Original Article

Skeletal adaptations in hemiplegic patients

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Background

Osteoporosis is commonly defined as generalized skeletal disorder, characterized by changes of microarchitecture. The critical role in pathogenesis is attributed to hormonal processes.

Our findings of tibial bone changes in hemiplegic resp. hemiparetic patients are not compatible with this view. The adaptations are neither generalized nor are they restricted to the microarchitecture, i.e., trabecular bone. Rather, changes are found in trabecular bone in the epiphysis as well as in cortical bone in the diaphysis. Nor can they adequately be explained by hormones, because they represent an individually different distribution of local changes. The usually cited characteristics of osteoporosis – generalized disorder of predominantly microarchitecture, determined by hormones – are thus called into question by our findings.

The bone adaptations found in the limbs of chronically hemiplegic resp. hemiparetic patients can be explained by the feedback principles of the muscle-bone-unit, in which bone strength is controlled by the muscle forces that act upon the bone. An overwhelming amount of data collected from athletes, hemiparetic and paraparetic individuals, as well as animal experiments, demonstrate that the reduction of muscle forces acting on bone lead to reduction of bone strength. Based on our ongoing study of post-stroke patients, it is our hypothesis that a comparison of the paretic limb with the contralateral side gives an insight into the structural bone adaptations which follow reduced muscle forces.

We make the assumption that the muscle forces acting

habitually on the paretic limb are considerably less than on the opposite side. This reduction of forces reduces the strain on bones. This leads to loss of bone mass and bone strength. We are testing this assumption in two parallel trials.

Sports with clearly different forces acting on different limbs can, similarly to hemiparesis and paraparesis, be seen as "experiments of nature" explaining the relationship between habitual forces and bones. These data support Wolff's law and the Utah paradigm[1-8]. Post-stroke patients have a two- to four-fold risk of hip fractures[9-13], presumably resulting from a combination of increased fall frequency and reduced bone strength (66% of fractures occur on the stroke-affected side[12]). However, the published bone data of post stroke patients are inconsistent, and often measured by DEXA, therefore lacking any information about architecture. Data collected by DEXA-technique only give information about mass changes and not about changes of structure.

Patients and methods

We are conducting two trials with post-stroke patients admitted to a geriatric rehabilitation clinic.

In the first trial, we are collecting data of muscle function during locomotor restitution in the post-acute phase, 3 to 8 weeks after a stroke. For this trial we recruit patients with a clinically successful restitution of motor function. We apply classic locomotor tests (chair rising, maximal gait speed) and the newly developed technique of mechanography (LEONARDO, Hans Schiessl, Novotec Pforzheim) to patients with a successful locomotor restitution in order to analyse the development of muscle function. Details of the mechanography are published elsewhere[14].

Through mechanography we register side-specific ground reaction forces, which are used to calculate velocity of the centre of gravity (COG) and power over the duration of a physiological movement. This specialized technique, in contrast to other methods of measuring movement, enables us to record data according to the conventions of physics, that is, in physical units (Newton, Watt, Velocity of the Center of Gravity). This allows an assessment of the side-to-side dif-

