Skeletal Muscle Pathology after Spinal Cord Injury: Our 20 Year Experience and Results on Skeletal Muscle Changes in Paraplegics, Related to Functional Rehabilitation

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Abstract

The present review on 20-year-experience on paralyzed skeletal muscle in paraplegics after traumatic spinal cord injury (SCI), reports changes in muscle fibres and microvasculature seen after morphological, morphometric and ultrastructural studies on open and needle biopsies. The changes were correlated with the time elapsed from SCI (1-17 months). Histopathological and enzyme-histochemical changes in muscle fibres were seen first after 1 month and increased thereafter. In all stages post SCI, paraplegics showed myopathic alterations, increase in the sarcoplasmic lipid contents and incidental denervation patterns. The main ultrastructural changes regard the myofibrillar apparatus and mitochondria. Probably a fibre type shifting to type 2 fibres occurs precociously, but only 7-8 months after SCI it is well manifested. The blood vessel qualitative and quantitative changes in paraplegics regard small vessels and capillaries and they may be important causes of the myopathic alterations in paraplegics. The influence of disuse and spasticity on morphological fibre and capillary modifications in paraplegics is reviewed and discussed. The knowledge of the muscle condition and of plastic capacities for fibre type shifting in paraplegics is important to oppose complications of SCI and to choice of an appropriate rehabilitative program directed to prevention of changes associated to disuse, spasticity and vascular damage.

Key words: fiber types, microvasculature, mitochondria, MHC, pathology, skeletal muscle, spinal cord injury.

Following spinal cord injury (SCI), upper motor neuron paralyzed muscles show widespread disuse atrophy and spasticity. In setting out a rehabilitation program of patients with motor disease it is necessary to evaluate the condition of the kinetic units, i.e. of the osteo-articular segments and of the related skeletal muscle groups. Experimental and clinical studies, and strategies related to rehabilitation of paraplegics as the muscular electric stimulation (FES) and the application of the mechanic orthoses, are directed toward the recovery of the muscle contractile properties and of the standing position or walking. These conditions are important in the preservation of the blood circulation in the paralyzed limb, of the Ca content in bones, of the renal function and for the reduction of spasticity and contractions in paraplegic patients.

However, the results of the functional and morphological changes of the muscle following SCI may condition the choice of the rehabilitative program in paraplegics.

In the last decades, many important experimental and applied studies were performed on the skeletal muscle in paraplegics following SCI, indicating marked changes in the muscle morphology and in their metabolic and contractile properties, and details on the plasticity of the muscle in the present condition. These alterations are well documented in animals, after experimental cord lesion [18, 32], and in humans, generally after traumatic cord lesions [3, 14, 15, 23, 31, 33, 50].

Our study group, in association with the Centre of Functional Recovery of paraplegics of Villanova sul l’Arda (Pc), widely contribute to the morphological documentation of changes in different skeletal muscles and of related pathologies in paraplegics, as the microcirculatory alterations and the heterotopic ossifications [19-22, 38, 40, 42, 45, 46].

Currently, the main mechanism responsible for the skeletal muscle atrophy in paraplegics is tought to be disuse, but muscle fibres following SCI begin to change their functional properties early post injury.
Paraplegia and muscle

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.

The present review reports the results of our 20 year experience on skeletal muscle morphology, on muscle histochemical and metabolic profile and on muscle microcirculation from paraplegics with SCI.

Skeletal Muscle Studies

Muscle fibre morphology and morphometry

Morphological and morphometric studies were performed on different paralyzed muscles. Morphological studies were performed on muscle transverse sections in paraffine-embedded material with routine stains as hematoxylin and eosin and Van Gieson. Quantitative analysis of muscle fibre diameter was performed using an automatic interactive image analysis system (IBAS I-II. Kontron, Bilanalyse, Munich). In the first study we analyzed open biopsies of the rectus femoris muscle in 22 paraplegic patients aged 16-66 years in subsequent stages (1-17 months) starting from the occurrence of SCI [38].

Next, our morphometric studies regarded open biopsies from the gastrocnemius and soleus muscle (composed predominantly of type 2 slow fibres) of 10 paraplegics aged 16-54 years, grouped on the basis to the time elapsed from SCI (1 to 10 months) [20, 21], and biopsies from rectus femoris muscle in 10 young paraplegics aged 16-28 years, divided in 2 groups, 1-5 months and 6-14 months post SCI, respectively [45]. More recently, a morphometric analysis on needle biopsies of quadriceps femoris muscle was performed in 15 male paraplegics aged 20-30 years, 7-14 months post SCI [41].

