

# Effect of nonweight bearing on tibial bone density measured by QCT in patients with hip surgery

MASAKO ITO<sup>1</sup>, TOMOKO MATSUMOTO<sup>2</sup>, HIROSHI ENOMOTO<sup>2</sup>, KUMIKO TSURUSAKI<sup>1</sup>, and KUNIYUKI HAYASHI<sup>1</sup>

<sup>1</sup>Department of Radiology, Nagasaki University School of Medicine, 1-7-1 Sakamoto, Nagasaki 852-8105, Japan

<sup>2</sup>Department of Orthopedic Surgery, Nagasaki University, School of Medicine, Nagasaki, Japan

**Abstract:** Tibial bone mineral density (BMD) was measured in 11 patients who had undergone hip joint surgery, including 10 women (22–61 years old; mean  $\pm$  SD =  $42.6 \pm 10.3$ ) and 1 man (61 years old). Four patients received total hip replacement (THR), while the others underwent rotational acetabular osteotomy (RAO). In one case, the start of rehabilitation was delayed until 4 months after the surgery because of infection at the surgical site. Nine patients underwent hip surgery for osteoarthritis and 2 patients for avascular necrosis. These 2 patients had a history of medication with corticosteroid. BMD of the tibia on the surgically treated side was measured by a peripheral quantitative CT (pQCT) system, which provided three different BMD values of trabecular BMD in the distal portion, total BMD in the distal portion, and total BMD in the diaphysis. The measurements were obtained preoperatively, and at several time points postoperatively, at 2, 4, 6, and 8 weeks and 3, 4, 5, 8, 12, 18, and 24 months after surgery. Brief periods of nonweightbearing lead to significant bone loss, and 1–1.5 years was required to recover to the baseline BMD. Accelerated bone loss was seen in patients in the perimenopausal state, with prolonged bed rest, and in patients receiving corticosteroid. Both trabecular and cortical components were influenced by nonweightbearing and restoration of weightbearing. The decrease in the cortical region occurs after the decrease in the trabecular and endosteal regions.

**Key words:** disuse osteoporosis, nonweightbearing, peripheral quantitative computed tomography (pQCT), bone mineral density (BMD)

## Introduction

Disuse osteoporosis is a localized or generalized loss of bone mineral density after a period of inactivity or immobilization subsequent to trauma or nontraumatic

conditions. The time course of disuse osteoporosis has been investigated. Several reports showed the greatest bone mass deficit was seen 4 months after fracture, and bone mass continued to decrease even after immobilization had been discontinued and the patients had returned to full weightbearing on the involved limb. Furthermore, there was no significant regeneration of lost bone after 1 year [1,2].

The previous reports determined bone loss after immobilization using noninvasive bone mineral measurements. Dual energy x-ray absorptiometry (DXA) is reliable for assessing bone loss in any bone site suspected of bone loss, and quantitative computed tomography (QCT) can selectively determine trabecular bone loss in the spine. Peripheral quantitative computed tomography (pQCT) is a QCT method to measure bone mineral density (BMD) of the forearm and leg. In this study, pQCT was used as a noninvasive method of assessing bone loss after nonweightbearing and to assess bone gain after restoration of weightbearing. It is possible to precisely assess both apparent density in the trabecular and cortical regions using this pQCT apparatus because it provides very high reproducibility using a multi-slice technique and area-adjusting method for repeated measurements [3]. This study investigated the pattern of changes in BMD demonstrated by pQCT after brief periods of nonweightbearing and restoration of weightbearing in patients receiving hip surgery.

## Materials and methods

Tibial BMD was measured in 11 patients who had undergone hip joint surgery, including 10 women (22–61 years old; mean  $\pm$  SD =  $42.6 \pm 10.3$ ) and 1 man (61 years old). Four patients received total hip replacement (THR), while the others underwent rotational acetabular osteotomy (RAO). The details are shown in

