**Efficacy of high sodium intake in a boy with instantaneous orthostatic hypotension**

**Introduction**

Orthostatic hypotension (OH) is a decline in blood pressure that occurs on the assumption of upright posture that results in symptoms of cerebral hypoperfusion, most commonly lightheadedness, fatigue, and syncope. In childhood, the disorder may result from decreased sympathetic nervous activity [1] or reversible nonautonomic causes, such as dehydration. Although the goal of therapy is to relieve symptoms, OH is difficult to treat pharmacologically because of varying responses and adverse effects. Accordingly, nonpharmacologic approaches to increase plasma volume are preferable, if effective. In the literature, high sodium intake was reported to improve OH [2], but no uniformly effective treatment regimen exists.

We here report the case of a boy with instantaneous orthostatic hypotension (INOH) [1], a common form of orthostatic hypotension in childhood that was treated with high sodium intake in combination with L-threo-3,4-dihydroxyphenylserine. We used a continuous non-invasive cardiovascular monitoring system to evaluate the effect of high sodium intake on circulatory responses.

**Case report**

A 14-year-old boy had orthostatic dizziness, chronic fatigue, and sleep disturbance. When he was 12, he began to complain of orthostatic dizziness and often missed school. Although he was given several alpha-adrenoceptor agonists, including L-threo-3,4-dihydroxyphenylserine (L-DOPS), 100 mg twice a day for two weeks, they were ineffective. Finally, he became unable to attend school at all. He was referred and hospitalized at our hospital for further investigation in May 1999.
On admission, he was 162 cm tall and weighed 41.7 kg (obesity rate: – 18.4%). In the supine position, blood pressure was 110/52 mmHg and heart rate was 78/min with a regular sinus rhythm.

White blood cell count was 3870/mm³, and hemoglobin was 14.2 g/dl. Electrolytes were within normal range (Na 138 mEq/l, K 4.2 mEq/l, Cl 98 mEq/l, Ca 9.7 mg/dl). Renal and hepatic function tests were normal. Thyroid function was normal (FT4 20 ng/dl, FT3 3.3 pg/ml, TSH 0.63 mU/ml). Plasma osmotic pressure was 284 mOsm/kg, plasma renin activity was 0.3 ng/ml/h in the supine position (normal: 0.3–2.9 ng/ml/h), whereas aldosterone was slightly decreased to 23 pg/ml (normal: 29.9–159 pg/ml).

On the active standing test, he showed a marked initial drop of blood pressure to the level of 40/25 mmHg (systolic/diastolic pressure) and delayed recovery. This initial fall of blood pressure was associated with complaints of dizziness, blurred vision, headache and strong fatigue. Orthostatic tachycardia (120/min) was also found. Thereafter, at min 1 of standing, he felt poorly and complained of nausea and could not tolerate upright posture in association with hypotension (60/30 mmHg). These symptoms were completely reproduced by passive head-up tilt test.

On another day, we studied plasma catecholamine responses to standing. This time the patient tolerated the study for five minutes. Increments of plasma noradrenaline level at min 1 and 5 of standing were rather low compared to controls, whereas those of adrenaline were not decreased. From these findings, we diagnosed his illness as a severe form of instantaneous orthostatic hypotension (INOH) according to the classification reported by Tanaka et al. [1].

Administration of L-threo-3,4-dihydroxyphenylserine, noradrenaline precursor (L-DOPS), 100 mg twice a day was tried again for three weeks, but did not improve his clinical symptoms and circulatory responses to the active standing test. He showed a large initial pressure drop to 42/18 mmHg (systolic/diastolic pressure) and could tolerate standing only for 80 s as shown in Fig. 1 (upper panel). However, we found that abdominal compression using an inflatable band [3] showed a quite marked effect. This was probably due to increased circulating plasma volume during upright posture by abdominal compression.

On the basis of the above evidence, we started treatment with high sodium intake (3 g NaCl two times a day per os in addition to regular diet (including 5–6 g NaCl/day) to expand plasma volume according to Pechere's report [4]. L-DOPS was also continued. Subsequently his OH and orthostatic dizziness were markedly reduced. Forty-eight hours after the start of high sodium intake, circulatory responses to orthostatic tests were dramatically improved: initial blood pressure drop upon standing and tachycardia diappeared, resulting in improvement of orthostatic tolerance (for 3 minutes) as shown in Fig. 1 (lower panel). We measured the patient’s body water using the multifrequency bioelectrical impedance method [5] (MLT – 100, Sekisui Kagaku Kogyo Co. Ltd, Tokyo), at 3:00 p.m. before and two days after the high sodium intake. Both extracellular- and intracellular-fluid increased with high sodium intake by 4.7% and 4.3%, respectively. Body weight was increased from 41.7 kg to 43.3 kg.

We measured his daytime average blood pressure, heart rate, and cardiac output before and after the high sodium intake by Portapres™ (TNO-IPD Biomedical Instrumentation). The high sodium intake resulted in a 19% increase in cardiac output, and a rise in systolic and diastolic blood pressure (before: after = 98/59 mmHg: 122/73 mmHg) (Table 1).

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After discharge, the patient continued to take a lesser amount of sodium (regular diet + NaCl 2 g/day P.O.) for 6 months with L-DOPS (200 mg/day) and propranolol (10 mg/day). His OH and indeterminate symptoms were relieved. No adverse effects of high sodium intake such as electrolytic imbalance and thirst were noted.

### Discussion

In the present case, high sodium intake for the purpose of increasing plasma volume remarkably alleviated our patient’s orthostatic intolerance.

Tanaka et al. reported a new entity of orthostatic intolerance, called INOH which is characterized by significant hypotension immediately after standing using a beat-to-beat blood pressure monitoring such as Finapres. Briefly, INOH is divided into two subsets: a mild form showing a recovery time for mean arterial pressure of more than 25 s, or a recovery time for more than 20 s with a 60% or greater decrease in mean arterial pressure at the initial drop, and a severe form showing prolonged reduction in systolic arterial pressure of more than 15% during the later stage of standing in combination with a similar initial circulatory change to a mild form. Patients with INOH usually show a marked reduction in blood pressure at the initial drop and nearly a half of the patients have delayed recovery time of more than 60 s as-

### Table 1: Daytime average blood pressure, heart rate and hemodynamic parameters before and one week after the high sodium intake.

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Before</th>
<th>After</th>
</tr>
</thead>
<tbody>
<tr>
<td>Blood pressure systolic/diastolic (mmHg)</td>
<td>98±17/59±12</td>
<td>122±14/73±8</td>
</tr>
<tr>
<td>Heart rate (bpm)</td>
<td>70±13</td>
<td>70±9</td>
</tr>
<tr>
<td>Stroke volume (ml)</td>
<td>45±7</td>
<td>53±5</td>
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<tr>
<td>Cardiac output (l/m)</td>
<td>3.1±0.6</td>
<td>3.7±0.5</td>
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<tr>
<td>Peripheral resistance index</td>
<td>23.4</td>
<td>24.7</td>
</tr>
</tbody>
</table>
