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Longitudinal study of the bone mineral content and of soft tissue composition after spinal cord section

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We present the results of a 1 year longitudinal study of bone mineral measurements and soft tissue composition in supra- and infra-lesional areas of 31 patients with a spinal cord injury (level D2-L3). Like others, we observed a rapid decrease of BMC in the paralysed areas, of ~4%/month during the first year in areas rich in trabecular bone and of ~2%/month in areas containing mainly compact bone. Lean soft tissue mass (muscle mass) decreases dramatically during the first months post injury in the legs, while fat content tends to increase. Though lean mass is better maintained in patients who develop spasticity, the evolution of BMC does not differ significantly between the groups of flaccid and spastic patients. In patients with partial or complete neurological recovery, a deficit in BMC of ~10% with regards to the initial value is still observed at 1 year in the lower limbs. The lean mass of the upper limbs increases early after the cord injury, because of intensive rehabilitation. No significant change in BMC was observed in the supra-lesional areas. These data confirm the rapid loss of bone in the paralysed areas of paraplegic patients, which occurs independently of the presence of spontaneous muscle activity or of passive verticalisation. In patients with recovery, BMC does not return to pre-injury values within 1 year. Thus, there would be an interest in preventing bone loss early in the course of the disease.

Keywords: spinal cord injury; disuse osteoporosis; tissue composition; bone mineral

Introduction

An important degree of osteoporosis can be observed radiologically in the paralysed areas of patients with traumatic paraplegia, particularly in areas rich in trabecular bone such as the superior tibial metaphysis.[1,2] Loss of bone in these patients is accompanied by an important increase in urinary calcium and hydroxyproline, two markers of an increased bone resorption,[3] which peak around 6 weeks after the spinal cord section. Few longitudinal studies have been conducted in relatively small numbers of patients to quantify bone loss and to evaluate its kinetics.[4,5,6] The main longitudinal study is that of Biering-Sorensen who followed bone mineral density (BMD) of the distal forearm, lumbar spine, femur and proximal tibia in eight patients with a spinal cord injury using DPA.[3] Their group included tetra (two)- and paraplegic (six) patients. At inclusion, the injury dated from less than 1 month to more than 5 months. The more rapid loss was observed at the upper tibia, which is rich in trabecular bone, with a total loss of ~50% attained at 18 months, followed by a new steady state. In the femoral shaft, the loss was more linear; it amounted to ~20% at 18 months, and continued afterwards. No loss was observed in the lumbar spine, nor in the arms. More recently, Garland *et al*[5] published a transverse/longitudinal study in which 25 male acute-spinal cord injured patients were included $3\frac{1}{2}$ months after their injury and compared with 10 normal subjects and 20 chronic spinal cord injured patients studied more than 5 years after their accident. Twelve of the 25 acute patients were re-measured $1\frac{1}{2}$ year after their injury. Tetra- and paraplegic patients were included in their groups. The bone mineral content was measured by DPA in the head, arms, trunk, pelvis and legs. The authors also report on the bone mineral density of the distal femur. They show bone loss in all the areas studied, except the trunk and the head, in acute-spinal cord injured patients. Bone mineral was stable in the arms thereafter, but it took $1\frac{1}{2}$ year to attain a new steady state in the pelvis and legs. In the arms, there was a significant difference in bone mineral content (BMC) between tetra- and paraplegic patients, but stability is observed after the acute period in both subgroups. On the other hand, there are few data on body composition after spinal injury. The main

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conclusion of these studies[6–8] is a general increase in fat content.

In this study, we took advantage of the new DXA technology to measure sequentially in spinal cord injury patients both total body and regional bone mineral (TBBM) and the lean and fat contents of body regions located in the paralysed and unharmed areas, during the first year following the injury. Only paraplegic patients with a complete lesion were included in the global analysis, although some patients undergoing partial or total neurological recovery were compared to this group. As it was shown that the loss occurs early, the first TBBM measurement was realised within 8 weeks post-injury at the latest (1 to 8 weeks).

Methods

Patients

A consecutive series of 31 patients (seven female and 24 male) presenting at the Centre de Traumatologie et de Revalidation within 8 weeks of a traumatic spinal cord injury were recruited for this study. Each patient gave informed consent. The mean age of the group was 32.5 years (range: 17.5–65.5) and the lesion level was T2 to L3 (no tetraplegic patients included). The first TBBM measurement was performed around 5 weeks ($n = 31$). Then, measurements were performed around 10 weeks ($n = 26$), 20 weeks ($n = 16$), 30 weeks ($n = 11$), 40 weeks ($n = 4$) and 50 weeks ($n = 2$). Soft tissue composition could not be calculated in one patient (no soft tissue bar). Three leg measurements could not be taken into account at 10 weeks and two at 20 weeks because of an artefact. A + or ++ degree of spasticity (estimated by the clinician) developed in 15 patients. Six patients recovered walking with an orthosis at the end of this study, and two others assumed a standing position. None of the patients developed clinical or radiological periarticular ossifications.

