient to a rapid drop in serum sodium levels.²

Based on the information presented thus far, you may ask, “Why haven’t I seen this diag-

nosed more often? There are a lot of beer bingers out there!” Good question. Let’s review the pathophysiology of beer potomania. When patients have poor protein and solute (food, electrolytes) intake, they can experience water intoxication with smaller-than-usual volumes of fluid. The kidneys need a certain amount of solute to facilitate free water clearance (the ability to clear excess fluid from the body). A lack of adequate solute results in a buildup of free water in the vascular system, leading to a dilutional hyponatremia.³

Free water clearance is dependent on both solute excretion and the ability to dilute urine. Someone consuming an average diet will excrete 600 to 900 mOsm/d of solute. This osmolar load includes urea generated from protein (10 g of protein produces about

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Credit: Carol and Mike Werner / Science Source

50 mOsm of urea), along with dietary sodium and potassium. The maximum capacity for urinary dilution is 50 mOsm/L. In a nutritionally sound person, a lot of fluid—about 20 L—would be required to overwhelm the body's capacity for urinary dilution.²

However, when you don't eat, the body starts to break down tissue to create energy to survive. This catabolism creates 100 to 150 mOsm/d of urea, allowing you to continue to appropriately excrete a moderate amount of fluid in spite of poor solute intake ... as long as you are not drinking excessive amounts of water.⁵

Alcoholics get a moderate amount of their calories via beer consumption and do not experience this endogenous protein breakdown or its resultant low urea/solute level. With low solute intake, dramatically lower fluid intake (about 14 cans of beer) will overwhelm the kidneys' ability to clear excess free water in the body.² Fortunately, most heavy beer drinkers continue to eat at least modestly, which is sufficient to avoid this rare type of hyponatremia. Chronic alcoholics who go on a drinking binge beyond their normal baseline alcohol consumption, or who develop a flulike illness that causes electrolyte depletion (via diarrhea or vomiting), are at higher risk for beer potomania.

Q A clinic patient of mine was recently admitted to the hospital with hyponatremia (serum sodium, 115 mEq/L). She was treated with 2 L of normal saline and discharged home 48 hours later, at her baseline mental status with a serum sodium level of 132 mEq/L. Two days

later, she was readmitted for mental status changes, and MRI showed brain swelling. The neurologist stated this was a result of the initial treatment for her hyponatremia. How is this possible?

The cause-and-effect relationship between rapid correction of chronic hyponatremia and subsequent development of neurologic problems was discovered in the late 1970s. Central pontine and extrapontine myelinolysis (known as *osmotic demyelination syndrome* or ODS) is a neurologic condition that can occur from rapid sodium correction. It is diagnosed by MRI, which shows hyperintense lesions on T2-weighted images. Clinical signs include upper motor neuron signs, pseudobulbar palsy, spastic quadriparesis, and mental status changes ranging from mild confusion to coma.²

Treatment for hyponatremia should be guided by symptom management.^{2,3} If a patient is asymptomatic, a simple and effective strategy is to keep NPO for 24 hours, except for medications. Simple food and fluid restriction will likely increase the serum sodium level because of obligate solute losses and urinary electrolyte free water loss.^{2,4} While the first instinct is to feed these patients, as they often appear malnourished, this can cause a solute load leading to a too-rapid sodium correction. After 24 hours, if intake restriction is not effective, use 0.5% normal saline but with limited dosing orders, as usual saline dosing can cause too rapid a correction.²

For symptomatic patients (confusion, seizures, coma), the goal is to initially elevate sodium by 1 to 2 mEq/L per hour for the



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serve as an advisory resource for the NKF, nurse practitioners, physician assistants, clinical nurse specialists, and the community in advancing the care, treatment, and education of patients with kidney disease and their families. CAP is an advocate for professional development, research, and health policies that impact the delivery of patient care and professional practice. For more information on NKF-CAP, visit www.kidney.org/CAP

first two to three hours. Do not exceed 10 mEq/L in 24 hours or 18 mEq/L in 48 hours. Exceeding these limits puts patients at high risk for ODS. In fact, even when staying within these parameters, there is some risk for overcorrection. It is always better to go slowly.^{2,3}

In the patient with hyponatremia due to low solute intake (eg, beer potomania), diuresis can start spontaneously after a period of food and fluid restriction. It can also be initiated with just a small amount of solute. For example, administering an IV antibiotic with a base solution of 100 mL of normal saline or a "banana bag" (an IV solution containing 0.5 to 1 L of normal saline with multivitamins/minerals that cause the fluid to be yellow) can produce several liters of diuresis.² Once you open the floodgate, you can unintentionally cause too-rapid correction that could lead to ODS.

In chronic hyponatremic patients, low antidiuretic hormone (ADH) levels are often found; thus when a solute is introduced, there is little ADH in the system to pro-

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Why You Shouldn't Start β -Blockers Before Surgery

A new meta-analysis finds that initiating β -blockers before surgery increases patients' risk for death.

