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Body Composition in Disabilities of Central Nervous System

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1. Introduction

Disability leads to immobilisation associated with profound changes in body composition. The potential risks involved with these changes i.e. loss of lean tissue mass (LM) and bone mineral density (BMD) vs. gain in fat mass (FM) in body composition have implications for the health of the disabled individuals (Jones et al., 1998). Body fat has been identified as a significant predictor of mortality in humans making body composition measurement to quantify nutritional and health status an important issue for human health. (Seidell et al., 1996; Bender et al., 1998; Van Der Ploeg et al., 2003). Moreover, some disorders such as carbohydrate intolerance, insulin resistance, lipid abnormalities, and heart disease occur prematurely and at a higher prevalence in disabled populations may be related to adverse changes in body composition that result from immobilization and skeletal muscle denervation (Spungen et al., 2003).

In traumatic and pathological lesions of the central nervous system (CNS) there are differences according to the evolution or not of the lesion (i.e. progressive multiple sclerosis vs. complete paraplegia), the type of injury (i.e. lesion with a level of injury vs. upper motor neuron pyramidal lesion), life expectancy, the residual mobility and functionality, the ability to walk and stand (i.e. incomplete paraplegia vs. quadriplegia vs. high-low paraplegia) and drug treatment (i.e. frequent corticosteroid therapy in multiple sclerosis vs. long-term therapy with anticoagulants in paraplegia). In addition there are differences in the degree of spasticity which is likely to play a regulatory role in maintaining bone density (Dionyssiotis et al., 2011a). We need to take into account the element of fatigue and muscle weakness in disabilities, especially in diseases like multiple sclerosis, which significantly reduces the mobility of these patients (Krupp et al., 2010).

The relative difference in energy expenditure between individuals with multiple sclerosis (MS) and able-bodied subjects is probably lower than the relative difference in physical activity, because individuals with MS have a higher energy expenditure of physical activity (Olgiati et al., 1988). Reduced physical activity (and probably reduced energy expenditure) in MS need to be accompanied by a reduction in energy intake otherwise body fat will increase (Lambert et al., 2002). Subjects with those motor disorders often face problems of depression and limit mobility (Dionyssiotis, 2011b). Moreover, in children with cerebral palsy (CP) studies suggest that increased stretch reflexes and muscle tone, weakness of

involved musculature, and severe limitation of movement reduce the capacity to perform normal movements creating ambulation barriers limiting physical activity. The dependency on mobility devices, common in all disabilities, and the frequent periods of immobilization after multiple operative procedures contribute to the hypoactivity status of such children. It could be assumed that, under these conditions, body composition may be significantly compromised (Chad et al., 2000).

Studies found that lean mass of the contralateral limb was lower compared to the ipsilateral limb in upper motor neuron injury, as occurs in stroke (Ryan et al., 2000; 2002). Similar findings of reduced muscle mass and increased intramuscular fat have been also published in individuals with incomplete spinal cord injury (SCI) (Gorgey et al., 2007) suggesting that reduced muscle mass is fundamentally related to poor fitness and physical performance capacity after stroke (Hafer-Macko et al., 2008).

On the other side the clinical equivalence of diseases with different physiopathology, location, evolution, etc. could be similar; i.e. a severe form of MS can result in a wheelchair bound patient a clinical figure equivalent to paraplegia or a MS patient may have a more appropriate walking gait pattern vs. a patient with incomplete paraplegia but may also be unable to walk at all, is bedridden and vice versa (Dionyssiotis, 2011b; 2011c; 2011d). In addition to these differences and according to osteoporosis the role of factors which do not change, such as race or gender of patients has not been yet clarified, although there are few studies in women debating that bone mass in women with disabilities is more affected than men (Smeltzer et al., 2005; Coupaud et al., 2009).

Therefore, the purpose of this chapter was to present the bone-mineral density, bone-mineral content, and bone-mineral-free lean and fat tissue mass alterations of ambulatory and non-ambulatory subjects with disabilities of the central nervous system.

2. Body composition measurements

2.1 Anthropometric and various techniques of body composition measurements

In a study which investigated a chronic spinal cord injury (SCI) population with paraplegia (Dionyssiotis, 2008a, Dionyssiotis et al., 2008b) values of body mass index (BMI, kg/m^2) did not present statistical significance in relation to the controls, which is a finding in line with the literature (Maggioni et al., 2003; Mamoun et al., 2004).. Nevertheless, there are studies which demonstrate the usefulness of BMI as an indicator of obesity, in body composition in people with spinal cord injury (Gupta et al., 2006). These studies, however, included both tetraplegics and middle-aged people unlike the Greek one which included relatively young individuals (Dionyssiotis et al., 2008a). Whether the criteria of BMI may assess obesity in people with spinal cord injury the latest studies show the opposite (McDonald et al., 2007).

BMI of a male paraplegic group was slightly greater compared to a tetraplegic one and distribution of BMI by level of injury was similar with 37.5% and 40.5% of the male tetraplegic and male paraplegic groups, respectively, falling into the recommended BMI range. Approximately 50% in each male group were overweight by BMI, and 12.5% and 10.8%, respectively, were classified as obese. Overall, when compared with the general population-observed distribution by BMI, a greater proportion of men with SCI fell into the desirable BMI range and fewer fell into the obese category (Groah et al., 2009).