Offprint requests to: M. Ito

Received: June 5, 1998 / Accepted: Aug. 25, 1998

**Table 1.** Age, sex, menopausal state, underlying disease, baseline BMD, and its T-score in individual patients

| Case | Age (years) | Sex | Menstruation | Disease | Operation | BMD in baseline study     |         |                            |         |                            |         | Remarks                            |
|------|-------------|-----|--------------|---------|-----------|---------------------------|---------|----------------------------|---------|----------------------------|---------|------------------------------------|
|      |             |     |              |         |           | D50 (mg/cm <sup>3</sup> ) | T-score | D100 (mg/cm <sup>3</sup> ) | T-score | P100 (mg/cm <sup>3</sup> ) | T-score |                                    |
| 1    | 22          | f   | Premeno.     | OA      | rt. RAO   | 370                       | +2.9    | 757                        | +2.5    | 1156                       | +1.0    |                                    |
| 2    | 34          | f   | Premeno.     | OA      | rt. RAO   | 121                       | -1.7    | 440                        | -1.9    | 836                        | -2.5    | Bed rest for 3 months              |
| 3    | 39          | f   | Premeno.     | AVN     | lt. THR   | 51                        | -2.9    | 303                        | -3.9    | 746                        | -3.5    | Receiving PSL more than 10yrs; SLE |
| 4    | 40          | f   | Premeno.     | OA      | rt. RAO   | 358                       | +6.8    | 733                        | +6.1    | 1121                       | +3.4    |                                    |
| 5    | 41          | f   | Premeno.     | OA      | lt. RAO   | 208                       | +2.0    | 641                        | +3.7    | 1199                       | +5.8    |                                    |
| 6    | 44          | f   | Premeno.     | OA      | lt. RAO   | 277                       | +4.2    | 719                        | +5.8    | 1183                       | +5.3    |                                    |
| 7    | 48          | f   | Perimeno.    | AVN     | rt. THR   | 101                       | -2.2    | 398                        | -1.8    | 818                        | -1.4    | Received PSL for 8yrs; PPH         |
| 8    | 48          | f   | Perimeno.    | OA      | rt. THR   | 138                       | -1.2    | 563                        | -0.3    | 917                        | -0.8    |                                    |
| 9    | 49          | f   | Premeno.     | OA      | lt. RAO   | 200                       | +0.4    | 685                        | +0.9    | 1055                       | +0.2    |                                    |
| 10   | 61          | f   | Postmeno.    | OA      | rt. RAO   | 101                       | -2.7    | 400                        | -6.4    | 762                        | -7.1    |                                    |
| 11   | 61          | m   | —            | OA      | lt. THR   | 200                       |         | 564                        |         | 1194                       |         |                                    |

BMD, bone mineral density; Premeno., premenopausal; Perimeno., perimenopausal; Postmeno., postmenopausal; OA, osteoarthritis; AVN, avascular necrosis; RAO, rotational acetabular osteotomy; THR, total hip replacement; D50, the mean bone density in a core area of the distal metaphysis; D100, the mean bone density of the whole cross-sectional area of the distal metaphysis; P100, the mean bone density of the whole cross-sectional area of the diaphysis

Table 1. In case 2, the patient started rehabilitation 4 months after surgery because of infection at the surgical site. Nine patients underwent hip surgery for osteoarthritis and two patients for avascular necrosis (case 3 and case 7). These two patients had a history of medication with corticosteroid. Case 3 had received corticosteroid for systemic lupus erythematosus (SLE) for more than 10 years, and case 7 had received corticosteroid for 8 years (1984–1992) for primary pulmonary hypertension (PPH), which remained stable during the observation period. Tibial pQCT data from 130 healthy volunteer women ( $55.5 \pm 12.3$  years) were used as control data.