Skeletal muscle from healthy subjects is composed by trophic fibres with multiple subsarcolemmal nuclei. The histographic analysis of the normal vastus lateralis muscle indicate a mean fibre diameter of 67.2 µm [37]. Fibres are surrounded by a thin endomysium composed by reticular connective tissue and by 4-8 capillaries. In Table 1, a summary of quantitative findings on rectus femoris and quadriceps femoris fibres and capillaries (the fibre atrophy grade, the fibre type percentage and the capillary density and percentage) in different patient groups are reported. Particularly, in a study on rectus femoris muscle in paraplegics [38], in the early times post SCI (1-2 months) muscle fibre atrophy was evident with mean fibre diameter 26 µm. The fibre diameter decreased progressively after SCI and, at least in the first year after injury, was directly proportional to the age of the cord lesion. In this period denervation atrophy patterns with small groups of angulated atrophic or targetoid fibres were observed. 7-9 months post SCI the mean fibre diameter was 20 µm, and at 10-17 months 16.5 µm.

The muscle atrophy in paraplegics 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 [13, 28, 38]. The last alteration may be responsible for the denervation changes seen in early stages post SCI [42]. In the later stages of paraplegia (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 (see Figure 1).

Muscle fibre ultrastructure

Many different ultrastructural changes have been observed in paralyzed muscle [38, 42]. The sarcolemma of atrophic fibres frequently present irregular projections containing many mitochondria, dilated sarcoplasmic reticulum and glycogen granules. In the biopsies obtained 10-17 months after SCI, numerous fibres show vesicular nuclei that sometimes migrate inside the fibres and many sarcoplasmic glycogen granules and vacuoles containing lipid osmiophilic material. These alterations

<table>
<thead>
<tr>
<th>Group (months post SCI)</th>
<th>Fibre diameter (µm)</th>
<th>Fibre type percentage</th>
<th>Capillary measures</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>I</td>
<td>IIA</td>
<td>IIB</td>
</tr>
<tr>
<td>Rectus femoris m.</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1 (1-5)</td>
<td>28*</td>
<td>49</td>
<td>28</td>
</tr>
<tr>
<td>2 (6-10)</td>
<td>22*</td>
<td>40</td>
<td>24</td>
</tr>
<tr>
<td>3 (11-17)</td>
<td>18*</td>
<td>30**</td>
<td>70**</td>
</tr>
<tr>
<td>Control</td>
<td>53</td>
<td>52</td>
<td>40</td>
</tr>
<tr>
<td>Quadriceps femoris m.</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1 (7-14)</td>
<td>26**</td>
<td>45</td>
<td>20</td>
</tr>
<tr>
<td>control</td>
<td>54</td>
<td>54</td>
<td>30</td>
</tr>
</tbody>
</table>

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are limited to the degenerating fibres and are considered myopathic changes in the immobilized and hypoxiemic muscle. The main ultrastructural alterations in paraplegics regard the myofibrillar apparatus, mitochondria and the sarcoplasmic lipid content of the muscle fibres.

In Table 2, morphometric results of the analysis of mitochondria (mean mitochondrial size and % of mitochondria per fibre area) and of lipid droplet percentage per fibre area are also reported.

**Myofibrillar apparatus**

Remarkable alterations in the myofibrillar apparatus have been frequently observed in the medium and old stage of the lesion, with loss and disruption of the myofilaments, abnormalities of the Z line, including smearing and streaming, and frequent formation of rods. These alterations are generally considered as consequence of muscle fibre degeneration.

**Mitochondria**

The mitochondria are generally small in size and sometimes show swelling and intracisternal inclusions. The mitochondrial size and percentage significantly decrease in paraplegics in comparison with normal muscle [38]. Similar changes were observed in muscle disuse in old aged subjects as result of low utilisation of the lipid energy sources [30, 37]. In the immobilized and atrophic muscle, the morphological mitochondrial alterations are accompanied by impairment of the mitochondrial oxidative enzyme activities [51]. Whereas some authors described diffuse morphological damage of mitochondria in disuse atrophy of skeletal muscle

Table 2. Morphometric results of mitochondrial and lipid droplet content in quadriceps femoris muscle from young paraplegics 7-12 months post SCI and control healthy subjects (mean age: 25). Mean ± standard error of the mean (SEM). Statistical level of significance: * P < 0.25; ** P < 0.001.