No differences were found in BMI between paraplegics in the acute phase of injury and controls, which is a finding in accordance with other studies in which, despite the same BMI, the body composition and the distribution of fat and fat free mass were altered in patients with spinal cord injury, with the fat free mass being statistically significantly lower in paraplegic patients in total body composition and in the lower, but not the upper limbs. As far as the fat mass is concerned, it was statistically significantly higher (kilograms and %) in the total body composition in the upper and lower limbs (Maimoun et al., 2006).

These findings show that using the BMI does not contribute substantially in determining the body composition of paraplegics and lowers the percentage of fat in this population, finding that agrees with other studies and shows that the anthropometric measurement with BMI in paraplegics, underestimates fat in body composition when measurements are compared with healthy subjects (Jones et al., 1998).

Body mass index is a very simple measurement of fat; however it does not distinguish the individual components of weight. The applicability of conventional BMI cut off values is into question (Buchholz, 2005; McDonald et al., 2007). BMI is an insensitive marker of obesity in subjects with SCI and measuring fat with BMI in chronic paraplegic patients is not enough to determine subject's percentage of fat in the body (Olle et al., 1993).

To standardize or index physiological variables, such as resting metabolic rate and power fat free mass (FFM) is usually used (Van Der Ploeg et al., 2003). Skeletal muscle represents 50% of the non fat component in the total body (Clarys et al., 1984; Modlesky et al., 2004) and exact quantification of the amount of skeletal muscle is important to assess nutritional status, disease risk, danger of illnesses, physical function, atrophic effects of aging, and muscle-wasting diseases (Forbes, 1987; Mojtahedi et al., 2008).

Because muscle wasting is a common sign of cerebral palsy (CP), even in well nourished children, the validity of using muscle wasting as evidence or measurement of malnutrition in CP is in doubt. Studies found that the triceps, midthigh, and calf skinfold thicknesses of the affected side were greater than those of the no affected side among children with hemiplegic CP (Stevenson et al., 1995). Useful information regarding fat provide triceps, subscapular skinfolds and arm-fat area (Patrick & Gisel, 1990). Other studies support the concept that the validity of skinfold thickness as an assessment of limb fat storage is dependent on the preservation of limb muscles (Ingemann-Hansen T et al., 1977) and suggested good sensitivity and specificity of triceps skinfold thickness for predicting mid-upper arm fat area probably were attributable to good preservation of mid-upper arm muscles among children with CP (Samson-Fang et al., 2000).

In disabled children techniques for measuring skinfolds are well established and standardised (Lohman et al., 1988) and equations are available for calculation of body fat from skin fold thickness (Slaughter et al., 1988) although unvalidated in this population, as are normative values for skinfold thickness (Frisancho, 1981; Kuperminc & Stevenson, 2008). Consequently, use of skinfold thickness as a measurement, especially for the affected limb, should be used with discretion in the assessment of children with CP, who tend to have muscle wasting.

In cerebral palsy neither bioelectrical impedance analysis nor predictive equations for skinfold thickness generated from normal, able-bodied adults accurately determined

percentage body fat (Hildreth et al., 1997). Body mass index (BMI), triceps skinfold thickness, subscapular skinfold thickness, suprailiac skinfold thickness, and circumferences of the biceps, waist, forearm, and knee were all significantly correlated with percentage body fat (Bandini et al., 1991).

BMI in patients with MS was statistically less compared to age comparable controls (Formica et al., 1997). In a recent study both total body fat and mass percent showed consistent significant dependence from BMI, as among normal subjects. Multiple linear regression analysis of bone mineral percent at all studied sites showed consistent dependence from BMI (increased with higher BMI) for both patient and control groups (Sioka et al., 2011).

Changes in body composition in spinal cord injured subjects can be assessed with various techniques including isotope-labelled water (Jones et al., 1998) total body potassium counting (Lussier et al., 1983; Spungen et al., 1992) anthropometric measures (Bulbulian et al., 1987) hydrodensitometry (Lussier et al., 1983; Sedlock, 1990) dual photon absorptiometry (DPA) (Spungen et al., 1992; Changlai, 1996) and dual energy X-ray absorptiometry (DXA) (Jones et al., 1998). However, some of these methods are not particularly suitable for use in the SCI population.

The hydrodensitometric model was regarded as the "gold standard" for body composition assessment. This model partitions the body into two compartments of constant densities [fat mass: 0.9007 g/cm^3 and FFM: 1.100 g/cm^3] and assumes that the relative amounts of the FFM components [water, protein, protein, bone mineral (BM), and non-BM] are fixed (Brozek et al., 1963; Van Der Ploeg et al., 2003). Hydrodensitometry is clearly inappropriate for individuals who deviate from these fixed and/or assumed values (e.g., children, elderly, blacks, obese), and its application is, therefore, somewhat limited (Womersley et al., 1976; Schutte, 1984; Lohman, 1986; Fuller et al., 1996).

Bioelectrical impedance analysis (BIA) has been used to measure cerebral palsy subjects. However, the inclusion of weight in the BIA predictive equation may reduce its accuracy in determining change in lean body mass (Forbes et al., 1992). The inability of BIA to accurately predict percentage body fat in the sample may be related to several factors. In the BIA method where the impedance of a geometrical system (i.e., the human body) is dependent on the length of the conductor (height) and its configuration, it is almost impossible to measure accurately height in subjects with CP because of their muscle contractures. An over- or underestimation of height by 2.5 cm can result in a l.0-L error in the estimation of TBW, producing a small error in the estimation of percentage body fat (< 5%). The second major problem is body asymmetry which renders the assumption of a symmetrical configuration of the human body invalid in this case. (National Institutes of Health Technology Assessment Conference Statement, 1994; Hildreth et al., 1997).