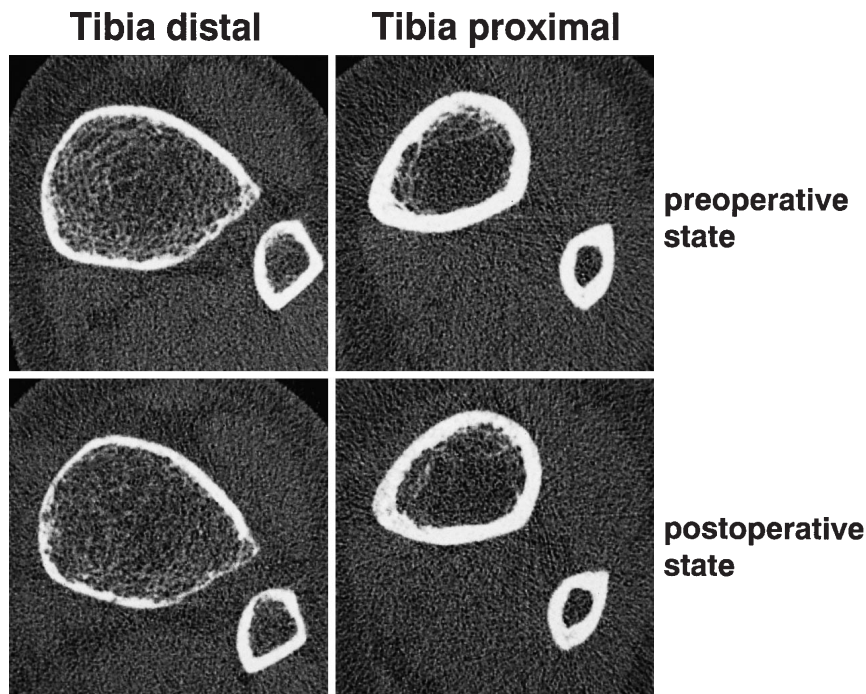
Activities in bed without weightbearing were allowed soon after surgery, and partial weightbearing was resumed 2 months after surgery. Bearing one-third of body weight was started at 2 months, two-thirds after 3 months, and full weightbearing was resumed 4 months after surgery. Postoperative rehabilitation followed this course in all patients except case 2.

Bone mineral density of the tibia on the surgically treated side was measured using the pQCT system Densiscan 1000 (ScancoMedical, Zurich, Switzerland). This pQCT system provides three different BMD values of D50, D100, and P100. A 100% value represented the mean bone density of the whole cross-sectional area, and a 50% value represented the mean bone density in a core area of the bone was obtained after peeling off 50% of all picture elements. The 100% value included both cortical and trabecular bone, and this parameter was obtained in the distal portion (D100) and the proximal portion (P100). The 50% value includes only trabecular bone in the distal portion (D50). High-resolution (200- $\mu$ m) images were also obtained using this system. Using this pQCT device, bone mineral density (D50, D100, and P100) was obtained along with two high-resolution images of the distal and proximal regions. Measurements were obtained preoperatively, and at several postoperative time points, basically 2, 4, 6, and 8 weeks and 3, 4, 5, 8, 12, 18, and 24 months after surgery. The endpoint of BMD measurement in each patient was when BMD had nearly recovered to the preoperative value.

The rate of increase and decrease in bone mass were calculated by adapting the linear regression. The paired two-tailed Student's *t*-test was used to determine the significance of differences in BMD after nonweightbearing or after restoration of weightbearing compared to baseline values and to maximally diminished BMD values. Values were represented as mean  $\pm$  standard deviation (SD).  $P < 0.05$  was considered significant.

## Results

Dramatic changes in the distal and proximal tibial high-resolution CT images were observed in case 2. Figure 1



**Fig. 1.** Distal (*left*) and proximal (*right*) tibial high-resolution CT images obtained preoperatively (*top*) and 8 weeks after surgery (*bottom*) in case 2. Tibial distal CT images show the change in trabecular network; tibial proximal CT images show cortical thinning, endosteal resorption, and intracortical porosity

