Total body potassium falls with age

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Summary

1. Plasma volume, 24 h urine sodium and total body potassium were similar among 89 previously untreated patients with mild hypertension and 89 control subjects matched for age and sex.

2. Total body potassium correlated with renin activity in both hypertensive patients and controls.

3. Renin activity and total body potassium fell significantly with age in both groups.

4. The relationship between renin and total body potassium was no longer present when the effects of age were allowed for in a partial correlation.

Key words: age, essential hypertension, renin, total body potassium.

Introduction

Plasma volumes, exchangeable sodium, total body potassium, renin and aldosterone are likely to be affected by previous drug treatment in hypertensive patients (Dunn & Tannen, 1974; Lowder & Liddle, 1974; Aurell, Patterson & Berglund, 1975) and are also likely to be affected by differences in age and sex. This paper provides data on exchangeable sodium, plasma volume, urinary sodium and potassium, total body potassium and supine and stimulated plasma renin activity in 89 previously untreated patients with essential hypertension and in 89 control subjects matched with the hypertensive patients for age and sex (71 males and 18 females in each group).

Patients and methods

Patients and control subjects were drawn from a community hypertension screening programme and their informed consent was obtained. Approval was given by the hospital ethics committee. Details of the screening procedure and the selection of patients and control subjects have been published previously (Thomas, Ledingham, Beilin, Stott & Yeates, 1978).

All subjects were investigated on an unrestricted diet. Plasma renin activity was measured by the method of Sealey, Gerten-Banes & Laragh (1972). Blood samples were taken at 11.00 hours after 2 h supine, at 13.00 hours after 2 h erect and at 14.00 hours, 1 h after intravenous frusemide (3 μmol/kg: 1 mg/kg).

Urinary sodium and potassium were measured by flame photometry.

Twenty-four hour exchangeable sodium was determined by isotope dilution. After intravenous injection of a known dose of 1-3 μCi of 24Na, the subject collected urine voided between 0-23 and 23-25 h. Exchangeable sodium was calculated from the ratio of stable to radioactive sodium in the 23-25 h sample, and the whole-body 24Na retained at 24 h; the latter quantity was estimated from the known dose by subtraction of the 0–23 h excretion and 50% of the 23–25 h excretion.

Total body potassium was estimated from measurements of whole-body radioactivity with an array of six large NaI(Tl) scintillators arranged at intervals above and below the mid-line of the supine subject (Newton & Eagle, 1972). The subject and detectors were wholly enclosed in a lead shield 102 mm thick (International Atomic Energy Agency, 1970). Estimates of total body potassium were based on the recorded radioactivity count rate between 1.30 and 1.61 MeV, bracketing the photopeak from the 1.46 MeV gamma rays of naturally occurring

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40 K. The subsequent injection of 24Na, administered to assess exchangeable sodium, enabled the calibration factor appropriate to each subject to be assessed accurately, since 24Na emits photons very similar in energy to those of 40 K. A second gamma-ray spectrum from the subject was recorded 2 h after the injection. From the recorded count rate in the 1.38 MeV photopeak, and the known dose of 24Na administered (less excretion measured in any urine voided between 0 and 2 h), the whole-body counting efficiency for 1.38 MeV gamma rays was deduced. The calibration factor for 40K was derived from this efficiency, with a small adjustment to allow for the slightly reduced attenuation of 1.46 MeV gamma rays. This approach was validated by administering 42K (12.5 h half-life, 1.52 MeV gamma rays) and 24Na on separate occasions to the same six subjects, and comparing the calibration factors for 40K derived by the two methods. The overall standard error in estimate of total body potassium was less than 3%.

Plasma volume was measured by a standard isotope dilution technique using 131I-labelled human serum albumin (Tarazi, Frohlich & Dustan, 1968).

Pearson’s correlation coefficients were used in the analysis of associations between variables. Differences between groups were assessed by Student’s unpaired t-test.

Results

Plasma sodium, plasma potassium, 24 h urinary sodium, total body potassium and plasma volumes were not significantly different in the two groups. Frequency distributions of renin activity and the relationships between age, blood pressure and renin activity in the two groups and in the 178 subjects as a whole have been described previously (Thomas et al., 1978).

Total exchangeable sodium was lower in the hypertensive group (P < 0.005) but hypertensive patients were heavier (77.26 ± 1.1 kg) than the controls (72.11 ± 1.08 kg). Twenty-four hour urinary potassium excretion was significantly lower in hypertensive than control subjects (Table 1). Total body potassium was similar in hypertensive and control subjects, but there were significant correlations between total body potassium and renin measurements in both groups. Total body potassium was also inversely correlated with age (P < 0.001) in both hypertensive (r = −0.40) and control subjects (r = −0.41). When the effects of age were allowed for by partial correlation, these apparent relationships between renin and total body potassium were no longer present.