Isotope dilution measures the water compartment of the whole body rather than a single area assumed to mimic the composition of the whole body. Thus, the use of a stable isotope to measure body composition is ideal for people with CP because it is non-invasive, does not require the subject to remain still for the measurement, and is independent of height and body symmetry. However, the prohibitive cost of the isotopes and the need for a mass spectrometry facility and highly trained technicians make this method impractical for routine clinical use (Hildreth et al., 1997).

To determine whether bioelectrical impedance analysis and anthropometry can be used to determine body composition for clinical and research purposes in children with cerebral palsy 8 individuals (two female, mean age=10 years, mean gross motor function classification=4.6 [severe motor impairment]) recruited from an outpatient tertiary care setting underwent measurement of fat mass, fat-free mass, and percentage body fat using BIA, anthropometry (two and four skinfold equations), and dual-energy x-ray absorptiometry. Correlation were excellent for determination of fat-free mass for all methods (i.e., all were above 0.9) and moderate for determination of fat mass and percent body fat (range=0.4 to 0.8). Moreover, skinfolds were better predictors of percent body fat, while bioelectrical impedance was a better predictor for fat mass (Liu et al., 2005). On the contrary another study investigated the pattern of body composition in 136 subjects with spastic quadriplegic cerebral palsy, 2 to 12 years of age, by anthropometric measures, or by anthropometric and total body water (TBW) measures (n = 28), compared with 39 control subjects. Body composition and nutritional status indicators were significantly reduced. Calculation of body fat from two skinfolds correlated best with measures of fat mass from TBW (Stallings et al., 1995; Kuperminc & Stevenson, 2008).

Magnetic resonance imaging (MRI) provides remarkably accurate estimates of skeletal muscle in vivo (Modlesky et al., 2004). MRI and also quantitative computed tomography (QCT) have been validated in studies of humancadavers in the assessment of regional skeletal muscle (Mitsiopoulos et al., 1998). Although, these devices have disadvantages of high radiation exposure and are expensive.

2.2 Dual-energy X-ray absorptiometry (DXA)

Recently, dual-energy X-ray absorptiometry (DXA) has gained acceptance as a reference method for body composition analysis (Mahon et al., 2007; LaForgia et al., 2009). Originally designed to determine bone density, DXA technology has subsequently been adopted for the assessment of whole body composition and offers estimation rapidly, non-invasively and with minimal radiation exposure (Van Der Ploeg et al., 2003; Dionyssiotis et al., 2008a). Moreover, is well tolerated in subjects who would be unable to tolerate other body composition techniques, such as underwater weighing (hydro-densitometry) (Laskey, 1996). DXA software determines the bone mineral and soft tissue composition in different regions of the body being a three-compartment model that quantifies: (i) bone mineral density and content (BMD, BMC), (ii) fat mass (FM); and (iii) lean mass (LM), half of which is closely correlated with muscle mass and also yields regional as well as total body values (Rittweger et al., 2000) for example in the arms, legs, and trunk (figure 1).

DXA analyzes differently the dense pixels in body composition. Soft tissue pixels are analyzed for two materials: fat and fat-free tissue mass. Variations in the fat mass/fat free tissue mass composition of the soft tissue produce differences in the respective attenuation coefficients at both energy levels. The ratio at the two main energy peaks is automatically calculated of the X-ray attenuation providing separation of the soft tissue compartment into fat mass and fat-free tissue mass (lean mass) (Peppler & Mazess, 1981; Pietrobelli et al., 1996). A bone-containing pixel is analyzed for "bone mass" (bone mineral content, BMC) and soft tissue as the two materials. Thus, the fat mass/fat free tissue mass of the soft tissue component of the bone pixels cannot be measured, but only estimated (Ferretti et al., 2001).