**Table 2.** Bone loss rates and bone gain rates in individual patients

| Case            | Age (years) | Sex | D50                |             |           | D100               |             |           | P100               |             |           |
|-----------------|-------------|-----|--------------------|-------------|-----------|--------------------|-------------|-----------|--------------------|-------------|-----------|
|                 |             |     | Loss rate (%/year) | Vs. control | Gain rate | Loss rate (%/year) | Vs. control | Gain rate | Loss rate (%/year) | Vs. control | Gain rate |
| 1               | 22          | f   | 2.55               | 1.6         | 1.39      | 5.35               | 2.6         | 4.59      | 2.89               | 2.2         | 3.27      |
| 2               | 34          | f   | 26.73              | 16.9        | 7.21      | 26.21              | 12.8        | 3.24      | 19.16              | 14.4        | 4.52      |
| 3               | 39          | f   | 17.28              | 10.9        | 5.70      | 7.00               | 3.4         | 4.00      | 4.22               | 3.2         | 1.03      |
| 4               | 40          | f   | 4.81               | 3.0         | 4.11      | 8.43               | 4.1         | 5.69      | 5.68               | 4.3         | 3.03      |
| 5               | 41          | f   | 4.16               | 2.6         | 1.40      | 3.26               | 1.6         | 0.89      | 3.31               | 2.5         | 2.56      |
| 6               | 44          | f   | 2.89               | 1.8         | 1.58      | 4.19               | 2.0         | 1.41      | 4.87               | 3.7         | 3.82      |
| 7               | 48          | f   | 6.50               | 1.7         | 7.24      | 30.27              | 8.0         | 2.96      | 11.11              | 4.0         | 2.20      |
| 8               | 48          | f   | 14.43              | 3.7         | 11.91     | 20.31              | 5.4         | 1.60      | 9.00               | 3.2         | 1.08      |
| 9               | 49          | f   | 3.18               | 1.6         | 2.66      | 8.93               | 2.4         | 2.88      | 16.77              | 6.0         | 6.08      |
| 10              | 61          | f   | 4.94               | 3.6         | 2.97      | 11.07              | 8.7         | 3.96      | 1.39               | 1.9         | 0.36      |
| 11 <sup>a</sup> | 61          | m   | 4.42               | 3.2         | 2.26      | 2.43               | 1.9         | 2.54      | 2.00               | 1.6         | 1.48      |

The loss rates are compared with menopause-matched control group.

Vs. control, bone loss rate in patient/bone loss rate in control; D50, the mean bone density in a core area of the distal metaphysis; D100, the mean bone density of the whole cross-sectional area of the distal metaphysis; P100, the mean bone density of the whole cross-sectional area of the diaphysis

<sup>a</sup> In case 11, postmenopausal female data were used as control data.

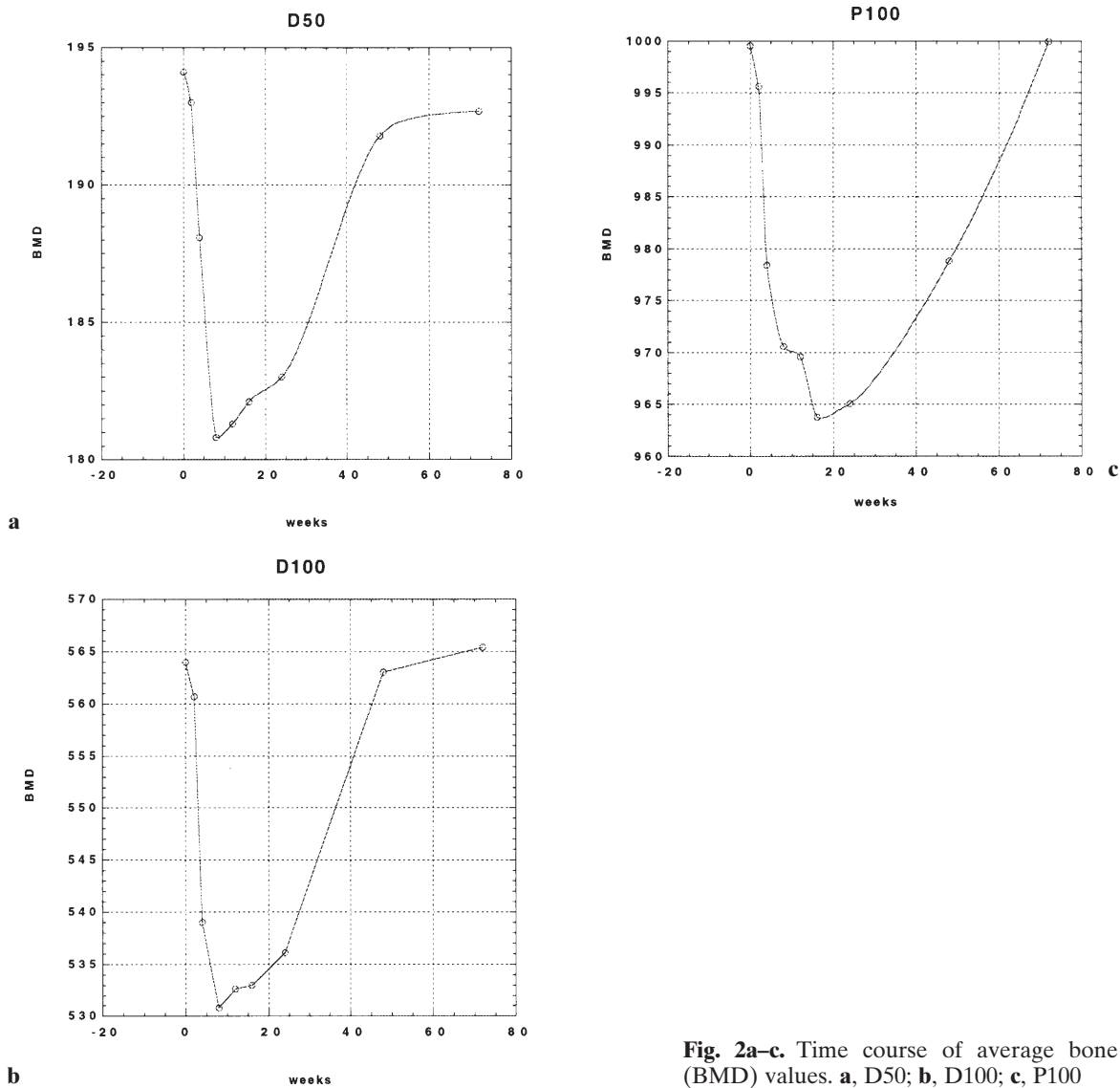
shows the preoperative and 8-week postoperative images in this case. The trabecular network had diminished in the distal portion, and cortical width thinning, endosteal resorption, and intracortical porosity were seen in the proximal portion.