<table>
<thead>
<tr>
<th></th>
<th>Mean mitochondrial size (µm)</th>
<th>% mitochondria per fibre area</th>
<th>% lipid droplets per fibre area</th>
</tr>
</thead>
<tbody>
<tr>
<td>Controls</td>
<td>0.14 ± 0.09</td>
<td>2.53 ± 0.3</td>
<td>0.48 ± 0.20</td>
</tr>
<tr>
<td>Paraplegics</td>
<td>0.008 ± 0.4**</td>
<td>1.70 ± 0.5*</td>
<td>3.20 ± 1.00**</td>
</tr>
</tbody>
</table>

**Lipid content**

Interspersed round vacuoles either empty or filled with osmiophylic lipid material, are seen in normal skeletal muscle fibres. Lipids are normally utilized by mitochondria to release energy for metabolism and muscle contraction. They increase in the muscle with aging and are expression of disuse and of abnormal mitochondrial function [26, 37]. In paraplegics, the lipid droplets were widely distributed in the cytoplasm, mainly in atrophic fibres, and their percentage per fibre area significantly increased (Table 3) [38]. This phenomenon may be related to decrease in mitochondrial percentage and to the less efficient utilisation of the lipid energy sources following muscle inactivity [26, 38].

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Paraplegia and muscle

Table 3. Mean muscle fibre type diameter in soleus and gastrocnemius muscles of paraplegics (age 16-54) and healthy control subjects.

<table>
<thead>
<tr>
<th>Group (months post SCI)</th>
<th>Soleus muscle</th>
<th>Gastrocnemius muscle</th>
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<tbody>
<tr>
<td></td>
<td>Fibre type</td>
<td>Fibre type</td>
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<tr>
<td></td>
<td>I</td>
<td>IIA</td>
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<td>1 (1-2)</td>
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<td>2 (3-4)</td>
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<td>3 (5-6)</td>
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<tr>
<td>4 (7-8)</td>
<td>26</td>
<td>24</td>
</tr>
<tr>
<td>5 (9-10)</td>
<td>16</td>
<td>21</td>
</tr>
<tr>
<td>Control</td>
<td>50</td>
<td>48</td>
</tr>
</tbody>
</table>

main cause of muscle atrophy in paraplegics. In the recent cord lesion, the degree of muscle atrophy was evident and denervation changes were also seen. In the later stages of paraplegia the muscle atrophy degree increased and myopathic changes with focal necrosis and more extensive fibre degeneration in about 4% of fibres were observed. The degenerative changes in paralyzed muscle may be due to the vascular changes reported below, and the denervation patterns might reflect the presence of some coincidental alterations of the peripheral nerve or trans-synaptic degeneration in the lower motor neuron [13, 28, 38]. In advanced stages post SCI, muscle atrophy and increase in the interstitial endomysial connective tissues and perifascicular fatty infiltration were evident. The significance of the ultrastructural changes, particularly of the decrease in the number of muscle mitochondria and of the increase in cytoplasmic lipid vacuoles, are related to muscle inactivity and degeneration.

Muscle fibre type composition

Normal quadriceps femoris muscle is composed of two major types of fibre characterized by enzymehistochemical methods (myofibrillar ATPase pH 9.6 and 4.6): type 1 and type 2 fibres, and numerous sub-types. In our studies the normal type 1 fibre diameter measures 52.2 µm and the type 2 fibre diameter 48.2 µm [20]. Type 1 fibres are often of smaller diameter, contain many mitochondria and do not stain histochemically for myosin ATP-ase activity. Type 2 fibres are often broad and possess few mitochondria but they stain intensely for myosin ATP-ase activity [8]. In Table 1 and 3, morphometric findings on the fibre diameter and on the fibre type percentage in rectus femoris, quadriceps femoris, soleus and gastrocnemius muscle biopsies from paraplegic patients at times ranging over 1-17 months post SCI are reported. In significantly early time periods post injury (1-4 months post SCI) evident preferential atrophy of type 2 fibres was observed, but no changes in the relative percentage of type 1 and 2 fibres were remarked.

