Serum Electrolytes in Children With Neurocardiogenic Syncope Treated With Fludrocortisone and Salt

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The combination of fludrocortisone and oral sodium chloride has been effective in the treatment of neurocardiogenic syncope in children.[1,2] Given the high sodium load, changes in serum electrolytes may be expected. The extent of these changes, however, is unknown. This study examines whether there were any significant changes in the serum electrolytes during therapy that would justify routine electrolyte testing.

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The study group consisted of 17 patients (11 girls and 6 boys) with a positive tilt test. Our tilt test protocol has been described in detail previously.[3,4] All patients had a structurally normal heart. Following a positive tilt test, all patients were treated with sodium chloride 1 g/day and fludrocortisone (0.3 mg/day for 7 days, then 0.1 mg/day). Serum electrolytes were measured at weekly intervals for the initial 4 weeks, and monthly thereafter for the duration of therapy. We retrospectively reviewed the results of all blood analyses that were available. The mean ± SDs were determined for each variable. Regression analysis and Student's *t* test were used when appropriate. A p value <0.05 was considered significant.

The patients' demographics are listed in Table I. Syncope was diagnosed to be cardioinhibitory, vasodepressor, or combined in 6, 8, and 3 patients, respectively. All patients were free of syncope while receiving therapy. A total of 104 tests were performed on the 17 patients. The results of serum electrolytes analyses are depicted in Figure 1. Serum sodium concentration was within the normal range in 100 of the 104 tests (96%); in 4 tests it was between 146 and 149 mEq/L. There was no correlation between the initial and follow-up serum sodium concentration. Serum potassium concentration was in the normal range in 98% of the tests performed; it was <3.4 mEq/L in 2 tests (2%). Serum chloride concentration exceeded the normal range in 8 tests (8%). Five of the abnormal assays were within the first month after the initiation of therapy. Serum bicarbonate concentration increased to beyond the normal range (18 to 27 mEq/L) in 41 assays (40%). The bicarbonate concentration was >30 mEq/L in 15 tests. There was a significant positive correlation between bicarbonate and sodium concentrations ($r = 0.38$; $p < 0.0001$). The anion gap was normal in all but 1 test. No electrolyte changes were associated with any clinical symptom. Renal function was normal in all patients (blood urea nitrogen 8 ± 3 mg/dL and creatinine 0.6 ± 0.1 mg/dL). In addition, baseline blood pressures were normal in all patients (systolic 112 ± 11 mm Hg and diastolic 63 ± 10 mm Hg), and there was no significant change after therapy (systolic 114 ± 13 mm Hg, diastolic 62 ± 9 mm Hg). There was a significant weight gain after therapy, from 47.2 ± 17.1 to 48.1 ± 17.8 kg; $p < 0.0003$. The standard therapy was continued without any modifications except for adding β blockers in 3 patients.

TABLE I Patients' Demographics and Follow-Up Information

Parameter	Mean ± SD	Range
Age at initial syncope (yr)	11.9 ± 3.7	7–17
Age at tilt test (yr)	13.1 ± 2.6	9–17.8
Follow-up (yr)	1.6 ± 0.9	0.1–3
Duration of therapy (mo)	5.8 ± 5.4	0.2–21
No. of blood tests per patient	6 ± 4	1–16