Table 2 shows changes in each BMD value and differences in the bone loss compared to those in the menopausal state-adjusted control group. Accelerated bone loss was seen in patients with prolonged bed rest, in patients who received corticosteroid, and in perimenopausal women. The bone loss rate correlated negatively with the baseline BMD, but the relationship

was not significant:  $r = -0.57$ ,  $P = 0.065$  in D50;  $r = -0.49$ ,  $P = 0.122$  in D100; and  $r = -0.32$ ,  $P = 0.345$  in P100.

Figure 2 shows the time course of average BMD values. There was immediate loss of bone mineral after surgery and a linear decrease in bone density during the postoperative period. Increase in BMD occurred slowly compared with the rate of decrease.

Table 3 shows the average BMD values at the baseline and 2 weeks, 4 weeks, 2 months, 3 months, 4 months, 6 months, 12 months, and 18 months postoperatively in all cases except case 2. It also shows the



**Fig. 2a-c.** Time course of average bone mineral density (BMD) values. **a**, D50; **b**, D100; **c**, P100

difference in BMD compared with both baseline data and the maximal diminished BMD values. D50 (purely trabecular BMD in the distal portion) decreased 6.85% over 2 months at maximum, D100 (both cortical and trabecular bone in distal portion) decreased 5.89% over 2 months, and P100 (both cortical and trabecular bone in proximal portion) decreased 3.57% over 4 months. D50 and D100 returned to the baseline BMD values over 12 months, and P100 returned over 18 months.

BMD continued to decrease for 5 months after surgery in case 2; at the maximum, 35.5% loss in D50, 18.6% loss in D100, and 12.8% loss in P100. The patient of case 3, who received corticosteroid, lost 43.1% of D50, 17.2% of D100, and 12.5% of P100 compared to baseline values. The bone gain rates in these two cases were also accelerated but the gains occurred more slowly than the loss.

## Discussion

The patients in this clinical study showed a wide range of BMD values in the baseline study. Their tibial BMD values were supposed to have been affected by overloading or underloading, as most of these patients had a long history of osteoarthropathy. They also showed a wide range of bone change according to the duration of the nonweightbearing period and restoration of weightbearing. Among 11 patients, accelerated bone loss was seen in perimenopausal patients, in patients with prolonged bed rest, and in patients receiving corticosteroid. Patients receiving corticosteroid or in the perimenopausal state are in a phase of highly increased bone resorption. Although there were only 2 perimenopausal women, the accelerated decrease in bone mass in these patients was in

