HYPER- and HYPO-HYDRATION

Question from a NUTRI-SPEC practitioner: "I have a "thought experiment" I would like to run by you. This experiment has to do with chronic overhydration/ dehydration leading to Anaerobic/Dysaerobic Imbalances. ----- Suppose I have a "normal" person and this person starts to overhydrate chronically, perhaps increasing his water intake by 25% higher than needed. Could this eventually make this patient Anaerobic, and what would be the mechanism? --- The reverse side of this would be a patient who is chronically hypohydrated. Could this lead to a Dysaerobic Imbalance?"

1. Your "thought experiment" was performed as a real-life experiment by Revici. (I just looked it up, and it is explained on page 595-596 of his book.)

2. Revici shows that increasing water intake causes the kidneys to produce excess ammonia, and thus a high ammonia, high pH, low specific gravity urine. Revici implies that the ammonia production as a means to eliminate nitrogen occurs almost solely in the kidney. However, the excess water intake and resultant ammonia production in the kidneys does have a systemic Anaerobic effect --- even if not direct.

3. The mechanism by which increased water intake indirectly causes or exacerbates an Anaerobic Imbalance, is because increased water intake stimulates excess aldosterone from the adrenal cortex. Aldosterone/mineralocorticoids are extremely Anaerobic (and can even cause Anaerobic seizures if even slightly in excess).

4. One mechanism by which aldosterone pushes a person Anaerobic is by increasing the use of protein to form ammonia (systemically, not just in the kidney). The ammonia, in turn, stimulates glycolysis, i.e., inefficient Anaerobic metabolism. Glucose is wasted, turning into lactic acid, while energy production and CO2 production is reduced. (Also --- the low CO2 yields a functional Respiratory Alkalosis/hyperventilation.)

   Now, we have a vicious cycle/positive feedback loop set up. CO2 is the means by which ammonia is eliminated --- the CO2 converts ammonia to urea in the Urea Cycle. Since the excess ammonia resulting from the aldosterone causes low CO2 production, we now have the inability to convert ammonia to urea. So now, ammonia starts to build up significantly --- both directly because of the glycolysis provoked by the ammonia (with the glycolysis resulting from the ammonia that came from the aldosterone), and now --- because of the inability to eliminate the ammonia due to the low CO2.

5. Additional note: Drinking excess water also increases both serotonin and prolactin. --- So --- the people following the health food craze of drinking 10 glasses of water daily, are running high aldosterone, high serotonin, high

prolactin, high ammonia, high lactic acid, and low carbon dioxide, plus all the consequences of a Hypocapnic Alkalosis.

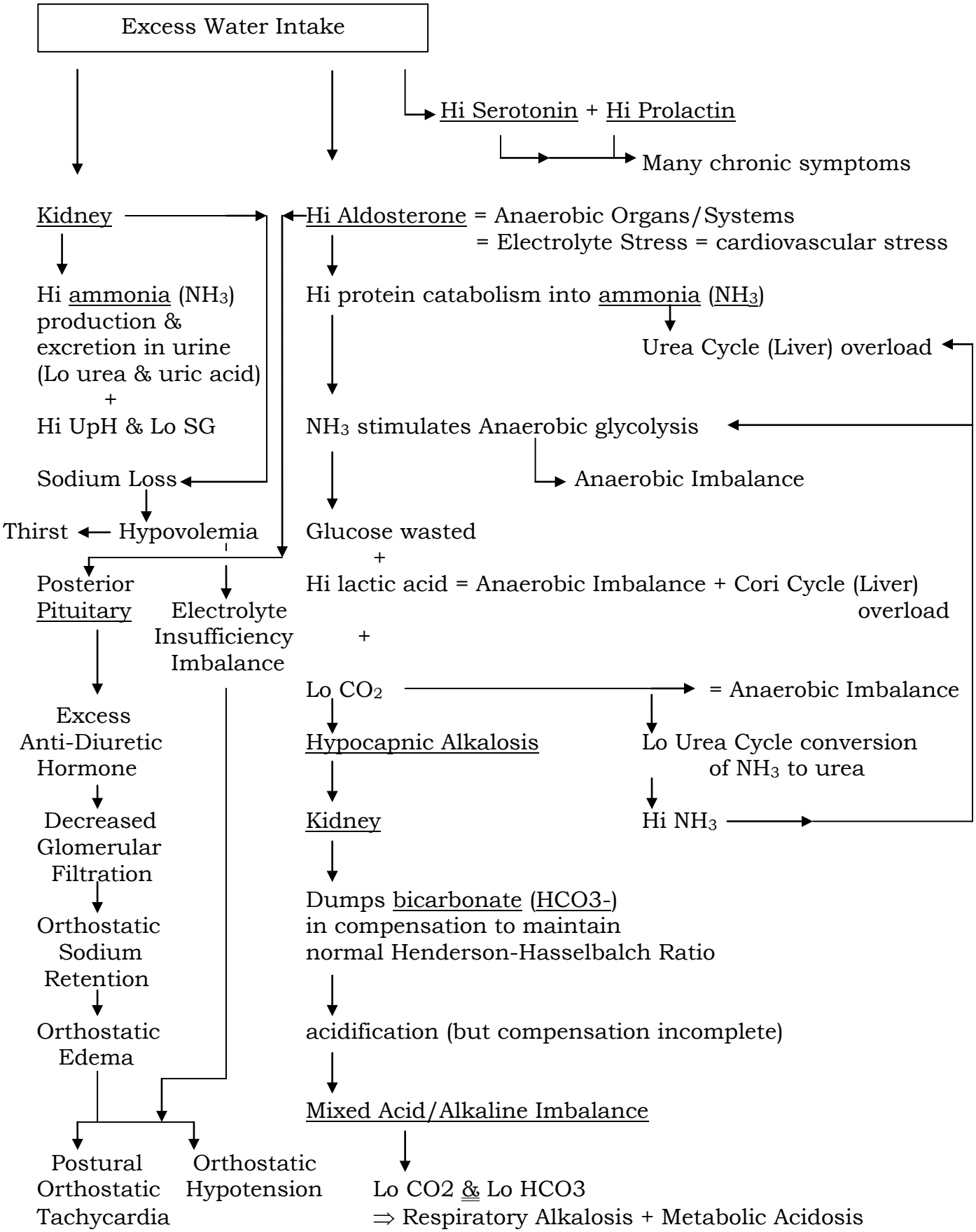
The symptoms of this systemic Alkalosis caused by excess water are on top of whatever symptoms derived from the Anaerobic Imbalance. They include muscle cramps, anxiety/depression/"stress", insomnia, constipation, and increased allergic reactivity.