Anne Mounsey, MD, Jodi M. Roque, MD, Mari Egan, MD

PRACTICE CHANGER

Do not routinely initiate β -blockers in patients undergoing intermediate- or high-risk noncardiac surgery. β -Blockers appear to increase the 30-day risk for all-cause mortality.¹

STRENGTH OF RECOMMENDATION

A: Based on meta-analysis of nine randomized controlled trials (RCTs).¹

ILLUSTRATIVE CASE

A 67-year-old woman with diabetes, hypertension, and hyperlipidemia presents for evaluation prior to a total hip arthroplasty. She is not taking a β -blocker. Should you prescribe one?

Current guidelines from the American College of Cardiology Foundation (ACCF) and the American Heart Association (AHA) recommend starting β -blockers to prevent cardiac events in patients about to undergo intermediate- or high-risk surgery or vascular surgery who have a history of inducible ischemia, coronary artery disease (CAD), or at least one risk factor for CAD.² However, the majority of the evidence for these guidelines, which were published in 2009 and are

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in the process of being updated, came from the DECREASE (Dutch Echocardiographic Cardiac Risk Evaluation Applying Stress Echocardiography) trials. These trials have been discredited due to serious methodologic flaws, including falsified descriptions of how outcomes were determined and fictitious databases.³

A new meta-analysis conducted by Bouri et al¹ that excluded the DECREASE trials found that, although preoperative β -blockers reduce the rate of certain nonfatal outcomes, they increase the risk for death and stroke.

STUDY SUMMARY

Preop β -blockers do more harm than good

Bouri et al¹ conducted a meta-analysis of published RCTs evaluating preoperative β -blockers versus placebo for patients undergoing noncardiac surgery. Of the 11 studies that met eligibility criteria, two were the discredited DECREASE trials. Thus, Bouri et al¹ analyzed nine high-quality RCTs that included 10,529 patients.

Most studies included patients undergoing vascular surgery. Some studies also included intra-abdominal, intrathoracic, neurosurgical, orthopedic, urologic, and gynecologic surgeries. β -Blockers were started no more than a day before surgery and were discontinued at hospital discharge or

up to 30 days postop. Metoprolol was used in five trials, bisoprolol in one trial, atenolol in two trials, and propranolol in one trial. The primary endpoint was all-cause mortality within 30 days.

A total of 5,264 patients were randomly assigned to receive β -blockers and 5,265 to placebo. There were 162 deaths in the β -blocker group and 129 deaths in the placebo group. Patients who received β -blockers had a 27% increased risk for all-cause mortality (risk ratio [RR] = 1.27). The number needed to harm was 160.

Six of the studies also evaluated rates of nonfatal MI, nonfatal stroke, and hypotension. β -Blockers lowered the risk for nonfatal MI (RR = 0.73) but increased the risk for nonfatal stroke (RR = 1.73) and hypotension (RR = 1.51).

This meta-analysis was dominated by the 2008 Peri-Operative ISchemic Evaluation (POISE) trial, an RCT that compared placebo to extended-release metoprolol (100 mg 2 to 4 h before surgery, followed by 200 mg/d for 30 d), in 8,351 patients with, or at risk for, atherosclerotic disease.⁴ While β -blockers reduced the risk for MI and atrial fibrillation, they increased the risk for mortality and stroke, likely due to drug-induced hypotension. The slightly larger-than-typical doses of β -blockers used in this study may have contributed to the excess mortality.

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WHAT'S NEW

Avoiding β -blockers in surgery patients will prevent deaths

Bouri et al¹ found that while β -blockers protect against nonfatal MIs, they increase the risk for nonfatal strokes and death. This new meta-analysis challenges the ACCF/AHA recommendations by suggesting that abandoning the use of β -blockers for preoperative patients who aren't already taking them will prevent a substantial number of perioperative deaths. Bouri et al¹ estimate that in the United Kingdom, where 47,286 deaths occur annually within 30 days of intermediate- or high-risk procedures, the number of iatrogenic deaths would drop by approximately 10,000 if β -blockers were not used.¹

CAVEATS

Don't stop β -blockers in patients who already take them

This meta-analysis did not evaluate outcomes in patients who were already taking β -blockers. These patients should continue to take them in the perioperative pe-

riod, which is in line with current ACCF/AHA guidelines.

CHALLENGES TO IMPLEMENTATION

Reluctance to disregard published guidelines

Some clinicians may not be comfortable ignoring the current ACCF/AHA guidelines that make a Class IIA recommendation (it is reasonable to administer this treatment) for the use of preoperative β -blockade for patients at risk for cardiovascular events who were not previously taking a β -blocker. This updated meta-analysis excludes the discredited DECREASE trials and challenges us to act against these current guidelines while we await updated recommendations. **CR**

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tect against excessive water loss and electrolyte imbalance. At the same time, excessive water loss can translate to higher sodium levels and increase the risk for cerebral edema. If rapid diuresis occurs, an infusion of D5W (5% dextrose in water) to match the rate of urine output may prevent a rapid serum sodium level rise. Frequent monitoring of serum sodium levels is often necessary. In instances where ODS is already present, there are case studies of improved neurologic outcomes

with reduction of serum sodium levels.^{2,3}

While the treatment of hyponatremia at first glance seems straightforward—replace that which is lost—it can actually transform a seemingly simple problem into a complicated clinical course requiring intensive care, due to the need for frequent monitoring and intervention. **CR**
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