Discussion

Most of the published observations on renin activity in hypertensive patients have been obtained from subjects previously treated and/or not adequately matched by appropriate controls (Dunn & Tannen, 1974). This study was designed to avoid the potentially distorting effects of previous therapy and to provide a series of control subjects precisely matched with the patients for age and sex. Such an approach has shown that renin activity in both hypertensive and control subjects is log-normally distributed, with no evidence of discrete renin

### Table 1. Measurements of sodium and potassium in the whole body and urine of hypertensive patients and control subjects and correlations between total body potassium and plasma renin activity

<table>
<thead>
<tr>
<th>Subjects</th>
<th>Arterial pressure (mmHg)</th>
<th>Urinary Na (mmol/24 h)</th>
<th>Urinary K (mmol/24 h)</th>
<th>Exchangeable sodium (mmol/kg)</th>
<th>Total body potassium (mmol/kg)</th>
<th>Plasma volume (litres/1.73 m²)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
<td>123.0 ± 1.3/78.0 ± 1.4</td>
<td>157.9 ± 5.0</td>
<td>66.1 ± 1.9</td>
<td>42.1 ± 0.6</td>
<td>46.0 ± 0.6</td>
<td>2.89 ± 0.03</td>
</tr>
<tr>
<td>Hypertensive</td>
<td>161.5 ± 2.1/104.2 ± 0.9</td>
<td>150.8 ± 5.6</td>
<td>60.1 ± 2.1**</td>
<td>39.7 ± 0.6**</td>
<td>44.4 ± 0.6</td>
<td>2.96 ± 0.04</td>
</tr>
</tbody>
</table>

**Table 1.** Measurements of sodium and potassium in the whole body and urine of hypertensive patients and control subjects and correlations between total body potassium and plasma renin activity

<table>
<thead>
<tr>
<th>Subjects</th>
<th>Supine plasma renin activity</th>
<th>Erect plasma renin activity</th>
<th>Post-frusemide plasma renin activity</th>
<th>Corrected for age</th>
<th>Supine plasma renin activity</th>
<th>Erect plasma renin activity</th>
<th>Post-frusemide plasma renin activity</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
<td>r 0.28</td>
<td>0.13</td>
<td>0.23</td>
<td>0.20</td>
<td>&lt;0.007</td>
<td>&lt;0.007</td>
<td>&lt;0.033</td>
</tr>
<tr>
<td>Hypertensive</td>
<td>r 0.15</td>
<td>0.23</td>
<td>0.26</td>
<td>0.08</td>
<td>0.08</td>
<td>0.08</td>
<td>0.08</td>
</tr>
</tbody>
</table>

**Table 1.** Measurements of sodium and potassium in the whole body and urine of hypertensive patients and control subjects and correlations between total body potassium and plasma renin activity

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<tr>
<th>Subjects</th>
<th>Uncorrected for age</th>
<th>Corrected for age</th>
</tr>
</thead>
<tbody>
<tr>
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<tr>
<td>Control</td>
<td>r 0.28</td>
<td>0.20</td>
</tr>
<tr>
<td></td>
<td>&lt;0.007</td>
<td>&lt;0.002</td>
</tr>
<tr>
<td>Hypertensive</td>
<td>r 0.15</td>
<td>0.08</td>
</tr>
<tr>
<td></td>
<td>&lt;0.168</td>
<td>0.08</td>
</tr>
</tbody>
</table>
sub-groups, and that renin tends to fall with age in both hypertensive and normotensive people: the greater fall in hypertensive patients may be related to the influence of arterial pressure itself on renin activity (Thomas et al., 1978). Despite these findings and those of Padfield, Beevers, Brown, Davies, Lever, Robertson & Tree (1975) and Thurston, Bing, Pohl & Swales (1978), there remains some interest in the possibility that 'low renin hypertension' is a separate entity perhaps caused by oversecretion of an unknown mineralocorticoid (Melby & Dale, 1979).

Without a proper control group, and without taking into account the possible effects of ageing, the data showing a correlation between total body potassium and renin in the hypertensive patients might have been taken to support this possibility. But the same relationship between total body potassium and renin was evident among normotensive people and it disappeared in both groups when the effects of age on renin and on total body potassium were considered. There have been previous reports of a fall in total body potassium with age from the teens to the nineties in apparently healthy people (Delawaide & Crenier, 1973; Pierson, Lin & Phillips, 1974).

There are difficulties in assessing total body potassium in relation to anthropomorphic measurements (Jones, 1970), but the relationship with age was present in both hypertensive and normotensive subjects in our series whether total body potassium was expressed in terms of weight, height or body surface area. The most likely explanation for this finding is a fall in the mass of muscle in relation to other tissues with ageing (Pierson et al., 1974).

Acknowledgments

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References


