Sodium and Hypertension*

Susumu Yorifuji

Any theory on the genesis of essential hypertension must explain the following facts:

1) It is a popular disease,
2) there is gradual transition from normal to hypertension, of which some cases are highly malignant,
3) heredity plays an important role,
4) some environmental factors, especially sodium intake and stress, may promote the occurrence of hypertension,
5) one cannot disregard aging as a factor of hypertension, and,
6) at the beginning of essential hypertension, no special changes are detectable but high blood pressure.

No disease of the special organ is thought to be able to have such features as described above but some metabolic change or insufficiency

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**Fig. 1.** Adrenaline and aldosterone increase the intracellular Na content, but chloropyrazinamide decreases it.

**Key Words:** Hypertension
Electrolyte Metabolism

*Department of Internal Medicine, Division 1 Kobe University School of Medicine*
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Table I

<table>
<thead>
<tr>
<th>Normotensive Rat</th>
<th>S.H.R.</th>
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<tbody>
<tr>
<td></td>
<td>Na</td>
</tr>
<tr>
<td>R-Atrium</td>
<td>79.98 ± 9.93</td>
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<tr>
<td>L-Ventricle</td>
<td>50.69 ± 6.18</td>
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<tr>
<td>Aorta</td>
<td>98.54 ± 6.38</td>
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</tbody>
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(mEq/Kg)

which aging may accompany.
My colleagues and I had been investigated on the relationship between hypertension and sodium metabolism, and the following results were obtained.
1) Drinking of 2 per cent NaCl solution can bring about mild hypertension in rats<sup>4,5</sup>.  
2) Sodium hypertension can be markedly augmented by
   I) unilaretal ligation of renal arteries  
   II) long term administratoin of adrenalin  
   III) carotid sinus denervation and  
   IV) ligation of both carotid arteries.  
3) In toad's heart infused with Ringer solution, adrenaline and aldosterone increase the sodium content of the heart muscle, but chlorothiazide decreases it. (Fig. 1)<sup>3,4,5</sup>
4) In spontaneously hypertensive rats (SHR-Okamoto) sodium content of heart is increased<sup>9</sup>. (Table I)

As heart is genetically thought to be an example of blood vessel, and as spontaneously hypertensive rat (SHR-Okamoto), is a sort of genetic hypertension, those results obtained are considered to show that hypertension is accompanied by the intracellular increase of sodium content in blood vessel and lowering of blood pressure by the decrease of it.

It is to be determined how hypertension occurs with the intracellular increase of Na content, but this fact is thought to be enough to fulfil the requirements which I have at the beginning said. Because it is highly probable that 1) disturbance of sodium metabolism is liable to occur with the hereditary weakness which become manifest with aging, 2) increased intake of sodium and stress may increase the intracellular sodium content, and 3) in extreme case, marked metabolic insufficiency may cause malignant hypertension.

REFERENCE

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5. INATOME, T.: to be published.

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