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An age-related medullary expansion can have implications for the long-term fixation of hip prostheses

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Background  Diaphyseal bone loss occurs mainly at the endosteal surface in the medullary cavity. Since the menopause is followed by an increase in bone loss, the size of the medullary cavity should theoretically increase during the postmenopausal period. If so, this might affect the long-term fixation of hip prostheses.

Patients and methods  This 19-year prospective study evaluated bone loss and geometrical changes in 112 women, all premenopausal at baseline. Bone mineral density (BMD) and skeletal geometry, with special reference to the size of the medullary cavity, were estimated every other year by single-photon absorptiometry at the cortical site of the distal radius.

Results  After menopause, a decrease of 1.7 (95% CI 1.6–1.8)% occurred in the BMD every year, while an increase of 0.9 (0.8-1.0)% in the medullary width (endosteal width) took place every year. The annual change in BMD was inversely correlated with the annual change in the medullary width (r = −0.5, p < 0.001). The quartile of women with the largest BMD loss had a greater medullary expansion than the quartile of women with the least BMD loss (p < 0.001).

Interpretation  If the age-related expansion of the radial medullary cavity is a general phenomenon, this may have implications for the long-term fixation of hip prostheses.

Estrogen deficiency, after the menopause, accelerates the age-related bone loss (Riggs et al. 1986, Ahlborg et al. 2001). Since the bone loss of the diaphyseal bones occurs mainly at the endosteal surface of the cortex (the inner surface of the cortex) and partly in the Haversian canals (Frost 1999), the medullary cavity should, hypothetically, increase in size after the menopause. Most studies evaluating the age-related structural changes include bone size alone, but, not changes in the endosteal and subperiosteal surfaces separately, and mainly in cadaveric and cross-sectional studies, with the risk of selection bias and cohort effects, which may confound the conclusions (Ruff and Hayes 1988, Bouxsein et al. 1994, Heaney et al. 1997, Mosekilde 1990). Case-control and cross-sectional studies, which suggest that the medullary cavity expands with age, have been published (Poss et al. 1987, Hofmann et al. 1989, Robinson et al. 1994), but if this could be verified in prospective studies, it would further strengthen the findings. If the medullary cavity increases in size with age, this might have implications for the long-term fixation of hip prostheses.

With this background, we hypothesized that the postmenopausal bone loss of the diaphyseal bones expands the medullary cavity and posed the questions: (i) is the BMD loss after the menopause associated with medullary expansion? (ii) do women with a high rate of BMD loss after menopause also have a greater expansion of the medullary cavity than women with a less significant BMD loss?

Patients and methods  We invited 241 Caucasian women aged 48 from Malmö, Sweden to this prospective study (Johnell and Nilsson 1984, Ahlborg et al. 2001). 49 were
excluded before baseline, as all participants in
the study had to be premenopausal and without
medication or disease known to interfere with bone
metabolism. Therefore 192 women were included
in the study in 1977–1978. A further 17 persons
were excluded, since they left the study during
the first 5 years because of surgical menopause or
relocation, 4 had errors in technical measurements,
17 had received estrogen treatment and 15 died,
leaving 139 women to be followed through meno-
pause. This report includes 112 women (81%) who
participated throughout the entire 23 years. Prospective
16-year data as regards bone loss and bone strength have been reported elsewhere (Ahl-

The last premenopausal measurement, made 2
years at most before the menopause, was defined
as menopause and therefore the baseline measure-
ment. The last measurement was done at 72 years
of age, but as the women had their menopause at
various ages, the mean postmenopausal follow-
up period was 19 (14–23) years. Menopause was
defined using the definitions of the WHO (1981)
—i.e., the permanent cessation of menstruation
due to the loss of ovarian follicular activity. There-
fore the onset of the menopause was determined
retrospectively, after 12 months of spontaneous
amenorrhea together with elevated serum levels
of follicle-stimulating hormone. This hormone
was analyzed by double antibody radio immuno-
assays every 3 months during the first year, every
6 months until one year after the menopause and
then once a year (Rannevik et al. 1995).