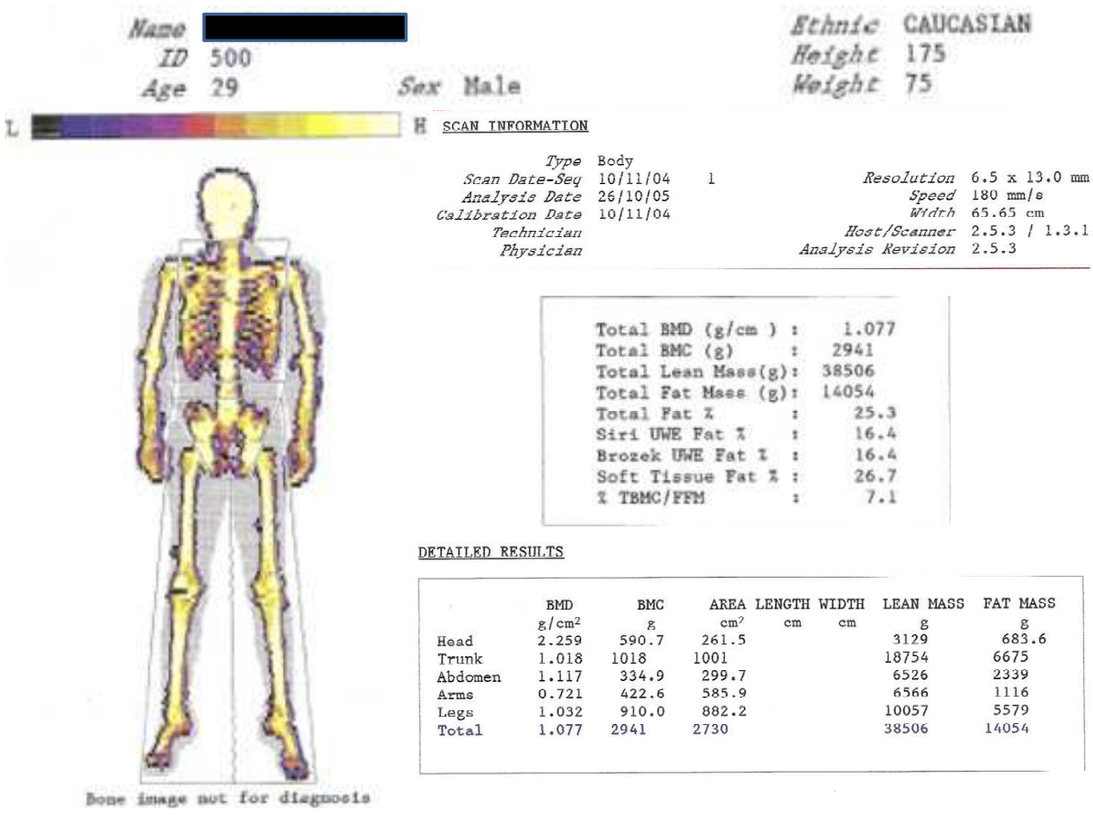
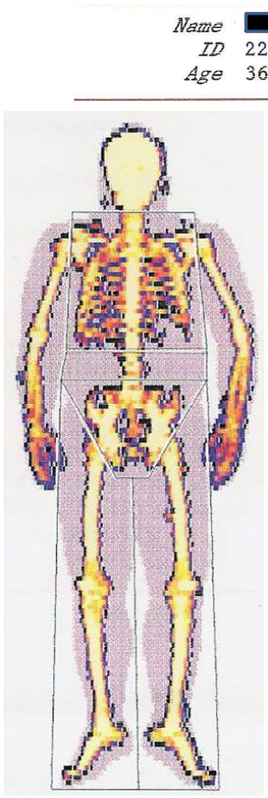


Fig. 1. Whole body and regional distribution of fat mass, lean mass, bone mineral content (BMC) and bone mineral density (BMD) from paraplegic subject thoracic 6 using whole body DXA (Norland X-36, Fort Atkinson, Wisconsin, USA) and values of measured parameters. Modified and translated with permission from Dionyssiotis, 2008a.

The important issue on this is the investigation of distribution of bone mineral, fat and mass throughout the body. These changes induce the risk for diseases such as diabetes, coronary heart disease, dyslipidaimias and osteoporosis (Bauman et al., 1992; Bauman & Spungen, 1994; Kocina, 1997; Garland et al., 1992). There is a need to quantify the alterations in body composition to prevent these diseases and their complications. Studies also reported that bone density measurements at one site cannot usefully predict the bone density elsewhere (Heymsfield et al., 1989) because different skeletal regions, even with similar quantities of trabecular or cortical bone, may respond variably in different physiopathological conditions (Laskey, 1996).

In disabled conditions the accuracy of skeletal muscle measured by DXA may be compromised when muscle atrophy is present. A lower ratio of muscle to adipose-tissue-free mass indicates a lower proportion of muscle in the fat-free soft tissue mass. Cross-sectional area of skeletal muscle in the thighs after SCI is extensively reduced (Castro et al., 1999). If this is the case muscle mass would be overestimated by prediction models that assume that muscle represents all or a certain proportion of the fat-free soft tissue mass, i.e.

in spinal cord injured subjects (Modlesky et al., 2004). DXA technique has been used in assessment of SCI and appears to be tolerated well by this population (Szollar et al., 1997; Uebelhart et al., 1995; Chow et al., 1996).



Name [redacted] Ethnic cauc
ID 2210 Height 180
Age 36 Sex Male Weight 125

Total BMD (g/cm) :	1.256
Total BMC (g) :	3527
Total Lean Mass(g):	79838
Total Fat Mass (g):	40884
Total Fat % :	32.9
Siri UWE Fat % :	29.6
Brozek UWE Fat % :	28.6
Soft Tissue Fat % :	33.9
% TBMC/FFM :	4.2

DETAILED RESULTS

	BMD g/cm²	BMC g	AREA cm²	LENGTH cm	WIDTH cm	LEAN MASS g	FAT MASS g
Head	2.218	586.9	264.6			4142	1035
Trunk	1.254	1181	942.0			40251	24108
Abdomen	1.542	564.3	365.8			17188	12251
Arms	0.781	456.0	583.7			8784	4205
Legs	1.280	1302	1017			26661	11536
Total	1.256	3527	2807			79838	40884

Fig. 2. Whole body and regional distribution of fat mass, lean mass, bone mineral content (BMC) and bone mineral density (BMD) from control male subject using whole body DEXA Norland X-36 and values of measured parameters. Modified and translated with permission from Dionyssiotis, 2008a.