	gait_max1 [s/10m]	gait_max2 [s/10m]	change [%]	Chair1 [s]	Chair2 [s]	change [%]	power1 [kW]	power2 [kW]	power increase [%]	force1 [kN]	force2 [kN]	force increase [%]
Pat1	21.4	11.8	44.9	27.7	13.4	51.6	0.33	0.47	**42.4**	0.91	1.10	**20.9**
Pat2	10.4	8.7	16.3	19.3	14.9	22.8	1.07	1.39	**29.9**	1.48	1.37	**-7.4**
Pat3	13.0	7.2	44.6	22.3	13.8	38.1	0.78	1.08	**38.5**	1.18	1.18	**0.0**
Pat4	15.5	8.4	45.8	9.0	7.2	20.0	1.06	1.56	**47.2**	1.48	1.46	**-1.4**
Pat5	8.3	5.7	31.3	7.8	8.3	-6.4	0.34	0.76	**123.5**	1.25	1.35	**8.0**
Pat6	20.4	13.6	33.3	10.7	10.5	1.9	1.74	2.58	**48.3**	1.54	1.93	**25.3**
Pat7	7.9	8.5	-7.6	12.1	6.8	43.8	1.24	1.34	**8.1**	1.14	1.35	**18.4**
Pat8	6.0	6.5	-8.3	11.0	8.0	27.3	0.52	0.57	**9.6**	0.94	0.96	**2.1**
Pat9	8.6	6.2	27.9	10.5	8.4	20.0	0.26	0.79	**203.8**	0.81	1.24	**53.1**
Pat10	7.9	8.0	-1.3	12.3	10.3	16.3	0.98	1.78	**81.6**	1.65	1.58	**-4.2**
Pat11	12.3	7.3	40.7	12.4	9.0	27.4	1.90	1.93	**1.6**	1.37	1.43	**4.4**
Pat12	8.3	6.5	21.7	9.9	7.6	23.2	1.15	1.36	**18.3**	1.13	1.15	**1.8**
Pat13	9.8	6.9	29.6	22.9	13.2	42.4	1.73	2.23	**28.9**	1.38	1.64	**18.8**
Pat14	11.3	7.2	36.3	14.4	10.0	30.6	0.82	0.84	**2.4**	0.99	0.97	**-2.0**
Pat15	16.2	13.6	16.0	10.1	9.0	11.0	1.04	1.50	**44.2**	1.49	1.57	**5.4**
Pat16	11.4	11.6	-1.8	10.4	8.0	23.1	0.84	1.23	**46.4**	1.33	1.17	**-12.0**
Pat17	5.4	5.2	3.7	10.2	8.3	18.6	1.49	1.77	**18.8**	1.47	1.37	**-6.8**
Mean	11.42	8.41	21.95	13.71	9.81	24.21	1.02	1.36	**46.68**	1.27	1.34	**7.3**
SD	4.64	2.65	18.98	5.74	2.53	14.71	0.50	0.58	**50.60**	0.25	0.25	**15.9**

Post-stroke patients during rehabilitation (2-6 weeks after onset), measure1= beginning of rehab, measure2= end of rehab 2-4 weeks later. Improvement of clinical measures: Gait_max = maximal gait velocity, time [seconds] for 10 m. Chair= chair rising test, 5x rising from a usual chair without using one's arms, time in seconds. Power and force: results of mechanography, kiloWatt resp. kiloNewton during vertical jumping. Apparently, the power is the parameter measuring the improvement (mean= 46.68 %), whereas force increases by only 7.3 percent on average, in some cases force even decreases contrary to the clinical improvement.

Table 1. Locomotor measures of 17 consecutive post-stroke patients with successful restitution of locomotion.

ference in force development during the locomotion of hemiparetic patients and the differentiation of concentric and eccentric phases.

Through mechanography we can thus characterize the locomotor restitution in the international nomenclature of physics, i.e., in terms of force and power.

In our second trial, bone adaptations are investigated by pQCT in patients who have had a clinically significant chronic hemiparesis for at least 6 months, comparing the affected side with the contralateral one. The pQCT measurements (pQCT XCT 2000, Stratec Pforzheim) are made on both lower legs (tibia), with cross-sectional examination of muscle and bone at 4%, 14% and 38% of tibia length, calculated from the distal ankle joint[15,16].

Results

We are here reporting preliminary data of an ongoing study. Our findings about force and power during locomotor restitution are very consistent: the level of force increases only a small amount, about 10%, whereas power increases by 40 to 100%. Table 1 presents data of a series of 17 consecutively admitted patients with successful rehabilitation. The results of the chair rising test and maximal gait velocity represent clinical improvement, and have to be seen as a surrogate for power, for we are measuring work (= force x distance) per time. The mechanography can distinguish force and power during vertical jumping, showing only small improvements of force (mean 7.3%), as opposed to large improvements of power (mean=47%).

We interpret these data as an increase in the ability of the locomotor system to recruit faster and more co-ordinated motor units. To summarize, in relation to the muscle-bone relationship: the force is principally maintained on the low level caused by the stroke, confirming and quantifying the clinical experience of diminished force in hemiparetic limbs.

Summarizing the preliminary results of bone assessment, on the lower legs of chronic hemiparetic patients we regularly find (with rare exceptions) apparent adaptations of mass and architecture of the affected leg compared to the contralateral. Mostly we find a loss of mass and bone strength.

However, these adaptations are inter-individually not at all homogeneous. We find a varied pattern of changes, with two poles of a spectrum. At one end of the spectrum the adaptations are predominantly at the trabecular bone of the epiphysis, at the other end of the spectrum on the cortical

bone of the diaphysis. Between these two extremes, we find various transitional stages with both epiphyseal and diaphyseal adaptations.