Bone mineral density (BMD, mg/cm²) of the
distal radius was measured 6 cm proximal to the
ulnar styloid process, on the average, every other
year, by single-photon absorptiometry (SPA), a
total of 11 times. A rectilinear scan across the
radius and ulna, with a radiation source (241Am)
and a detector moving simultaneously, was taken
using Nauclér et al.’s method (1974). Both fore-
arms were scanned and all traits presented below
are the average value of the right and left forearms.
The bone size (subperiosteal width), the medullary
width (endosteal width) and the cortical width of
the distal radius were calculated from the graph of
the scan (Nauclér et al. 1974).

The reproducibility was 4% (coefficient of varia-
tion) in vivo, determined by duplicate measure-
ments in 20 persons measured twice at intervals
of weeks or months apart. The estimate of cortical
thickness from the graph of the scan had a repro-
cducibility of 8% (coefficient of variation) and was
found to be proportional to the cortical thickness on
x-rays from the same site (Nauclér et al. 1974). In
this study the same densitometer was used through-
out the study and, during follow-up, repeated mea-
surements of a standardized phantom were taken
every other week. To determine whether long-term
drift of the densitometer occurred during follow-
up, all phantom data were analyzed, using a linear
regression equation. This analysis showed no sig-
nificant long-term drift of the equipment, either in
BMD or skeletal width estimates (0.1%/year (95%
CI –0.2–0.4) and 0.08%/year (95% CI –0.01–0.17),
respectively). Due to a replacement of the radiation
source in 1980, all the following BMD measure-
ments were adjusted by using the phantom data.

The data are given for all 112 participants and
have been divided into quartiles according to the
annual relative changes in BMD. The annual per-
centage change was calculated for each woman
as the ratio of the slope fitted to each woman’s
repeated measurements divided by the baseline
value. Figure 1 shows the relative change as the
ratio of the value observed at each time as com-
pared to each woman’s baseline value. A linear
regression equation was used to determine the
association between annual change of BMD and
medullary width. Student’s t-test between means
was used to compare women within the highest
and the lowest quartiles of BMD loss, with adjust-
ment for menopausal age by analysis of covari-
ance. The data are presented as the mean, with a
95% confidence interval (95% CI). The study was
approved by the local ethics committee.

Results
The BMD declined annually by 1.7 (95% CI 1.6–
1.8)% while the medullary width increased by 0.9
(0.8–1.0)% and the bone size by 0.6 (0.6–0.7)%
during the two decades after menopause (Table
1, Figure 1). The absolute changes in BMD and
the absolute changes in medullary width were cor-
related (r = –0.6, p < 0.001), as also were the rela-
tive changes (r = –0.5, p < 0.001) (Figure 2).
The quartile of women with the largest BMD loss (> 2.1% a year, n = 28) had, as compared to the quartile of women with the least BMD loss (< 1.3% a year, n = 28) a greater annual medullary expansion (1.0 (0.7–1.4)% p < 0.001) and a greater annual bone size expansion (0.3 (0.2–0.4), p < 0.001) (Table 2). After adjustment for baseline differences in age at menopause, the findings were the same (1.0 (0.7–1.4)% and 0.3 (0.2–0.5)%, respectively). That is, the women with the greatest BMD loss had the greatest medullary expansion and the greatest gain in bone size (Figure 3).

Table 1. Skeletal structure and bone mass in the distal radius, measured 6 cm proximal to the styloid process of the ulna by single photon absorptiometry (SPA), and anthropometric data at menopause (MP) with the absolute and relative changes a year estimated by individual regression slopes until the age of 72 years