Technique

Total body and regional bone mineral were measured by DXA using a Hologic W2000 apparatus. A soft tissue phantom was measured together with the patient in order to calculate the soft tissue composition. The software used to calculate bone mineral contents and tissue composition are those of the constructor. Long term imprecision of TBBM and of fat content with this device is given as 1%. Only areas in which the result could not be altered by the presence of synthesis material were analysed. This excludes most often the trunk, sometimes a broken limb. The results of bone measurements are given in BMC (g), or are expressed as a percentage of their initial values in order to make pooling of the results possible. The results of the fat content and of lean tissue mass are given in g or in % of the initial value. Linear and non-linear regression were used to calculate rates of variation of BMC or soft tissues.

Results

Bone mineral content

(1) Patients with a complete lesion showing no trend towards recovery ($n = 25$) The trunk and lumbar spine were excluded from the analysis because they were too often the place of an osteosynthesis.

The area where bone loss was the fastest was the pelvis (Figure 1A), with a loss of 40 to 45% of the initial value after one year. In the lower limbs taken as a whole, the loss was linear and attained only 25% at the end of the first year (Figure 1B). As observed by Biering-Sorensen[3] in the upper tibia, which is also rich in trabecular bone, the loss in the pelvic area was non-linear and tended to level-off already at 1 year. In the lower limbs however, the loss is steadily continuing at the end of the first year. The bone mineral content in the arms does not vary significantly during the same period. In the skull, the BMC first decreases by ~10%, and then increases again from the 4th to the 12th month.

(2) Patients with a partial recovery ($n = 6$) These patients, who have been fully paralysed for some weeks after their trauma, lose bone during this period in the paralysed areas, and do not recover bone mass in these areas during the observation period, notwithstanding the motor recovery and assuming a standing position. At the end of 1 year, bone loss is nevertheless less than occurs in the paraplegic patients who do not recover, being as a mean of 30% in the pelvis and of 10% in the lower limbs (Figure 2).

(3) Comparison of flaccid ($n = 12$) and spastic ($n = 11$) paraplegia BMC of the paralysed areas varies in a very similar way in the lower limbs of spastic and flaccid patients (Figure 3).

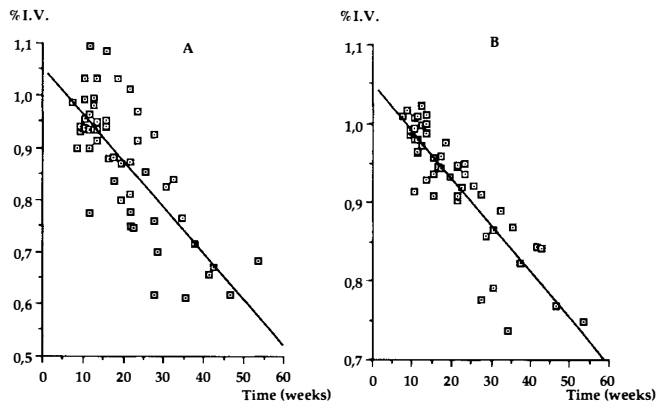

Figure 1 Evolution in BMC in the pelvis (A) and legs (B) of paraplegic patients who do not recover walking. The evolution of BMC with time (weeks) is described by the relations: pelvis: BMC = 1.03 − 0.0089 × ($r = 0.765$); legs: BMC = 1.03 − 0.0059 × ($r = 0.879$)

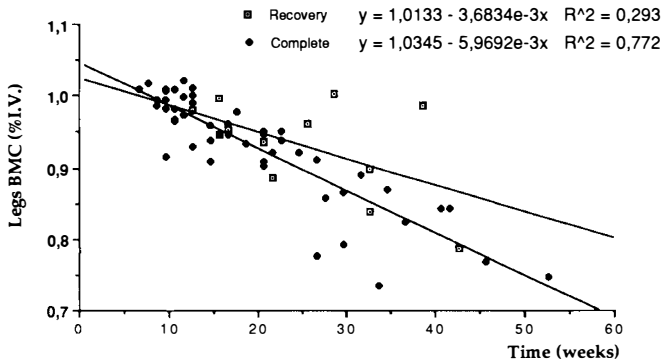

Figure 2 Evolution of BMC in patients with no recovery as compared with those who recover walking