The usual finding at the epiphysis is a loss of trabecular bone mass and true (volumetric) density, whereas at the diaphysis, a loss of mass and cortical area by thinning of the diaphyseal wall thickness are common occurrences. The inter-individually in homogeneous findings make a statistical evaluation difficult. We need a far greater number of patients to arrive at a conclusion or to make a comprehensive analysis. Two examples typify the different patterns of bone adaptations which we observed (Figure 1 and 2). Table 2 presents data of the first 26 participants, showing the loss of bone mass of the hemiparetic legs compared with the non-affected side.

Discussion

We are able to demonstrate through follow-up mechanography in post-acute stroke patients that locomotor restitution is mainly a restitution of power, rather than one of force. The consequences to be drawn for clinical practice and research are as follows: a clear distinction must be made between these two terms. Locomotor improvement must be measured by power.

Perhaps we can explain this finding by better recruitment of motor units referring to time and local distribution. Movement cannot be measured by force alone. Movement is always the action of a force over a distance in a certain time, i.e., force x distance/time = force x velocity = work/time = power. Thereby, we need the velocity of the body to calculate power. The exclusive use of the term "force" in describing or even quantifying movement thus effectively excludes "time" and "distance" from the concept "movement". The false notion created thereby is a violation against the laws of physics, which results in negative consequences for research and clinical practice. Through mechanography we are able to register force, velocity and power of physiological, unrestricted movements separately, thus including kinetics and kinematics.

However, for diagnosing the relations between bone and muscle we have to refer to force, because bone strength is controlled by muscle peak forces. Thus, it is muscle force which must be given major consideration in the analysis of the muscle-bone unit.

In relation to bone we are interested in the question: when does bone break? In posing this question, we are looking for the ultimate force required for breaking a structure = bone strength. For answering this question we need only the knowledge of three bone factors: the material properties of bone (N/mm^2, = ultimate strength), the bone architecture, properties of surface – and amount and direction of the acting force. The relevant literature reports rather uneven results with respect to "hemiosteoporosis" in hemiparetic patients[9-12]. These reported inconsistencies can be explained by the questionable results usually yielded by the DEXA

ID_number	difference in bone mass at 4% of tibia length (epiphysis)	difference in bone mass at 38% of tibia length (diaphysis)
1	1.2	-11.9
2	-16.8	0.3
3	1.1	-3.9
4	3.1	1.4
5	-4.8	-6.4
6	-6.1	-10.6
7	0	-3.8
8	-10.1	-11.5
9	-10.4	-4.9
10	-28.6	-4.9
11	-14.1	-8.2
12	-6.7	-6.5
13	-11.6	-6.2
14	8.8	-10.5
15	2	2.2
16	-7.3	-5.4
17	-27.1	-16.4
18	0	-8.8
19	3.6	0.7
20	-2.1	-4.7
21	-4.7	-14.2
22	-5.2	-9.3
23	-22.6	-36.2
24	-13.6	-0.7
25	-31.9	-17.2
26	3.4	0.4

Difference in bone mass in hemiparetic legs compared with the contralateral limb at epiphysis and diaphysis. Percent values are related to g/cm, i.e., bone mineral content of one slice of bone of 1 cm length at 4% (epiphysis) and 38% (diaphysis) of tibia length, measured from distally. Most hemiparetic legs show a considerable loss of bone mass up to –36.2% compared to the non-affected leg, but the bone loss is differently distributed in the epiphysis or diaphysis. 7 out of 26 patients show no significant loss (ID 3, 4, 7, 15, 19, 20, 26).

Table 2. Side-to-side comparison of bone mass in the legs of hemiparetic patients.

method. The results of this method are highly correlated to fractures, but unsuited for giving information on bone architecture, which is the decisive determinant for ultimate breaking force.