Biopsies performed 4-9 months post SCI showed atrophy of both fibre types with a reduction in the relative percentage of type 1 fibres. Following long term SCI (10-17 months post SCI), upper motor neuron paralysed muscles lose the normal type 1 and 2 type mosaic pattern and become predominantly composed of type 2 fibres. Interesting results have been obtained from the study of the paretic soleus muscle, that normally is predominantly composed by slow type 1 fibres. A significant shift of type 1 fibres to type 2B was observed in the 7-10 months post SCI patient group [20, 21, 38, 42].

The above described results support the presence of progressive changes in paralyzed muscles probably occurring early after cord injury, but most evident 4 months post SCI. The main interesting change in the contractile properties of paralyzed muscle after SCI is the fibre type transformation phenomenon, with type 1 fibre change to type 2, representing down-regulation of the slow MHC isoform and upper-regulation of the fast isoform in those fibres. The shift to type II fibres was more evident in quadriceps and rectus femoris, and in soleus muscle, while in the gastrocnemius muscle the fibre type conversion was less remarkable. Moreover, the plastic modifications of the fibre types was more evident in young paraplegics than in older subjects. This result is probably related to modifications in muscle morphology and in their contractile properties described in the normal sedentary aging man [30, 37]. The fast type II predominance in longstanding paraplegia may explain the problem of muscle fatigability encountered during rehabilitation exercise using FES. Studies on experimental spinal cord transaction showed changes in the rat and cat skeletal muscle, with almost complete type I to type II fibre transformation [18, 52]. These changes are different from those seen in immobilisation in which large increase in the ratio for fast resistant and slow units are seen [24]. It may be suggested that in long term paraplegia the loss of the upper motor neuron control and the spasticity may induce phenomena of fibre type transformation. The described muscular changes in paraplegics are reversible.
after FES and electrically induced training, with partial recovery of the muscle atrophy and with modification in
the fibre type transformation [25].

Myosin heavy chain isoform profile

Studies on myosin heavy chain (MHC) content in muscle fibres from paraplegic patients are very scarce.

The adult normal skeletal muscle fibres express only one MHC isoform. Type 1 fibres have the slow MHC isoform, type 2 fibres have the fast MHC isoform and type C fibres co-express both MHC isoforms. In our studies, the expression in MHC isoform content of the whole biopsy was analyzed with an electrophoretic separation technique [6] on medial gastrocnemius and soleus muscles in paraplegics 1-10 months post SCI.
The results indicated in the early period post SCI (1-6 months) a predominant type 2 muscle fibre atrophy, without changes in the relative percentage of fibre types and MHC content. Eight months post injury, we remarked atrophy of both fibre types with increase in the relative percentage of type 2 fibres and fast MHC content. However, the shift of the type 1 to type 2 fibres was evident 7-8 months post SCI [42]. Fibre type transformation of type 1 to type 2 fibres goes through a transitional phase where they co-express slow and fast myosin isoforms. Data presented by Burnham et al [3], by means of immunofluorescence determination of single fibre MHC isoform, suggest that the onset of fibre shifting to type 2 fibres in paraplegics may be earlier than previously described, probably occurring about 1 month post SCI. This study was performed on vastus lateralis muscle biopsies from 12 paraplegics with SCI over a period of 1-219 months after SCI. Thus, following long term SCI induced paralysis, fibres in the quadriceps femoris and vastus lateralis muscle take on a new state profile defined by a shift in the MHC expression to the fast isoform.

FES and electrically induced training determine in the paralyzed muscle expressing a majority of MHC isoform II B fibres, a fibre type transformation towards the more fatigue resistant MHC isoform II A [25].

Interesting results on paralyzed muscle derive from the characterization of fibre type profile with enzymehistochemistry and from the study of the MHC isoforms over a wide range of post SCI periods. The findings support the concept of progressive stages of change in muscle from paraplegics, suggesting that the earliest alterations post SCI are confined to the morphological muscle aspects (fibre atrophy, focal fibre degeneration with changes in the ultrastructural profile and capillary dilatation). The onset of the fibre type shifting to type 2 fibre probably occurs 1 month after SCI, but only 7-8 months after injury the shift of the type 1 to type 2 fibres is well manifested. This fibre type conversion is related to muscle plasticity, defined as a functional fibre remodeling after different normal and pathological conditions, inducing important changes of the subcellular

