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EFFECT OF LONG-TERM HYPOKINESIA ON THE ELECTROLYTIC COMPOSITION OF THE BLOOD IN PATIENTS WITH OSTEOARTICULAR TUBERCULOSIS

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76 patients with osteoarticular tuberculosis were divided into two groups, one of which was required to maintain strict bed rest and the other of which was allowed unrestricted motor activity. A study of blood electrolyte composition in the two groups revealed that bed rest for these patients results in decreased plasma potassium, calcium, and magnesium content, but that these indices improved after the patients were allowed to move freely. The study suggests that patients with osteoarticular tuberculosis who are on bed rest be carefully observed for alterations in blood electrolytes and that proper electrolyte balance be maintained.

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ERRCT OF LONG-TERM HYPOKINESIA ON THE ELECTROLYTIC COMPOSITION OF THE BLOOD IN PATIENTS WITH OSTEOARTICULAR TUBERCULOSIS

V. P. Zakutaeva
Kirgiz Scientific Research Institute of Tuberculosis

The hypokinetic syndrome is a complex body state characterized by functional deconditioning of various organs and systems due to prolonged restriction of motor functioning. Besides the cardiovascular system, which sharply reduces its activity during hypokinesia, (V. V. Parin, M. Bayevskiy, O. G. Gazenko, 1965; V. S. Georgiyevskiy, V. M. Mikhaylov 1968; L. A. Ioffe, 1971; V. P. Zakutaeva, N. I. Matiks, 1975; etc.), a muscle activity deficit affects the functions of respiration and metabolism, (M. I. Mikhashev et al., 1969; L. I. Kakurin et al., 1970, etc.) and decreases the body's immunobiological reactivity, (P. A. Sorokin, et al., 1969; P. G. Petrova, 1971; etc.). Research done by many authors has discovered disordered protein, vitamin, hormone, and enzyme balances in the human body during restricted physical activity, (P. A. Orlova, 1959; M. S. Seregin et al., 1959; A. S. Vittolo, A. S. Ushakov, 1970; Ye. Simonov, I. V. Fodorov, 1970; etc.). Dramatic shifts in electrolyte metabolism have been found in Cosmonauts exercising limited muscle activity within the confines of space craft and in healthy persons restricted to bedrest in simulation experiments. A distinct tendency towards reduced blood plasma potassium content was shown by the end of sixty days of hypodynamia, (I. I. Ivanov et al., 1969). Bone decalcification accompanied by calciuria has been noted in Cosmonauts and in experimental animals subjected to immobilization, (B. B. Yegorov et al., 1969; Zawton, 1962; D. etlein, 1964; etc). However, the status of electrolyte metabolism during prolonged hypokinesia attending the bedrest necessitated by various pathological body conditions, (myocardial infarction, paralysis, and locomotor apparatus diseases) has not been clarified in the literature.

The purpose of our work was to study blood electrolyte content

*Numbers in the margin indicate pagination in the foreign text.
in patients with osteoarticular tuberculosis during prolonged bedrest. Under study were 76 patients with specific infection of the locomotor apparatus, 42 of which observed strict bedrest for from three to twelve months; 34 patients with unrestricted physical activity and 26 healthy persons served as the control group. Potassium content in blood plasma, \((K_D)\) and erythrocytes, \((K_I)\) and plasma sodium \((Na_D)\) was determined using flame photometry. Complexometric titration was used to ascertain blood calcium, \((Ca_D)\) and magnesium, \((Mg_D)\). The electrolyte ratios, \(K_I/K_D\) and \(Na_D/K_D\) were calculated simultaneously, where \(K_I\) is intracellular potassium, \(K_D\) is extracellular potassium and \(Na_D\) is extracellular sodium.

As can be seen in the table, considerable shifts in electrolyte balance were found in patients from both groups with osteoarticular tuberculosis upon admission; the shifts were manifested as elevated extracellular potassium content and reduced erythrocyte potassium concentrations, causing a decreased coefficient, \((K_I/K_D)\). Plasma potassium content was low. Disordered potassium metabolism obviously is a characteristic of the tubercular process, as attested by information from some authors concerning elevated plasma potassium during tuberculosis of the lungs, (Z. V. Matyushina, 1974; L. P. Andreyev, 1976; etc.). The deficit in blood potassium for patients with tubercular infection of the locomotor apparatus is linked to mineral imbalance in both the entire body and in the tubercular focal zones in osseous tissue, as proved in the experimental studies of M. V. Syromyatnikova, (1966).

As the tubercular process subsided and complications of the basic disease were eliminated in the patients allowed to move about freely, potassium metabolism had begun to normalize after three months of observation, and only the low plasma potassium content evidenced the unresolved process in osseous tissue.

Characteristic shifts in blood mineral composition arose during the observance of bedrest. Plasma potassium content normalized during the first month of hypokinesia, then gradually decreased,
falling well below normal by the sixth month. Intracellular potassium concentration, the level of which was beginning to differ even from the low initial figures (P < 0.01) after one month of hypokinesia, experienced a parallel decrease. This parallel reduction in extra- and intracellular potassium was not materially reflected in potassium gradient dynamics, but remained low over the entire period of observation. A low plasma sodium level established itself beginning with the first month of immobilization and continued through the whole bedrest period. The $\frac{Na_E}{K_E}$ ratio did not change, which was evidence of an ion equilibrium between these two most important electrolytes. Potassium content did not substantially change during the entire period of hypokinesia and approximated the low initial level.