6. Regarding hypohydration --- Revici is a little vague on the mechanism by which low water intake induces catabolism of purines over a period of several days. I have always been able to see clearly how a Dysaerobic Imbalances causes a high density/low water urine, but it is not clear to me how the cause-and-effect chain could be reversed such that low water intake would cause a Dysaerobic/ catabolic metabolism.

   The restricted water intake would stimulate the release of antidiuretic hormone (ADH) as a water conserving measure. I do not know of any specifically catabolic effects of ADH. I do know, however, that excess ADH decreases the renal GFR. That leads to both sodium and water retention and edema. The edema is patially aggravated by prolonged standing. In many cases this dependent edema causes albumin leakages from the serum to the interstitium. There may be orthostatic hypotension or POTS.

7. The abnormal purine catabolism alluded to by Revici (but not thoroughly explained) would certainly increase the systemic uric acid, and thus contribute to a Metabolic Acidosis. But the tissue Alkalosis of a Dysaerobic Imbalance? Revici explains that this is generally due to excess chloride fixation by trienic fatty acids, thus leaving sodium free cellularly and in the interstitium to combine with carbonates/bicarbonates and thus alkalize the tissues. How that whole mechanism might be provoked by water intake restriction or by ADH increase I do not know, beyond the obvious --- that excess ADH causes excess sodium retention.

8. Following is a flow chart illustrating the multiple metabolic pathways pushed off-balance by drinking too much water.


Excess Water Intake
Hi Serotonin + Hi Prolactin
Many chronic symptoms
Kidney
Hi Aldosterone = Anaerobic Organs/Systems
= Electrolyte Stress = cardiovascular stress
Hi ammonia (NH_3) production & excretion in urine (Lo urea & uric acid)
+
Hi UpH & Lo SG
Hi protein catabolism into ammonia (NH_3)
Urea Cycle (Liver) overload
NH_3 stimulates Anaerobic glycolysis
Anaerobic Imbalance
Sodium Loss
Thirst
Hypovolemia
Glucose wasted
+
Hi lactic acid = Anaerobic Imbalance + Cori Cycle (Liver) overload
+
Posterior Pituitary
Electrolyte Insufficiency Imbalance
Lo CO_2
= Anaerobic Imbalance
Excess Anti-Diuretic Hormone
Hypocapnic Alkalosis
Lo Urea Cycle conversion of NH_3 to urea
Decreased Glomerular Filtration
Kidney
Hi NH_3
Orthostatic Sodium Retention
Dumps bicarbonate (HCO3-) in compensation to maintain normal Henderson-Hasselbalch Ratio
Orthostatic Edema
acidification (but compensation incomplete)
Mixed Acid/Alkaline Imbalance
Postural Orthostatic Tachycardia
Orthostatic Hypotension
Lo CO2 & Lo HCO3
⇒ Respiratory Alkalosis + Metabolic Acidosis