**Table 3.** Average baseline and postsurgical BMD values

| Average BMD <sup>a</sup> | D50                                |                     |                   | D100                               |                     |                   | P100                               |                     |                   |
|--------------------------|------------------------------------|---------------------|-------------------|------------------------------------|---------------------|-------------------|------------------------------------|---------------------|-------------------|
|                          | Mean ± SD<br>(mg/cm <sup>3</sup> ) | Vs. baseline<br>(P) | Vs. bottom<br>(P) | Mean ± SD<br>(mg/cm <sup>3</sup> ) | Vs. baseline<br>(P) | Vs. bottom<br>(P) | Mean ± SD<br>(mg/cm <sup>3</sup> ) | Vs. baseline<br>(P) | Vs. bottom<br>(P) |
| Baseline                 | 194.1 ± 105.1                      |                     |                   | 564.0 ± 157.7                      |                     |                   | 999.5 ± 185.5                      |                     |                   |
| 2w after surgery         | 193.0 ± 106.0                      | ns                  |                   | 560.7 ± 160.7                      | 0.05                |                   | 995.6 ± 186.6                      | 0.05                |                   |
| 4w after surgery         | 188.1 ± 104.6                      | ns                  |                   | 539.0 ± 186.2                      | 0.05                |                   | 978.4 ± 209.1                      | 0.01                |                   |
| 2m after surgery         | 180.8 ± 110.5                      | 0.05                |                   | 530.8 ± 165.3                      | 0.0005              |                   | 970.6 ± 193.5                      | 0.005               |                   |
| 3m after surgery         | 183.8 ± 110.7                      | 0.05                | ns                | 532.6 ± 171.6                      | 0.0005              | 0.05              | 969.6 ± 199.2                      | 0.005               |                   |
| 4m after surgery         | 185.1 ± 110.5                      | 0.05                | ns                | 533.0 ± 169.7                      | 0.0001              | 0.005             | 963.8 ± 205.3                      | 0.005               |                   |
| 6m after surgery         | 186.0 ± 108.9                      | 0.05                | ns                | 536.1 ± 166.1                      | 0.0005              | 0.05              | 965.1 ± 206.6                      | 0.01                | ns                |
| 12m after surgery        | 191.8 ± 107.6                      | ns                  | ns                | 563.1 ± 160.8                      | 0.01                | 0.05              | 978.8 ± 198.6                      | 0.05                | ns                |
| 18m after surgery        | 192.7 ± 108.2                      | ns                  | ns                | 565.4 ± 160.2                      | ns                  | ns                | 999.9 ± 198.8                      | 0.05                | 0.01              |

<sup>a</sup> Average BMD values at baseline and 2 weeks (w), 4 weeks, 2 months (m), 3 months, 4 months, 6 months, 12 months, and 18 months after surgery for all cases except case 2. The significance of differences (P) are shown in comparison with the baseline BMD value (Vs. baseline) and the maximally decreased BMD value (Vs. bottom)

agreement with the findings of Nilsson and Westlin [4].

The differences in bone loss and restoration patterns between trabecular and cortical bone were investigated. More significant bone loss in spinal trabecular bone after immobilization was demonstrated by QCT in comparison with peripheral cortical bone loss determined by single photon absorptiometry (SPA) shown in an animal study [5]. In the study of prolonged bed rest after surgery, bone loss after immobilization was more significant in the lumbar vertebrae than in the long bones of the extremities [6]. In these studies, axial trabecular bone change was compared with appendicular cortical bone change. We used pQCT to evaluate trabecular and cortical bone change in the same tibia. D50 represents trabecular BMD in the core area of the distal metaphysis; D100 or P100 includes the trabecular and cortical components and endosteal region of the distal metaphysis or diaphysis. D100 mainly represents change in the trabecular and endosteal region, and P100 mainly represents that in the cortical region. The amount of bone loss was greatest although not significant in D50; D100 and P100 showed significant bone loss and significant bone gain with nonweightbearing or restoration of weightbearing. The maximal loss in D50 and D100 appeared after 2 months and returned to the baseline BMD value after 12 months after surgery, while that of P100 appeared after 4 months and recovered after 18 months. Our result indicates that both trabecular and cortical components are affected by nonweightbearing, and that bone loss of the trabecular and endosteal regions reached the maximally diminished value faster and recovered to the baseline BMD faster than the cortical component.