Blood magnesium dynamics in the patients with osteoarticular tuberculosis were of interest. Plasma magnesium concentrations were normal in the initial period for patients of both groups, and during the entire bedrest period. Magnesium levels significantly increased after three months of treatment for the patients with unrestricted motor activity and one month following the end of bedrest for the other group. The impression was created that an elevated concentration of extracellular magnesium is a favorable prognostic sign. Hence, this experiment led us to conclude that prolonged bedrest substantially worsens the disordered electrolytic balance in patients with osteoarticular tuberculosis. A sharp reduction in intra- and extracellular potassium and plasma sodium is characteristic of the hypokinetic syndrome in patients on bedrest for specific infection of the locomotor apparatus. Blood electrolytes drop considerably below permissible limits, corresponding in our observations to: $K_E$ from 4.55 to 4.13, $K_I$ from 95.85 to 89.39, and $Na_E$ from 137.80 to 128.58 mEq/l (at $P=0.01$). A low potassium gradient was noted over the entire bedrest period, demonstrating a plasma and intracellular electrolyte imbalance in these patients. More pronounced blood electrolyte shifts arose after three and more months of bedrest. A blood potassium deficit accompanied the hypokinesia.
Table 1. Blood Electrolyte Composition in Patients with Osteoarticular Tuberculosis Relative to the Nature of their Motor Regime ($M \pm m$).

<table>
<thead>
<tr>
<th>Stages of Study</th>
<th>$K_E$ mEq/l</th>
<th>$K_I$ mEq/l</th>
<th>$Na_E$ mEq/l</th>
<th>$K_I/K_E$</th>
<th>$Na_E/K_E$</th>
<th>$Ca_E$ mEq/l</th>
<th>$Mg_E$ mEq/l</th>
</tr>
</thead>
<tbody>
<tr>
<td>Initial period</td>
<td>1.52 ± 0.14</td>
<td>0.39 ± 0.14</td>
<td>1.84 ± 0.14</td>
<td>0.72 ± 0.08</td>
<td>1.15 ± 0.06</td>
<td>2.31 ± 0.31</td>
<td></td>
</tr>
<tr>
<td>1 month</td>
<td>1.0 ± 0.06</td>
<td>0.33 ± 0.06</td>
<td>1.75 ± 0.06</td>
<td>0.72 ± 0.06</td>
<td>1.15 ± 0.06</td>
<td>2.31 ± 0.31</td>
<td></td>
</tr>
<tr>
<td>3 months</td>
<td>1.16 ± 0.02</td>
<td>0.38 ± 0.02</td>
<td>1.74 ± 0.02</td>
<td>0.72 ± 0.02</td>
<td>1.15 ± 0.02</td>
<td>2.31 ± 0.29</td>
<td></td>
</tr>
<tr>
<td>Over 3 months</td>
<td>1.11 ± 0.08</td>
<td>0.38 ± 0.08</td>
<td>1.84 ± 0.08</td>
<td>0.72 ± 0.08</td>
<td>1.15 ± 0.08</td>
<td>2.31 ± 0.29</td>
<td></td>
</tr>
<tr>
<td>Before &quot;arising&quot;</td>
<td>1.30 ± 0.11</td>
<td>0.43 ± 0.11</td>
<td>1.53 ± 0.11</td>
<td>0.72 ± 0.11</td>
<td>1.15 ± 0.11</td>
<td>2.31 ± 0.27</td>
<td></td>
</tr>
<tr>
<td>1 week</td>
<td>1.29 ± 0.10</td>
<td>0.42 ± 0.10</td>
<td>1.52 ± 0.10</td>
<td>0.72 ± 0.10</td>
<td>1.15 ± 0.10</td>
<td>2.31 ± 0.27</td>
<td></td>
</tr>
<tr>
<td>Over 2 months</td>
<td>1.35 ± 0.13</td>
<td>0.47 ± 0.13</td>
<td>1.47 ± 0.13</td>
<td>0.72 ± 0.13</td>
<td>1.15 ± 0.13</td>
<td>2.31 ± 0.27</td>
<td></td>
</tr>
<tr>
<td>Initial period</td>
<td>1.60 ± 0.08</td>
<td>0.49 ± 0.08</td>
<td>1.53 ± 0.08</td>
<td>0.72 ± 0.08</td>
<td>1.15 ± 0.08</td>
<td>2.31 ± 0.27</td>
<td></td>
</tr>
<tr>
<td>1 month</td>
<td>1.31 ± 0.06</td>
<td>0.48 ± 0.06</td>
<td>1.52 ± 0.06</td>
<td>0.72 ± 0.06</td>
<td>1.15 ± 0.06</td>
<td>2.31 ± 0.27</td>
<td></td>
</tr>
<tr>
<td>Over 3 months</td>
<td>1.25 ± 0.08</td>
<td>0.47 ± 0.08</td>
<td>1.46 ± 0.08</td>
<td>0.72 ± 0.08</td>
<td>1.15 ± 0.08</td>
<td>2.31 ± 0.27</td>
<td></td>
</tr>
<tr>
<td>Healthy persons</td>
<td>1.31 ± 0.08</td>
<td>0.49 ± 0.08</td>
<td>1.53 ± 0.08</td>
<td>0.72 ± 0.08</td>
<td>1.15 ± 0.08</td>
<td>2.31 ± 0.27</td>
<td></td>
</tr>
</tbody>
</table>

Designations: * -- statistically significant discrepancies vs. healthy persons.
The disordered blood electrolyte composition did not fully normalize even after two or more months following the patients' transition to normal motor activity.

Shifts in blood electrolytes for the patients on bedrest caused the general body asthenization and disordered cardiac activity we noted earlier in the same patients, (V. P. Zakutayeva, 1976). Hence, treatment of patients with osteoarticular tuberculosis on bedrest requires systematic observation of blood electrolyte composition with corresponding correction of the imbalance as needed.

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