These findings demonstrate that reductions in electrocardiographic LVH are mirroring significant reductions in LVM. The implications are that the absence of LVH regression by 6 months to 1 year after initiation of antihypertensive therapy in the setting of substantial reductions in systolic and diastolic blood pressure should be of concern, particularly given the important associations between regression of LVH and reductions in cardiovascular morbidity and mortality.43 This might warrant adjusting the antihypertensive regimen to achieve regression of LVH.

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Fluid Replacement for Severe Hyponatremia

To the Editor: Dr Alam and colleagues1 described 40 children treated with oral rehydration solution for diarrheal dehydration who experienced seizures associated with severe hyponatremia (sodium, <120 mEq/L). In their study, hyponatremia was treated with 12 mL/kg of 3% sodium chloride over a 4-hour period. This would be expected to raise the serum sodium by a minimum of 12 mEq/L in 4 hours, a more rapid increase than may be safe.2 The appropriate use of 3% sodium chloride for the treatment of symptomatic hyponatremia is controversial, as some studies have suggested that an excessive correction of hyponatremia can lead to the development of cerebral demyelination.3 If these children had a good neurological outcome, it would support the safety of using 3% sodium chloride in children with symptomatic hyponatremia.

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In Reply: Dr Moritz raises concerns about the safety of infusing 3% sodium chloride (12 mL/kg) over a 4-hour period in the management of seizure associated with severe hyponatremia. This was a phase 4 study to determine if routine use of a new formulation of reduced osmolarity oral rehydration solution (compared with a previous formulation) would be associated with symptomatic hyponatremia; unlike physiological studies, it did not have the provision for frequent monitoring of serum electrolytes.

Although the use of intravenous infusion of 3% sodium chloride in the treatment of severe symptomatic (seizure/coma) hyponatremia is recommended, a slower correction is generally recommended to limit increase in the serum sodium to a maximum of 10 mEq/L in 24 hours, which is considered safe.1 However, in routine clinical practice under a constrained-resource and phase 4 trial situation, controlled elevation of serum sodium in a child with severe hyponatremia may not be practical or possible.2 Severe cholera is associated not only with higher fecal concentration of sodium but with greater absolute loss of sodium,3 which is expected to offset a potential sharp increase in serum sodium associated with a limited amount of 3% sodium chloride infusion. Because the patients in our study were experiencing diarrhea with active purging, it is unlikely that 12 mL/kg of 3% sodium chloride infused over 4 hours would have increased the serum sodium by more than 10 mEq/L over a 24-hour period; however, because of our monitoring frequency, we could not document whether it did or did not.

Medical literature also suggests that hyponatremia should be corrected at a rate similar to that at which it develops, and an aggressive correction should be performed in patients with severe symptoms.4 Most patients who develop hyponatremic seizure as a result of acute-dehydrating diarrheal diarrhea do so within 24 to 48 hours of the onset of diarrhea, mostly during inappropriate rehydration. Hyponatremia is associated with abnormal mentation, a practical problem in the oral rehydration and maintenance of hydration that also poses feeding difficulties. Rapid correction of hyponatremia improves mentation, allowing patients to eat and drink within a shorter period of time.

At the Dhaka Hospital of the International Center for Diarrheal Disease Research Bangladesh, we have provided treatment and care to approximately 100,000 patients with diarrheal disease (an estimated 30,000 with cholera each year). Although anecdotal, we have not observed any short-term adverse events associated with the infusion of 12 mL/kg of 3% intravenous sodium chloride solution over a 4-hour period, along with maintaining hydration with oral rehydration solution (containing 75 mEq/L of sodium). However,
in the absence of long-term follow-up, we cannot comment about the possibility of neurological sequelae. That would require specific investigation, such as brain imaging for detection of cerebral demyelination.

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Does This Patient Have a Migraine?

To the Editor: In their Rational Clinical Examination article, Mr Detsky and colleagues1 evaluated the usefulness of the history and physical examination to determine whether a patient has migraine or some other condition requiring neuroimaging for diagnosis. I agree that patients with migraine do not ordinarily require neuroimaging. However, it is also important to consider that neuroimaging will not always define the illness in patients with other serious types of headache.

In patients older than 50 years with new-onset headache, particularly if there is tenderness of the scalp or head, tests such as measurement of erythrocyte sedimentation rate (ESR) and C-reactive protein (CRP) level should be performed to assess for possible giant cell or temporal arteritis. In the presence of this disease, the ESR is usually greater than 50 mm/h and may exceed 100 mm/h, but even if the ESR is 20 to 40 mm/h, giant cell arteritis is still a consideration.2 In a study of test characteristics, using a cutoff for ESR of 33 mm/h in men and 35 mm/h in women and a cutoff for CRP level of 0.5 mg/dL resulted in sensitivity of 92% and specificity of 92% for detecting temporal arteritis.3 However, criteria most strongly suggestive of temporal arteritis included a CRP level greater than 2.45 mg/dL (odds ratio of obtaining a positive temporal artery biopsy result, 3.2) and an ESR 47 mm/h or more (odds ratio for a positive biopsy result, 2.0 for an ESR of 47 to 107 mm/h and 2.7 for an ESR greater than 107 mm/h). A temporal artery biopsy is usually necessary to diagnose the condition.

Treatment of patients with temporal arteritis is critical to avoid vision loss, and therapy should be initiated based on clinical suspicion, not biopsy results.2 Neuroimaging is unlikely to be helpful in determining the cause of headache in a patient with temporal arteritis.

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1. Detsky ME, McDonald DR, Baerlocher MO, et al. Does this patient with headache have a migraine or need neuroimaging? JAMA. 2006;296:1274-1283.

In Reply: We agree with the suggestion made by Dr Brenner that not all sinister causes of headaches will be discovered by neuroimaging. Examples of such etiologies are temporal arteritis and bacterial meningitis. This emphasizes the importance of thorough history taking and physical examination in a patient with headache. Pertinent findings during this initial portion of the clinical examination may serve to guide further diagnostic tests or therapy.

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CORRECTION

Data Error and Incorrect Wording: In the Original Contribution entitled “Survival Associated With Treatment vs Observation of Localized Prostate Cancer in Elderly Men” published in the December 13, 2006, issue of JAMA (2006;296:2683-2693), two tables had data errors and another table had incorrect wording. In Table 3, the fourth quintile of the 10-year overall survival for the observation group should have read 0.61 (0.58-0.65) and in the treatment group, the 10-year overall survival for the fourth quintile should have read 0.68 (0.67-0.70) for the propensity scores and their respective 95% confidence intervals. In the final row of Table 7, the hazard ratio and 95% confidence interval should have read 0.97 (0.94-1.00). In Table 6, the column head that reads "No. of Patients Who Died" should read "No. of Patients."