

Fasting, Water and Solute: The Hidden Neurological Risk

ABSURD HEALTH INSTITUTE | 15 OCTOBER 2026 | IV. The diluted brain and osmotic neurology

Abstract

This concept paper examines fasting, water and solute: the hidden neurological risk. It develops an argument drawn from *The Diluted Mind*; *Terrain Medicine: Fasting Science*, identifies the relevant biological or clinical mechanisms, and considers where that argument may fail. This is a narrative perspective, not a systematic review or an original clinical study.

The central problem

Fasting changes dietary solute intake, endocrine signaling, renal water handling and electrolyte balance. The proposed argument is that advice to consume large amounts of plain water while dramatically reducing food can be dangerous under some conditions. The hazard is not ordinary hydration, but inadequate renal free-water excretion in relation to fluid intake and available osmoles, especially when medication or illness modifies antidiuretic signaling.

Mechanisms and causal architecture

Urinary water excretion depends partly on the kidney's ability to excrete daily solute load. Low protein and salt intake can lower that load; antidiuretic hormone activity can concentrate the urine and further limit free-water elimination. Prolonged restriction may also alter total body potassium, magnesium, phosphate and nutritional status. These states cannot be corrected responsibly with a universal salt formula. Individuals with heart failure, kidney disease, hypertension, diuretics or endocrine disorders have fundamentally different risks from healthy short-term fasters.

A discriminating example

A person on a prolonged food-restriction program begins drinking multiple liters of plain water while consuming little dietary solute. That history raises a legitimate concern if neurological symptoms emerge, but it does not prove hyponatremia. Serum chemistry and acute assessment are necessary. Any approach that reinterprets confusion or seizure as a cleansing sign would be dangerously misleading.

Competing explanations and limitations

Not every person fasting develops hyponatremia, and many physiological changes during fasting are appropriate adaptation. Conversely, neurological symptoms in a fasting person may signal hypoglycemia, dehydration, infection, thiamine deficiency or other urgent conditions rather than low sodium. Interpreting worsening symptoms as beneficial “detoxification” is particularly unsafe. Severe headache, confusion, vomiting, seizures or collapse require assessment, not reassurance that a cleansing phase has begun.

What evidence would change the interpretation?

The discriminating clinical data are fluid intake, dietary solute intake, relevant medications, serum electrolytes and osmolality, urine measures and symptoms over time. Claims that a proprietary electrolyte ratio prevents osmotic injury would require specific evidence across differing populations; the source books do not establish that.

Implications for medical interpretation

This is a patient-safety concept paper. It supports individualized medical supervision of prolonged fasting rather than promoting a fasting or water-consumption protocol.

Selected scientific reading

Spasovski et al. (2014), Clinical practice guideline on diagnosis and treatment of hyponatraemia, European Journal of Endocrinology.

<https://doi.org/10.1530/EJE-13-1020>

Longo & Mattson (2014), Fasting: molecular mechanisms and clinical applications, Cell Metabolism.

<https://doi.org/10.1016/j.cmet.2013.12.008>

Adaptation of the Brain to Hyponatremia and Its Clinical Implications (2023), Journal of Clinical Medicine.

<https://doi.org/10.3390/jcm12051714>

Original source: The Diluted Mind; Terrain Medicine: Fasting Science. All source books supplied by the author.

Conceptual research commentary. Not a peer-reviewed clinical study, original trial, systematic review, validated diagnostic instrument or treatment protocol. Consult qualified care for medical decisions.