<table>
<thead>
<tr>
<th>Measurement at menopause</th>
<th>Absolute change a year</th>
<th>Relative change a year (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>(n = 112)</td>
<td>(n = 112)</td>
<td>(n = 112)</td>
</tr>
<tr>
<td>mean 95% CI</td>
<td>mean 95% CI</td>
<td>mean 95% CI</td>
</tr>
<tr>
<td>Skeletal structure</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Bone size (mm)</td>
<td>13.0</td>
<td>0.08</td>
</tr>
<tr>
<td></td>
<td>12.8 to 13.2</td>
<td>0.07 to 0.08</td>
</tr>
<tr>
<td>Medullary width (mm)</td>
<td>6.8</td>
<td>0.06</td>
</tr>
<tr>
<td></td>
<td>6.5 to 7.0</td>
<td>0.05 to 0.06</td>
</tr>
<tr>
<td>Cortical width (mm)</td>
<td>6.2</td>
<td>0.02</td>
</tr>
<tr>
<td></td>
<td>6.1 to 6.4</td>
<td>0.01 to 0.03</td>
</tr>
<tr>
<td>Bone mass BMD (mg/cm²)</td>
<td>559</td>
<td>-9.7</td>
</tr>
<tr>
<td></td>
<td>549 to 570</td>
<td>-10 to -9.0</td>
</tr>
<tr>
<td>Anthropometrics</td>
<td></td>
<td>-1.73</td>
</tr>
<tr>
<td>Height (cm)</td>
<td>164</td>
<td>-1.84</td>
</tr>
<tr>
<td></td>
<td>163 to 165</td>
<td>-1.62</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>64</td>
<td>-0.1</td>
</tr>
<tr>
<td></td>
<td>62 to 66</td>
<td>-0.1 to -0.1</td>
</tr>
<tr>
<td>Age at MP</td>
<td>52</td>
<td>-0.07</td>
</tr>
<tr>
<td></td>
<td>52 to 53</td>
<td>-0.08 to -0.06</td>
</tr>
</tbody>
</table>

a p < 0.001, when significantly different from zero
Discussion

In this prospective study, we report (i) that the BMD loss following menopause is associated with a medullary expansion and (ii) that women with a large loss risk a large expansion. This study has several advantages: (i) a well-defined, female, Caucasian population was followed, (ii) all were 48 years of age at baseline, (iii) all lived in the same city, (iv) the estimate of the menopause was accurate, (v) the high attendance rate was maintained throughout the 23 years, (vi) no diseases or medication interfered with the normal skeletal development and confounded the data, (vii) repeated SPA measurements were done by the same technician, using the same densitometer, and (viii) all the analyses were done by the same technician. To perform a similar study today would be almost impossible, because of the present use of bisphosphonates and selective estrogen receptor modulators (SERMS) (Black et al. 1996, Ettinger et al. 1999, McClung et al. 2001). Due to, with the large intra-individual variations in the shape of the bone and the documented changes in skeletal geometry with aging (Bouxsein et al. 1994, Heaney et al. 1997), a pro-

Table 2. Skeletal structure and bone mass in the distal radius, measured 6 cm proximal to the styloid process of the ulna by single photon absorptiometry (SPA), and anthropometric data at menopause (MP), with absolute and relative changes a year estimated by individual regression slopes until the age of 72 years in the quartile of women with the largest BMD loss (> 2.1%/year) and in the quartile of women with the least BMD loss (< 1.3%/year)

<table>
<thead>
<tr>
<th>Measurement at menopause</th>
<th>Absolute change in BMD loss per year</th>
<th>Relative change in BMD loss per year (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Largest BMD loss (n = 28)</td>
<td>Least BMD loss (n = 28)</td>
<td>Largest BMD loss (n = 28)</td>
</tr>
<tr>
<td>Bone size (mm)</td>
<td></td>
<td>Bone size (mm)</td>
</tr>
<tr>
<td>13.1</td>
<td>12.8</td>
<td>0.09</td>
</tr>
<tr>
<td>12.7 to 13.6</td>
<td>12.4 to 13.3</td>
<td>0.08 to 0.11</td>
</tr>
<tr>
<td>Medullary width (mm)</td>
<td></td>
<td>0.09</td>
</tr>
<tr>
<td>7.0</td>
<td>6.6</td>
<td>0.09</td>
</tr>
<tr>
<td>6.5 to 7.5</td>
<td>6.1 to 7.0</td>
<td>0.07 to 0.11</td>
</tr>
<tr>
<td>Cortical width (mm)</td>
<td></td>
<td>0.004</td>
</tr>
<tr>
<td>6.1</td>
<td>6.3</td>
<td>0.004</td>
</tr>
<tr>
<td>5.9 to 6.3</td>
<td>6.0 to 6.6</td>
<td>–0.01 to 0.02</td>
</tr>
<tr>
<td>Bone mass</td>
<td></td>
<td>–0.14 to 0.31</td>
</tr>
<tr>
<td>BMD (mg/cm&lt;sup&gt;2&lt;/sup&gt;)</td>
<td></td>
<td>–13.6</td>
</tr>
<tr>
<td>552</td>
<td>563</td>
<td>–14.5 to –12.6</td>
</tr>
<tr>
<td>528 to 575</td>
<td>541 to 586</td>
<td>–2.59 to –2.34</td>
</tr>
<tr>
<td>Anthropometrics Height (cm)</td>
<td></td>
<td>165</td>
</tr>
<tr>
<td>165</td>
<td>163</td>
<td>–0.2 to –0.1</td>
</tr>
<tr>
<td>163 to 166</td>
<td>161 to 165</td>
<td>–0.2 to –0.1</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td></td>
<td>0.3</td>
</tr>
<tr>
<td>66</td>
<td>67</td>
<td>0.3</td>
</tr>
<tr>
<td>61 to 71</td>
<td>62 to 72</td>
<td>0.1 to 0.5</td>
</tr>
<tr>
<td>Age at MP</td>
<td></td>
<td>0.18 to 0.71</td>
</tr>
<tr>
<td>53</td>
<td>51 &lt;sup&gt;a&lt;/sup&gt;</td>
<td></td>
</tr>
<tr>
<td>52 to 54</td>
<td>51 to 52</td>
<td></td>
</tr>
</tbody>
</table>

