Vertebral bone loss: an unheeded side effect of therapeutic bed rest

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Summary
1. The skeletal effects of simple bed rest and re-ambulation were studied in a consecutive series of 34 patients (aged 18–60 years) hospitalized with low backache due to protrusion of a lumbar intervertebral disc. The bone mineral content of the second, third and fourth lumbar vertebrae was determined by dual-photon (153Gd) absorptiometry immediately after admission to the hospital, at the end of the bed-rest period (mean 27 days, range 11–61 days) and approximately 15 weeks later (range 11–24 weeks).
2. During recumbency a mean decrease in lumbar spine bone mineral content of 0.9% per week was observed.
3. Re-ambulation resulted in bone mineral gain, and restoration of lumbar spine bone mineral content was nearly complete after 4 months.
4. The findings suggest that the simple therapeutic bed-rest regimen leads to excessive vertebral bone loss. Recurrent bed-rest periods may predispose to spinal osteoporosis.

Key words: bone mineral content, immobilization, osteoporosis, spine.

Introduction
Weight-bearing and physical activity are important mechanical stimuli to bone growth and bone remodelling [1]. The inferential concept of a close relationship between bone mass and functional loading is strongly supported by clinical observations. The localized bone loss that follows fracture immobilization [2, 3] and the disuse osteopenia of strict bed rest [4–6] and weightlessness [7] are well known. A similar effect of the commonly prescribed bed-rest regimen has not been recognized [8]. Any elucidation of the possible consequences of short-term bed rest has been hampered by the lack of methods capable of detecting minor changes in the bone mass of the weight-bearing axial skeleton.

With the use of the technique of dual-photon absorptiometry [9] the present study was undertaken to investigate whether the bone mineral content of the second, third and fourth lumbar vertebrae was influenced by simple therapeutic bed rest and re-ambulation.

Material and methods
The subjects comprised a consecutive series of 34 patients (17 females, 17 males) admitted to hospital with low backache due to protrusion of a lumbar intervertebral disc. Their mean age was 38 years (range 18–60 years). The diagnosis of the disc disease was based on common clinical criteria. None had other spinal abnormalities.

The standard treatment was bed rest, analgesic and anti-inflammatory drugs and traction. The diet was unrestricted. All patients were allowed to go to the lavatory and bathroom (i.e. simple bed rest). A daily programme of active physical exercise was conducted by one of the physiotherapists. It was administered with the patient in recumbency. Any loading of the vertebrae was omitted. Re-ambulation was initiated if the patient showed obvious clinical improvement. Twelve patients were referred to subacute discus surgery and another three received surgical treatment during re-ambulation.
Lumbar bone mineral content was determined by dual-photon ($^{153}$Gd) absorptiometry as previously described [9, 10]. Lumbar bone mineral content was expressed in arbitrary units having the dimension of mass. The SD of repeated measurements in normal subjects was 1.0 unit (bone mineral content range 36.5–65.3 units), corresponding to a coefficient of variation of 2%. The patients were investigated immediately after admission to the hospital ($n = 34$), at the end of the bed-rest period (mean 27 days, range 11–61 days; $n = 22$) and after re-ambulation for approximately 15 weeks (range 11–24 weeks; $n = 15$).

Serum concentrations of calcium, inorganic phosphate, total alkaline phosphatase and creatinine, and 24-h urinary excretion of calcium, phosphorus and creatinine were measured weekly during recumbency and once after re-ambulation. Blood and urine samples were determined by routine methods. Serum calcium concentration was corrected to a serum albumin concentration of 0.70 mmol/l.

Informed consent to participation was obtained from all patients in accordance with the Helskinki II Declaration. Patients later subjected to surgery were subsequently excluded. The post-ambulation examination was missed by four patients.

Student’s t-test for paired samples was used for statistical comparisons. $P$ values less than 0.05 were considered significant. The changes in bone mineral content during bed rest were calculated as a rate: the difference between the paired measurements divided by the time interval between the measurements. Results are presented as means ± SEM.

**Results**

The mean lumbar bone mineral content of the 34 patients who entered the investigation amounted to 104 ± 4% of the mean of sex- and age-matched normal subjects (not significant, N.S.). Fig. 1 illustrates the individual changes in lumbar bone mineral content during the study. The mean total change during bed rest was a decrease of 1.66 ± 0.57 units or 3.6 ± 1.2%. The mean rate of bone loss was 0.40 ± 0.15 unit per week or 0.9 ± 0.3% per week ($n = 22$, $P < 0.01$). The predicted involutional bone loss in such patients averaged 1.0 ± 0.5% per year [10, 11].