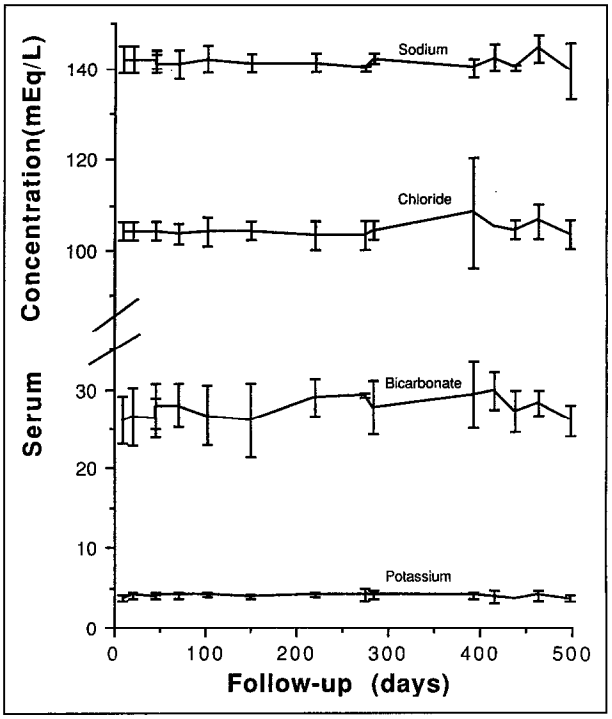

FIGURE 1. The mean values ± 1 SD of serum electrolytes (mEq/L) versus the interval (days) after therapy was started when the tests were obtained.

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The mechanism of neurocardiogenic syncope in children is still undefined. A proposed mechanism suggests that a postural decrease in blood return to

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the heart causes stimulation of left ventricular baroreceptors leading to vasodilation and hypotension.[5] Sodium chloride and mineralocorticoids are used widely in the treatment of this type of syncope. They expand the extracellular fluid volume and decrease the postural changes in cardiac filling and, hence, prevent syncope. This increase in extracellular volume in our patients was manifested as significant weight gain after therapy. The combination of a high salt load and mineralocorticoid therapy may have caused an increased serum sodium concentration. However, we did not observe an abnormal sodium concentration in a significant number of our patients, probably because of additional water retention. Chloride concentration tracked that of sodium. Moreover, because these patients had a normal acid-base balance there was no increase in the anion gap. The changes in serum potassium were limited. The lack of significant hypokalemia was probably related to the relatively low dose of fludrocortisone used. In addition, we encouraged our patients to increase their intake of potassium-containing foods. The most significant observation was an increase in serum bicarbonate concentration. Metabolic alkalosis was found in 40% of test results. The most plausible explanation for this phenomenon is a decrease in proximal renal tubular sodium reabsorption due to the expansion in extracellular fluid volume. The increased sodium delivery to the distal nephron results in an enhanced reabsorption of sodium in exchange for potassium and hydrogen ions. The bicarbonate generated from the secretion of hydrogen ions causes "physiologic" metabolic alkalosis.[6] This also explains the positive correlation between the serum concentrations of sodium and bicarbonate.

In conclusion, the treatment of syncope with sodium chloride supplement and fludrocortisone is associated with minimal and nonsignificant changes in serum electrolytes. The greatest change observed is an increase in serum bicarbonate. This change is not sufficient to require modification of the treatment. The added cost and discomfort associated with serial serum electrolytes testing do not appear to alter therapy.

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Contrast-Enhanced Doppler Ultrasound for Noninvasive Assessment of Pulmonary Artery Pressure During Exercise in Patients With Chronic Congestive Heart Failure

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Pulmonary artery (PA) pressure response to exercise[1] has been used as an objective indicator of functional impairment in patients with chronic congestive heart failure (CHF). Doppler echocardiography has been shown to accurately estimate systolic PA pressures at rest[2] and during exercise in patients with chronic obstructive lung disease[3] and mitral valve disease.[4] Recently, exercise Doppler estimation of PA pressure was also applied to patients with congenital heart disease[5] and to heart transplant recipients.[6] Feasibility and accuracy of exercise Doppler in estimating PA pressures in patients with chronic CHF have not been well characterized. Therefore, we investigated whether contrast-enhanced Doppler echocardiography can be used to estimate PA pressure responses to exercise in patients with chronic CHF.

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The study protocol was approved by the ethics committee of the University of Heidelberg. All patients gave written informed consent before entering the study. The study group consisted of 19 patients (4 women and 15 men, age 53 ± 11 [mean ± SD] years) with chronic CHF (17 with dilated cardiomyopathy and 2 with coronary artery disease) confirmed by routine left- and right-sided cardiac catheterizations and coronary angiography. Patients were asked to participate in the study when echocardiographic ejection fraction was ≤40%. Patients with coronary artery disease were excluded from the study when angina or signs of

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