3. Physiopathological context

3.1 Spinal cord injury

Spinal cord injury (SCI) always results in substantial and rapid bone loss predominately in areas below the neurological level of injury. The predominant finding of SCI on bone is a large loss of bone during the first year of injury (Spungen et al., 2003) and an ongoing demineralisation 3 years after trauma in tibia (Biering-Sörensen et al., 1988) with a progressive bone loss over 12 to 16 months prior to stabilizing (Lazo et al., 2001) was demonstrated.

Cancellous bone is more affected than cortical bone after SCI. In a prospective study, six acute tetraplegics were followed up for 12 months, and the trabecular and cortical BMD's of the tibia were found to be decreased by 15 and 7% (Frey-Rindova et al., 2000), while in

paraplegics trabecular metaphysical-epiphyseal areas of the distal femur and the proximal tibia are the most affected sites (Jiang et al., 2006). A cross-sectional study (Dauty et al., 2000) in SCI subjects demonstrated a significant demineralization at the distal femur (-52%) and the proximal tibia (-70%), respectively.

There is no demineralization of the upper limbs in paraplegics. Studies reported a minor increase of BMD while at the lumbar spine trabecular bone demineralization remains relatively low compared to long bones cortical bone demineralization of (Dauty et al., 2000). Normal (Chantraine et al., 1986; Biering-Sorensen et al., 1988; Kunkel et al., 1993) or even higher than normal values were found (Ogilvie et al., 1993), a phenomenon known as "dissociated hip and spine demineralization" (Leslie, 1993) One reason for preservation of bone mass in the vertebral column is because of its continued weight-bearing function in paraplegics but also lumbar spine arthrosis, bone callus, vertebral fracture, aortic calcification, osteosynthesis material, etc. Degenerative changes in the spine may be the most possible reason to give falsely higher values of BMD (Dauty et al., 2000).

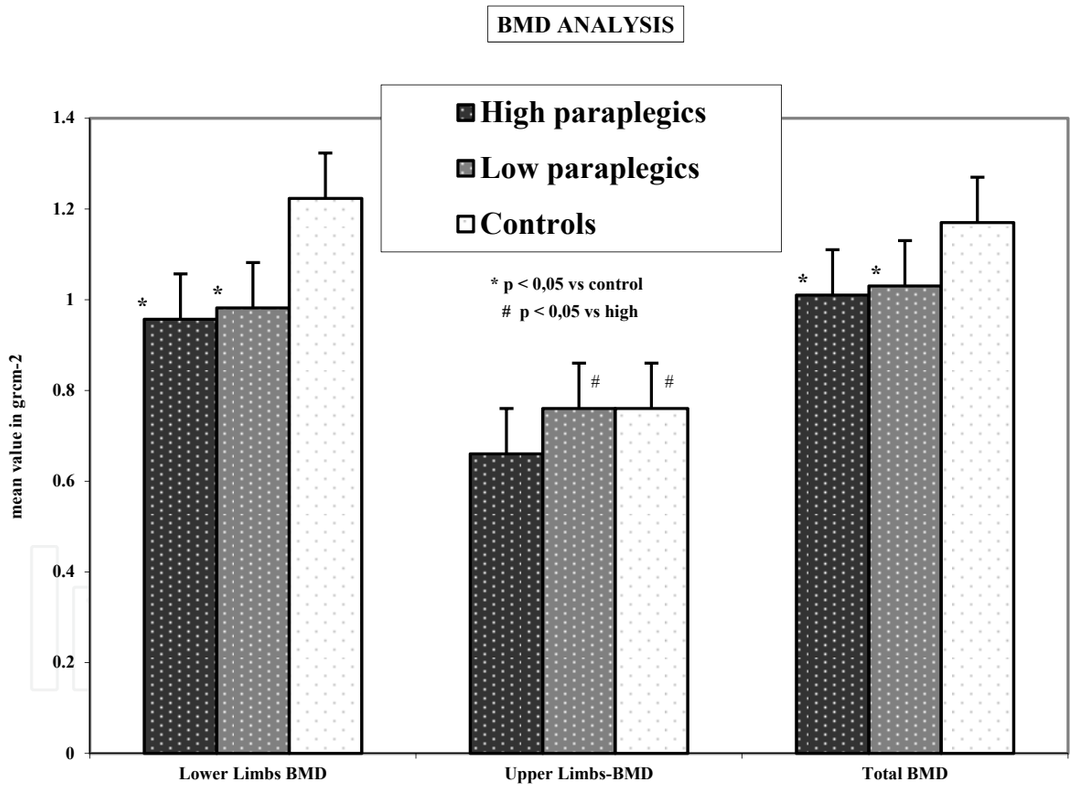


Fig. 3. The picture depicts the analysis of bone mineral density (BMD) in high and low level paraplegics and controls. A statistically significant reduction in total BMD ($p<0.001$) and lower limbs BMD in body composition compared to able-bodied males was observed. On the contrary, upper limbs BMD was higher in low paraplegics and controls, an unexpected finding explained in the paper of Dionyssiotis et al., 2008b. Diagram modified and translated from Dionyssiotis, 2008a.