![Fig. 1. Individual changes in lumbar spine bone mineral content during the study. Paired measurements are connected by straight lines. In those patients who were measured again after ambulation, a change in the measured trend in mineral content is indicated by angulation of the line at the second measurement. The bold line connects mean values.](image-url)
Bone loss during immobilization

Table 1. Biochemical variables at admission, during bed rest and after re-ambulation for approximately 15 weeks

Bed rest and post-ambulation values are expressed as derivations from the values obtained at admission. Significance of deviations (paired t-test): *P < 0.05.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Admission (n = 34)</th>
<th>Bed rest (n = 22)</th>
<th>Post-ambulation (n = 15)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean</td>
<td>SD</td>
<td>Mean deviation</td>
</tr>
<tr>
<td>Serum calcium (corrected) (mmol/l)</td>
<td>2.29</td>
<td>0.07</td>
<td>0.03*</td>
</tr>
<tr>
<td>Inorganic phosphate (mmol/l)</td>
<td>1.14</td>
<td>0.18</td>
<td>0.07*</td>
</tr>
<tr>
<td>Alkaline phosphatase (units/l)</td>
<td>145</td>
<td>42</td>
<td>-4</td>
</tr>
<tr>
<td>Creatinine (mmol/l)</td>
<td>87</td>
<td>15</td>
<td>0</td>
</tr>
<tr>
<td>Urine calcium (mmol/24 h)</td>
<td>5.0</td>
<td>2.6</td>
<td>0</td>
</tr>
<tr>
<td>Phosphorus (mmol/24 h)</td>
<td>24</td>
<td>10</td>
<td>3</td>
</tr>
<tr>
<td>Creatinine (mmol/24 h)</td>
<td>10.8</td>
<td>3.0</td>
<td>1.3*</td>
</tr>
</tbody>
</table>

The 15 patients (nine males, six females) who completed the entire study, showed a mean rate of bone loss of 0.62 ± 0.17 unit per week or 1.2 ± 0.4% per week. After re-ambulation for approximately 15 weeks an apparent bone mineral gain of 1.59 ± 0.89 units or 4.4 ± 2.2% was demonstrated (n = 15, N.S.). The predicted increase in lumbar bone mineral content was 4.7 ± 2.2% (n = 15, P < 0.05) after allowance for the involutional diminution. The post-ambulation lumbar bone mineral content was 0.54 ± 0.51 unit lower than that at admission (n = 15, N.S.).

The biochemical findings are summarized in Table 1. During recumbency there was a small but significant increase in serum calcium concentration (1.3 ± 0.5%), serum phosphorus concentration (5.9 ± 2.8%) and 24-h urinary excretion of creatinine (17 ± 7%). No significant change in 24-h urinary excretion of calcium and phosphorus was found.

Discussion

The hazards of therapeutic bed rest have been reviewed by Browse [12]. Impaired work performance and vasomotor instability need no emphasis. Our results provide evidence of an excessive vertebral bone loss during simple short-term bed rest. The present decrease in lumbar bone mineral content of 1% per week during simple short-term bed rest is nearly 50 times larger than the predicted involutional diminution [10, 11]. The enhanced creatinine excretion suggests an associated rapid decrease in muscle mass. The findings are in agreement with the results obtained by Hansson et al. [13], who demonstrated a bone loss from the fourth lumbar vertebra of nearly 2% per week during strict bed rest after scoliosis surgery. However, we found no obvious increase in calcium excretion as reported in prolonged strict bed rest [4, 5].

A localized vertebral bone loss of 1% per week might result in a negative calcium balance of 2–8 mmol/24 h if compensatory mechanisms were not operative [14]. An unchanged urinary calcium excretion as shown here presumes that short-term recumbency is accompanied by a diminished net absorption of calcium or redistribution of bone mineral within the skeleton (e.g. formation of new bone in other parts of the skeleton). Little is known about mineral redistribution. However, the dietary intakes of calcium and other nutrients are likely to be decreased during bed rest, the diet being unrestricted, and parathyroid activity might be suppressed by the slightly increased serum concentration of calcium, leading to malabsorption of calcium [15].

It has been suggested that the rapidly remodeling trabecular bone is more sensitive to a cessation of mechanical loading than is compact bone [4]. This would imply that disuse osteopenia predominantly occurs in the trabecular part of the weight-bearing skeleton. The localized vertebral bone loss observed in the current study supports this hypothesis.

The fact that re-ambulation tends to reverse the decline in lumbar bone mineral content suggests that the bone loss and bone gain simply reflect an adaptive response to decreasing (immobilization) and increasing (gravity stress, physical activity) degrees of vertebral strain [1]. This would explain the apparent failure of medical prevention of disuse osteopenia [16]. A daily 3-h exposure to gravity stress seems to prevent calcium loss in young normal subjects [17]. It can be seen that the rate of bone restoration is much lower than the rate of bone loss.
The involutional bone loss from the lumbar vertebrae in normal women immediately after the menopause amounts to nearly 7% per year [10]. The present study comprised only two post-menopausal women. However, the post-ambulation bone mineral gain of 1% per month suggests that spinal osteoporosis to some extent might be prevented by increasing the functional loading on the vertebrae either by weight-bearing or by physical training [18, 19]. Recurrent bed rest is probably a risk factor in the genesis of spinal osteoporosis.

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References