Our findings show enormous complexities in change that occur in the hemiparetic limb as compared to the not-directly-affected contralateral side. Thus, we need data of architecture and the differentiation of trabecular and cortical bone to gain deeper insight in the adaptation of bone during the course of hemiparesis. The number of chronic hemiparetic patients in our study is not yet sufficient to calculate the statistical relations between different bone adaptation patterns and different muscle status.
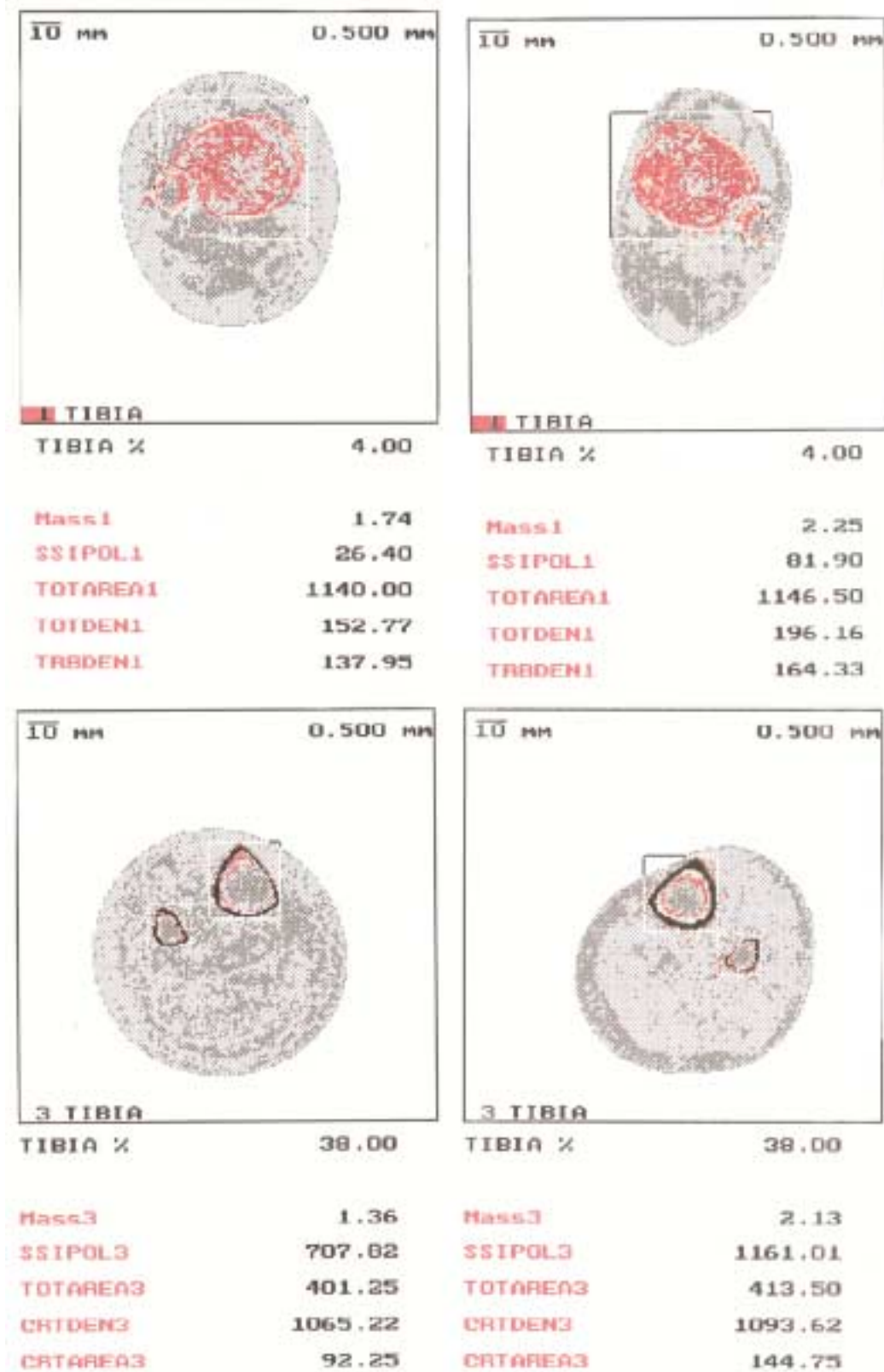
Figure 1. pQCT of lower legs of a patient with chronic hemiparesis of the left leg. Left leg= left figures.