The neurological level of the lesion i.e. the extent of impairment of motor and sensory function is important, because tetraplegics are more likely to lose more bone mass throughout the skeleton than paraplegics (Tsuzuku et al., 1999). In paraplegics legs' BMC was reduced vs. controls, independently of the neurological level of injury and negatively correlated with the duration of paralysis in total paraplegic group, but after investigation according to the neurological level of injury this correlation was due to the strong correlation of high paraplegics' legs BMC with the duration of paralysis, meaning that the neurological level of injury determines the extent of bone loss (Dionyssiotis et al., 2009). The similar severity of demineralization in the sublesional area was shown between paraplegics and tetraplegics, and the extent of the bone loss may be variable (Demirel et al., 1998; Tsuzuku et al., 1999; Dauty et al., 2000).

The duration of paralysis has an inverse relationship with leg percentage-matched BMD and trunk percentage-matched BMD (Clasey et al., 2004). In addition in complete paraplegics, with high (thoracic 4-7) and low (thoracic 8-12) neurological level of injury, upper limbs FM and lower limbs BMD were correlated with the duration of paralysis in total paraplegic group but after investigation according the neurological level of injury this correlation was due to the strong correlation of high paraplegics' lower limbs BMD with the duration of paralysis. The explanation of this strong correlation could possibly lie on higher incidence of standing in the group of low paraplegics and direct effect of loading lower limbs while standing and walking with orthotic equipment. Moreover, the association of the duration of paralysis with parameters below and above the neurological level of injury (upper limbs FM) raises the question of the existence of a hormonal mechanism as an influential regulator in paraplegics' body composition (Dionyssiotis, 2008a; Dionyssiotis et al., 2008b; 2009).

Actually, little is known regarding the nature and time frame of the influence of complete SCI on human skeletal muscle because published data are coming from cross-sectional studies, where different groups with few subjects have been examined at different times, usually in the chronic phase of paralysis. Disuse was thought to be the mechanism responsible for the skeletal muscle atrophy in paraplegics, but muscle fibres following SCI begin to change their functional properties early post injury. Muscle fiber cross-sectional area (CSA) has been suggested to decline from 1 to 17 months after injury and thereafter to reach its nadir. Conversion to type II fibers has been suggested to occur between 4 months and 2 years after injury, resulting in even slow-twitch muscle becoming predominantly fast twitch thereafter (Castro et al., 1999). Metabolic enzymes levels in skeletal muscle might be expected to be reduced after SCI because of inactivation. In support of this contention, succinic dehydrogenase (SDH) activity, a marker of aerobic-oxidative capacity, has been reported to be 47–68% below control values in fibers of tibialis anterior muscle years after injury in support of this contention (Scelsi, 2001).

The muscle atrophy in SCI is of central type and depends on the disuse and loss of upper connections of the lower motor neuron, sometimes associated to the loss of anterior horn cells and transinaptic degeneration. The last alteration may be responsible for the denervation changes seen in early stages post SCI. In the later stages (10-17 months post SCI) diffuse muscle atrophy with reduction of the muscle fascicle dimension is associated to fat infiltration and endomysial fibrosis. In all stages post SCI, almost all patients showed myopathic changes, as internal nuclei, fibre degeneration and cytoplasmic vacuolation due to lipid accumulation (Scelsi, 2001)

It is evident that other co-factors as spasticity and microvascular damage, contribute to the induction of the marked morphological and enzyme histochemical changes seen in the paralyzed skeletal muscle (Scelsi, 2001). Small fibers, predominantly fast-twitch muscle, and low mitochondrial content have been reported years after injury in cross-sectional studies. These data have been interpreted to suggest that human skeletal muscle shows plasticity (Castro et al., 1999).

On the contrary, force loss during repetitive contractions evoked by surface electrical stimulation (ES) of skeletal muscle in humans does not appear to be altered within a few months of injury (Shields, 1995) but it is greater a year or more after SCI (Hillegass & Dudley, unpublished observations). The greater fatigue, when evident, was partially attributed to lower metabolic enzyme levels (Scelsi, 2001).

Muscular loading of the bones has been thought to play a role in the maintenance of bone density (de Bruin et al., 1999; Dionyssiotis et al., 2011d). However, the ability to stand or ambulate itself does not improve BMD or prevent osteoporosis after SCI.

Controversial results have also been reported regarding the effect of spasticity on BMD in SCI paraplegics. A cross-sectional study of 41 SCI paraplegics reported less reduction of BMD in the spastic paraplegics SCI patients compared to the flaccid paraplegic SCI patients (Demirel et al., 1998). Others reported that spasticity may be protective against bone loss in SCI patients, however, without any preserving effect in the tibia (Dionyssiotis et al., 2011a; Eser et al., 2005). A possible explanation for that could lie in the fact paraplegics to be above thoracic (T)12 level with various degrees of spasticity according to the Ashworth scale. In addition, muscle spasms affecting the lower leg would mainly be extension spasms resulting in plantar flexion thus creating little resistance to the contracting muscles. Furthermore, the measuring sites of the tibia did not include any muscle insertions of either the knee or the ankle extensor muscles (Dionyssiotis et al., 2011a, 2011d). Other investigators also have not been able to establish a correlation between BMD and muscle spasticity (Lofvenmark et al., 2009).