After the brief period of nonweightbearing in our study, there was a great gain of bone mass after restoration of weightbearing. Bone loss after a space flight ranged from 2.9% to 15.1% [7]. Photon absorptiometry suggested that only trabecular bone is affected, but there may be undetectable losses in cortical bone. Precise separate measurement of the trabecular and cortical regions can be useful for investigating changes in each region. BMD decreased promptly after surgery and then increased at a slower rate. A brief period of nonweightbearing did not prevent restoration of BMD, but it required 1–1.5 years to recover to the baseline BMD.

Several reports showed the greatest BMC deficit was seen 4 months after fracture, and BMC continued to fall even after all immobilization had been discontinued. Furthermore, the lack of significant regeneration of the lost bone was observed after 1 year [1,2]. Whether bone loss is reversible or irreversible depends on whether remobilization is begun during or after the active phase of disuse osteoporosis. It is well known that the greatest

recovery in bone mass in immobilized dog limbs occurred when remobilization was begun during the active phase [8]. The responses to restoration of bone may differ between inactivity caused by fracture and postoperative inactivity because the period of inactivity is shorter, the amplitude of bone loss is smaller, and rehabilitation starts earlier in the latter.

Another possible factor in gaining sufficient restoration is weight loading. Houde et al. reported in a study of the forearm after surgery on the wrists or hands that bone mass levels had not returned to preimmobilization levels after 28 weeks of activity, regardless of whether remobilization was begun either at 12 weeks or 32 weeks [9]. The difference in findings of restoration between our study and their study may arise from differences in bone type: weightbearing bones and nonweightbearing bones. Muscle strength and functional scores were factors positively correlated with BMD values in the injured extremity [10]. Weight loading probably influenced the restoration of BMD, although the magnitude of change after surgery was small in our study.

In conclusion, brief periods of nonweightbearing after hip surgery lead to significant bone loss but do not prevent restoration of BMD. BMD decreases promptly after surgery, but restoration occurs more slowly than the bone loss, requiring 1–1.5 years to recover to the baseline BMD value. The bone loss rate was higher in perimenopausal women than in premenopausal women. Accelerated bone loss was seen in patients with prolonged bed rest and in patients receiving corticosteroid. Both trabecular and cortical components were influenced by the nonweightbearing and restoration of

weightbearing. The decrease in the cortical region occurred after those in the trabecular and endosteal regions.

*Acknowledgment.* This study was partly supported by a grant from the Japanese Osteoporosis Foundation.

## References

1. Finsen V, Benum P (1989) Osteopenia after ankle fractures. *Clin Orthop* 245:261–268
2. Andersonson SM, Nilsson BE (1979) Changes in bone mineral content following tibia shaft fractures. *Clin Orthop* 144:226–229
3. Muller A, Ruegsegger E, Ruegsegger P (1989) Peripheral QCT: a low-risk procedure to identify women predisposed to osteoporosis. *Phys Med Biol* 34:741–749
4. Nilsson BE, Westlin NE (1975) Long-term observations on the loss of bone mineral following Colles' fracture. *Acta Orthop Scand* 46:61–66
5. Cann CE, Genant HK, Young DR (1980) Comparison of vertebral and peripheral mineral losses in disuse osteoporosis in monkeys. *Radiology* 134:525–529
6. Hansson TH, Roos BO, Nachemson A (1975) Development of osteopenia in the fourth lumbar vertebra during prolonged bed rest after operation for scoliosis. *Acta Orthop Scand* 46:621–630
7. Mack PB, LaChance PA, Vose VP, Vogt FB (1967) Bone demineralisation of foot and hand on Gemini-Titan IV, V and VII astronauts during orbital flight. *Am J Roentgenol* 100:503–511
8. Jaworski ZFG, Uhthoff HK (1986) Reversibility of nontraumatic disuse osteoporosis during its active phase. *Bone (NY)* 7:431–439
9. Houde JP, Schulz LA, Morgan WJ, Breen T, Warhold L, Crane GK, Baran DT (1995) Bone mineral density changes in the forearm after immobilization. *Clin Orthop* 317:199–205
10. Kannus P, Jarvinen M, Sievanen H, Oja P, Vuori I (1994) Osteoporosis in men with a history of tibial fracture. *J Bone Miner Res* 9:423–429