Tibia at 4% (1 TIBIA) and 38% (3 TIBIA) of tibia length (calculated from distal joint). Muscle atrophy at the left side, bone loss left compared to right at epiphysis and diaphysis.
TRBDEN= trabecular density [mg/ccm], CRTDEN= cortical density [mg/ccm], TOTAREA= total area. MASS= mass [g per a slice of 1 cm].
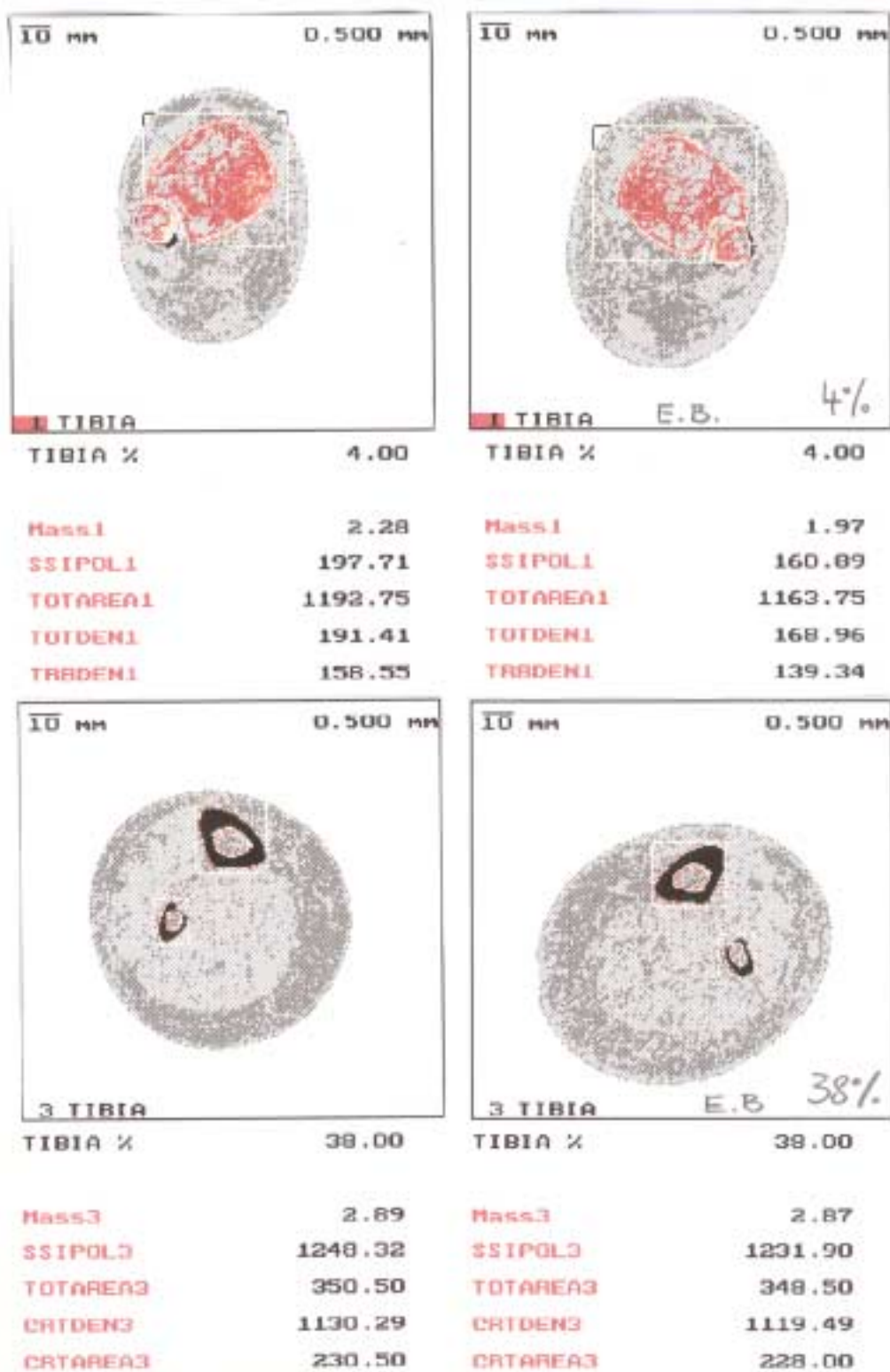
Figure 2. pQCT of lower leg of a patient with chronic hemiparesis of the right leg.
Tibia at 4% (1 TIBIA) and 38% (3 TIBIA) of tibia length. Left leg= left figures.
Muscle atrophy at the right side, bone loss at right epiphysis near ankle (cf. TRBDEN and Mass). No significant bone loss at diaphysis.

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