The hormone leptin is secreted by fat cells and helps regulate body weight and energy consumption (Fruhbeck et al., 1998). The percentage of fat in people is positively correlated with the amount of leptin in the circulation (Maffei et al., 1995). In SCI, when compared with healthy subjects, higher levels of leptin have been found, possibly due to greater fat tissue storage (Bauman et al., 1996). Leptin activates the sympathetic nervous system (SNS) through a central administration. The disruption of the sympathetic nervous system i.e. in tetraplegia and high level paraplegia may modify the secretion and activity of the leptin, because the sympathetic preganglionic neurons become atrophic in these subgroups (Elias et al., 1998; Correia et al., 2001) leading to disturbed irritation from leptin below the neurological level of injury. In addition, extensive obesity is known to reduce lipolytic sensitivity (Haque et al., 1999; Horowitz et al., 1999, 2000).

In high level spinal cord injuries there is a disorder of the autonomic nervous system and combined to the fact that the hormone leptin activates the sympathetic nervous system through central control it could be suggested that "the closure of paths" of the central nervous system disrupts the effect of leptin and possibly increases the risk of obesity in SCI subjects with high-level injury (Krassioukov et al., 1999; Jeon et al., 2003). However, after separation of SCI subjects into those with an injury above or below Thoracic (T) 6, leptin levels were significantly higher in the former group. T6 appears to be the lowest level of

injury in most patients with SCI to develop autonomic dysreflexia. With SCIs above the level of T6, there is reduced SNS outflow and supraspinal control to the splanchnic outflow and the lower-extremity blood vessels while serum leptin levels in men with SCI correlated not only with BMI but also with the neurologic deficit. This finding supports the notion that decentralization of sympathetic nervous activity relieves its inhibitory tone on leptin secretion, because subjects with tetraplegia have a more severe deficit of sympathetic nervous activity (Wang et al., 2005).

3.2 Multiple sclerosis

No significant difference between ambulatory multiple sclerosis (MS) patients and non MS controls in body composition was found despite lower physical activity in ambulatory MS patients (Lambert et al., 2002). In MS subjects there was no significant relation between any of the body composition measures and the level of disability as measured by the Expanded Disability Status Scale (EDSS). Others found no difference in body fat percent between ambulatory MS patients (Formica et al., 1997) and lower physical activity in ambulatory MS patients vs. controls (Ng & Kent-Braun, 1997). A possible explanation for the similar body composition may be lower energy intake in MS individuals who are ambulatory and greater energy cost of physical activity (walking) in MS than it is with non MS controls (Lambert et al., 2002).

A significant inverse relation between free fat mass (FFM) and EDSS score when ambulatory and non ambulatory MS subjects were combined was found (Formica et al., 1997). On the contrary others without including non ambulatory subjects did not find a significant inverse relation between FFM percent and EDSS score (Lambert et al., 2002). It would seem apparent that ambulatory patients with MS and controls would strengthen the inverse relation between FFM and EDSS score.

The finding of no relation between EDSS score and body fat percent (Lambert et al., 2002) fits well with studies which found no significant relation between the level of physical activity, and the level of disability in individuals with MS (Ng & Kent-Braun, 1997) because MS would likely have a much greater effect on physical activity than on energy intake. According to these findings it appears that the level of disability of ambulatory individuals with MS does not predict body composition. This suggests that a significant level of disability does not force these individuals to be physically inactive and does not result in a greater body fat content. There are many detrimental manifestations of excess body fat, such as hyperlipidemia, insulin resistance, and type II diabetes (Lambert et al., 2002). The largest component of FFM is muscle mass (Lohman, 1986). If muscle mass is lower in individuals with MS than in controls, it may also contribute to the impaired ability to ambulate and perform other activities of daily living. Muscle fiber size from biopsy specimens of the tibialis anterior were 26% smaller than specimens from control subjects (Kent-Braun et al., 1997). Thus, at least for this small muscle, muscle mass was lower in MS. This relationship may not hold for other muscle groups or for whole-body muscle mass (Lambert et al., 2002).

Another reason for skeletal muscle alterations is glucocorticoid usage. The prolonged duration of glucocorticoid causes catabolism of skeletal muscle. Decreased amino acid transport into muscle and increased glutamine synthesis activity with resultant muscle atrophy are some of the concomitant effects of glucocorticoid use on skeletal muscle.

Endogenous glucocorticoid excess also produces generalized osteoporosis, most prevalent in trabecular-rich skeletal regions (Formica et al., 1997).

Beside corticosteroids, immunomodulatory, antiepileptic and antidepressant drugs usually used in individuals with MS, high incidence of vitamin D deficiency, molecular mechanisms and disuse-loss of mechanical stimuli in bone have an effect on bone integrity (most believe that immobilization of these patients is a minor factor in the etiology of osteoporosis) (Dionyssiotis, 2011).

3.3 Stroke

Longitudinal studies of body composition in the elderly have shown that body cell mass decreases with age and is lower in women than in men (Steen et al., 1985). A decline in body fat in both the dependent and independent groups nine weeks after admission was found, indicating consumption of energy stores. In contrast, the change of body cell mass between admission and after 9 weeks was significantly greater in the dependent patients compared with the independent (Unosson et al., 1994). Immobilized individuals lose muscle mass irrespective of nutritional intake because of reduced synthesis of proteins, while the rate of breakdown of proteins is unchanged (Schonheyder et al., 1954). During the recovery period the stroke patients seemed to break down body fat to compensate for energy needs, independent of their functional condition. However, change of body cell mass appeared to relate to the patients' functional condition after stroke (Unosson et al., 1994).