<sup>a</sup> p = 0.03  
<sup>b</sup> p < 0.001, when comparing the two quartiles

Figure 3. Estimated mean cross-sectional geometry of the cortical site of the distal radius at menopause and at the age of 72 years, in the quartile of women with the largest BMD loss (>2.1%/year) and in the quartile of women with the least BMD loss (<1.3%/year). Dotted line indicates the inner cortical surface at menopause.
spective study design is preferable for studying the age-related, geometrical changes. Since the study was originally designed to follow changes in bone density of the distal radius, its weakness is that only one diaphyseal bone was evaluated. We can therefore assume only that the skeletal remodeling observed is a general phenomenon found in all diaphyseal bones. Earlier human cross-sectional and cadaveric studies of the femur shaft support this view since they found an age-related increase in medullary width and bone size (Smith and Walker 1964, Ruff and Hayes 1988).

Some reports indicate that loosening of hip prostheses may be associated with an expansion of the medullary cavity (Poss et al. 1987, Hofmann et al. 1989, Robinson et al. 1994). Hofmann et al. (1989) found, in a case-control study including 60 persons with total hip arthroplasty (THA), a four times higher medullary expansion of the femur in the group with aseptic femoral stem loosening than in a group with stable hip prostheses. Radiostereometric studies have shown that most of the motion between the femur and the implant takes place at the cement-implant interface during the first two postoperative years (Nivbrant et al. 1999), but also to some extent at the bone-cement interface. The motion at this interface seems to occur whether or not the implant is designed to adhere to the cement or has a tapered form with smooth surface designed to move inside the cement mantle (Alfaro-Adrian et al. 1999, Nivbrant 1999). Long-term retrieval studies have shown that implants which have functioned well for many years have signs of trabecular bone in direct contact with the cement mantle, and radiolucent lines at the bone-cement interface. This phenomenon has been attributed to endosteal remodeling (Schmalzried et al. 1993). Our hypothesis is that late loosening, as opposed to early loosening, may, to some extent, be due to medullary expansion.

Moreover, as bone loss occurs mainly at the endosteal surface, it is not surprising to find a larger medullary expansion in those with a high loss of BMD. Treatment with bisphosphonates would then reduce not only the resorption of trabecular bone, but theoretically also the medullary expansion by inhibiting osteoclastic activity. As a result, this might reduce the effect at the bone-cement interface (Hilding et al. 2000, Åstrand and Aspenberg 2002). Some short-term data support this view by suggesting that early migration of the tibial component after total knee replacement can be inhibited by anti-resorptive drugs (Hilding et al. 2000).

The mean age of menopause in Sweden is 52 years, as in this study, while the mean age of women undergoing THA is generally higher. Since the BMD loss and the medullary expansion in the current study seem to occur preferably during the first 15 years after menopause, it is possible that the consequences of medullary expansion as regards THA are confined to women undergoing surgery before or shortly after this. Future studies in the field should therefore include women undergoing surgery before the age of 60 years.

No competing interests declared.