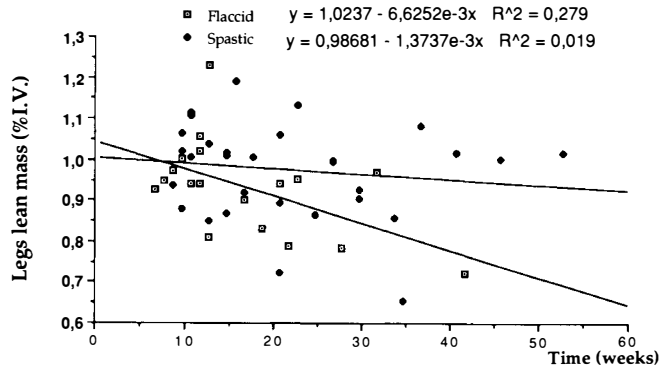

Figure 4 Leg lean mass in spastic and flaccid paraplegic patients

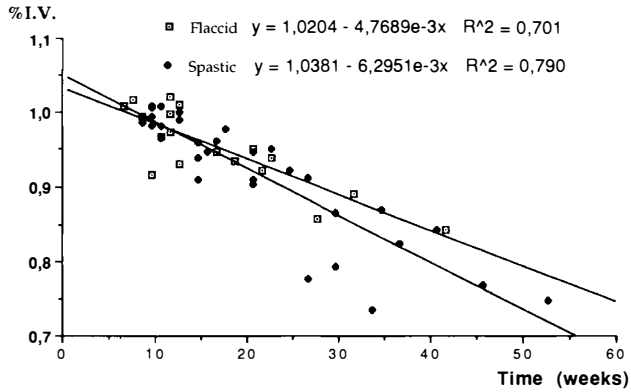

Figure 3 Evolution of BMC in spastic versus flaccid paraplegic patients

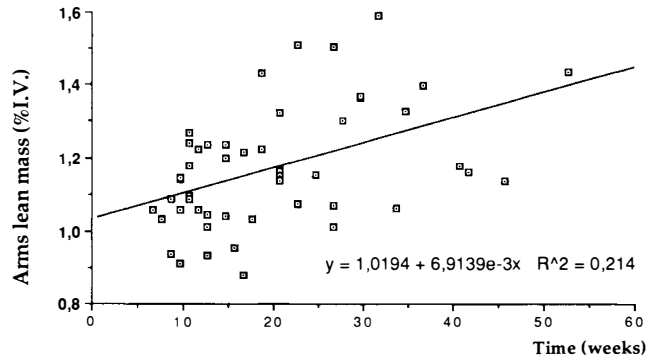

Figure 5 Evolution of arms lean mass during the first year of paraplegia

Regional lean mass

In the legs, the lean mass was decreased by ~15% after 1 year. This decrease occurred rapidly after the spinal injury (15 weeks). The decrease of lean mass was different between the spastic and flaccid groups; as a mean, spastic patients did not lose lean mass at all in their paralysed areas (Figure 4).

In the arms, as a result of active rehabilitation, lean mass increased by 30% after 6 months. At 1 year, it stabilised, being about 15% higher than its initial value (Figure 5).

Regional fat mass

A large dispersion of the individual evolutions prevents any general overview of the evolution of the fat content. There is clearly a trend for the fat content to increase in the lower limbs and to decrease in the upper limbs of some of the patients, but this variation can be obscured by the patients' weight, which affects more generally the fat content.

Discussion

Loss of bone in the paralysed areas is a well known complication of spinal cord injury.[1–5] It is not harmless, since it will make bones fragile and increase the fracture risk in this population. Fractures are indeed a frequent event in paraplegic patients undergoing modern rehabilitation programs,[9] and their treatment may present unusual difficulties.[10] The aim of our longitudinal study was to describe the natural history of bone loss in a population of paraplegic patients. Our results are confirmatory that an appreciable amount of bone is lost rapidly in the paralysed areas after the spinal cord injury.

Evolution of the lean mass is what is to be expected: a dramatic decrease in the paralysed areas, reflecting muscle waste, in flaccid patients; a relative conservation in patients with spasticity; and a variable increase in the upper extremities, depending on the compliance with the rehabilitation program.

The loss of bone in the paralysed areas occurs more rapidly in areas rich in trabecular bone, such as the pelvis, but it continues more steadily in cortical bone, so that the final result could in the end be approximately the same (~50% loss in the long term).[4] Our study adds some information concerning subgroups of paraplegic patients: (1) though the muscular mass is more conserved in patients with spasticity than in those who are flaccid, the loss of bone in the paralysed areas

does not significantly differ between these groups. This shows that simple conservation of muscle mass is not sufficient to maintain skeletal integrity, in the absence of normal motion; (2) patients who recover a near normal mobility still lose bone at 1 year. Thus, in patients who are likely to recover, a programme of prevention of bone loss should be instituted early in the course of the disease.

The evolution of BMC in the non-paralysed areas remain controversial: some authors[4] did not find bone loss in the upper extremities of their patients while Garland[5] observed an initial decrease of BMC in the supra-lesional areas, followed by a partial recovery. The evolution of the skull BMC of our patients would be similar to this, but with complete recovery. In the arms, patients did not lose any bone mineral. Differences between studies with regard to the evolution of bone mineral in the non-paralysed areas could result from the intensity and precocity of the rehabilitation program, and from differences in the patient's population, with patients more severely injured and in worse general condition losing bone, the others not. It is noteworthy that our patients did not have a significant bone gain in the upper extremities, in front of a significant increase of muscular mass and activity. Probably an effect of exercising on bone mass will take longer than the period of intensive rehabilitation. After this period, patients are discharged and lose at least in part the muscle they had gained.

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