A study in 35 stroke patients compared the body composition, including lean tissue mass, fat tissue mass, and bone mineral content, of the paretic leg with that of the non affected leg in patients with stroke and evaluated the effects of time since stroke, spasticity, and motor recovery on the body composition specifically within the first year after stroke found lean tissue mass and bone mineral content of the paretic side to be significantly lower than those of the non affected side; a significant correlation was found between the lean tissue mass and bone mineral content of both the paretic and non affected legs after adjusting for age and weight. On the contrary bone mineral content and lean tissue mass of both the paretic and non affected sides were negatively correlated with time since stroke in patients with stroke for less than 1 year and a higher lean tissue mass and bone mineral content were found in patients with moderate to high spasticity in comparison with patients with low or no spasticity (Celik et al., 2008).

3.4 Cerebral palsy

Bone mineralization in children with CP has been found lower (bone-mineral values for the total body and total proximal femur) than sex- and age-matched able bodied children. This is illustrated by the BMC Z – scores determined at each skeletal site. The factors that contribute to low bone mineralization include genetic, hormonal, and nutritional problems (especially calcium and vitamin D) and weight-bearing physical activity, oral-motor dysfunction and anticonvulsant medication (Henderson et al., 1995).

Free fat mass (FFM) in cerebral palsy subjects was found significantly lower than that in a normal adolescent population. In 60% of the studied population body fat exceeded the 90th

percentile for age, even if most of the CP children had a low height and weight for age. In female subjects anthropometric measurements were highly correlated with measures of body fatness. Measuring fat by ^{18}O dilution a hydration factor of 0.73 was assumed for FFM. A possible increase in the hydration factor would diminish measured FFM meaning that body fat appears increased. Moreover muscle spasms and spasticity in CP subjects deplete body glycogen. If glycogen is reduced the intracellular water would be reduced and the ratio extracellular water/total body water would increase. The same could result with a loss of body cell mass or an increase in the hydration factor (Bandini et al., 1991).

4. Conclusions

Other important issues according alterations of body composition are the completeness of lesions (an absence of sensory or motor function below the neurological level, including the lowest sacral segment), because body composition seems to be worst than subjects with incomplete lesions (partial preservation of motor and/or sensory function below the neurological level, including the lowest sacral segment) (Sabo et al., 1991; Demirel et al., 1998; Garland et al., 1992) and aging which contributes to major alterations of body composition.

In disabled subjects the most important issue according to body composition is how to promote optimal body weight to reduce risk of diseases such as coronary heart disease, non-insulin dependent diabetes mellitus, lipid abnormalities and fractures because of bone loss. Dietary changes, individualized physical activity programs and medication should be taken in mind in therapy when we deal with this subgroup of subjects. However, self-management of dietary changes to improve weight control and disease should be the case, which means they need to follow diets with lower energy intake and at the same time to eat regularly foods rich in nutrients (Groah et al., 2009).

We need to take in mind that healthy BMI values often underestimate body fat and may mask the adiposity and spasticity did not defend skeletal muscle mass and bone, supporting the concept that in neurologic disabilities the myopathic muscle could not recognize correctly the stimulation because of the neurogenic injury. Moreover, disabled subjects mostly transfer much of the weight-bearing demands of daily activities to their upper extremities reducing the weight-bearing of the affected paralyzed muscles triggering a cycle of added muscle atrophy which interacts with the continuous catabolic action caused by the neurogenic factor. Finally, an irreversible (once established) decline in bone mineral density, bone mineral content as well as geometric characteristics of bone is expected and the duration of lesion-injury is positively correlated with the degree of bone loss.

Further research about body composition is needed in all physical disabilities and more longitudinal studies to quantitate and monitor body composition changes and to modify our therapeutic interventions. However, prevention rather than treatment may have the greatest potential to alleviate these major complications. Therapies should focus on how to perform weight bearing, standing or therapeutically walking activities early in the rehabilitation program to gain benefits according to muscles and bones.

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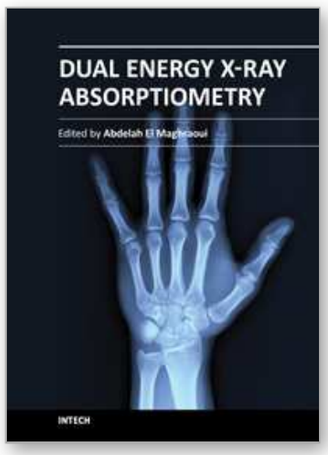

Dual Energy X-Ray Absorptiometry

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The World Health Organization (WHO) has established dual-energy x-ray absorptiometry (DXA) as the best densitometric technique for assessing bone mineral density (BMD) in postmenopausal women and has based the definitions of osteopenia and osteoporosis on its results. DXA enables accurate diagnosis of osteoporosis, estimation of fracture risk and monitoring of patients undergoing treatment. Additional features of DXA include measurement of BMD at multiple skeletal sites, vertebral fracture assessment and body composition assessment, including fat mass and lean soft tissue mass of the whole body and the segments. This book contains reviews and original studies about DXA and its different uses in clinical practice (diagnosis of osteoporosis, monitoring of BMD measurement) and in medical research in several situations (e.g. assessment of morphological asymmetry in athletes, estimation of resting energy expenditure, assessment of vertebral strength and vertebral fracture risk, or study of dry bones such as the ulna).

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