Figure 5. A. Normal capillary distribution in a paraffin embedded transverse section of muscle fibres from an healthy subject. Rectus femoris muscle. Immunohistochemical reaction for CD 36. X 60. B. Reduction in the number of capillaries and marked dilatation of the lumen of small blood vessels in gastrocnemius muscle. 12 months after SCI. Immunohistochemical reaction for CD36. X 60.
structure, of the enzyme content, of the metabolic and biochemical characteristics, and of the contractile properties of muscle fibres [10, 34]. The spasticity causes pathological changes in reflex function, such as loss of reciprocal inhibition, cocontraction of agonist and antagonist muscles and the loss of activity in the inhibitory pathways. These alterations, together with disuse and other co-factors, are undoubtedly an appropriate opportunity for the development of the plastic changes in the hypertonic paralyzed muscle [4].

Muscle microcirculation studies

Blood vessels

The first studies on microvasculature from human skeletal muscle concerned normal healthy adults, athletes and normal subjects trained in endurance or strength [1, 2, 7]. They demonstrated a close relationship among the muscle fibre diameter, fibre type and capillary number. The greatest number of capillaries surrounds the type 1 slow fibres. Literature data on normal muscle microcirculation concern qualitative and quantitative analysis of capillaries. In our studies on muscle microcirculation in paraplegics, microscopic qualitative studies were performed using histological and ultrastructural methods. More recently these studies utilized the immunohistochemical characterization of capillaries by mean of antibodies as endotheline and CD 36.

The quantitative evaluation of capillaries was performed with an automatic Interactive Image Analysis Sistem-IBAS I,II (Kontron-Bildanalyse Munich, Germany) on histological sections, the number of capillaries per fibre (C/F), capillaries surrounding a single fibre (CAF), and the capillary density per square millimeter (CD) were determined (Table 1). The clinical and morphological evidences of vascular alterations in paraplegics, as vasomotor disturbances and decrease in the venous distensibility and capacity [16] and structural alterations in the muscle blood vessels [45] may be important causes of the degenerative myopathic changes in paralyzed muscle fibres. These alterations are similar to those seen in patients with chronic venous insufficiency, who develop microangiopathy of cutaneous blood and lymphatic capillaries [11]. In recent SCI (1-4 months post injury), when paralysis with profound hypotonic flaccidity is evident, marked capillary dilatation and interstitial vasogenic oedema were seen. In later stages of paraplegia (10-17 months post SCI), when spasticity becomes evident, paraplegics showed thickening of the arteriolar wall with reduplication of the basal lamina of capillaries and of capillary wall associated to reduction in the capillary number [20, 38, 42, 45]. The above mentioned microvascular changes are also present in the skin of paralyzed muscle and may be expression of a microangiopathy in paraplegics after SCI [11, 20, 42]. These alteration are important and similar changes, as the reduplication and thickening of the capillary basal lamina, have been described in diabetes mellitus, in dystrophia myotonica and in other different microvascular diseases [36, 48]. Human and experimental studies on skeletal muscle inactivity which causes muscle atrophy, showed reduction of capillaries as well as the long standing denervation atrophy that cause reduction and damage of mitochondria and of capillaries [5, 35, 51].

On the basis of these observations, the capillary qualitative and quantitative changes in paraplegics may be due to interaction of different factors: disuse fibre atrophy, reduction in muscle volume, incidental over-imposed denervation, loss of type 1 fibres that are normally surrounded by a great number of capillaries, and shift to the type 2 muscle fibres. Moreover they could be interpreted as a negative phenomenon inducing the ischemic degenerative myopathic changes described in muscle fibres in long term paraplegia [42].

The results of a study on young paraplegics after use of gait orthoses (HGO, ORLAU Parawalker), the period after supply ranging from 5-6 months [41, 43] support the latter hypothesis.

In Table 4, overall capillary and fibre morphometry in rectus femoris muscle from untrained and Parawalker trained paraplegics were reported. After orthotic walking training there was a discrete preservation of the muscle fibre diameter and increase of the muscle capillary supply in comparison with untrained paraplegics. These results are in agreement with those described in experimental spinal cord lesions in animals in which favourable exercise-induced changes in biochemical and contractile properties of muscle fibres and in muscle capillarity were observed [9, 17].

Lymphatics

In paraplegics, immobilization, prolonged bed rest and loss of muscle contraction are main causes of venous stasis in the lower extremities. The venous stasis is frequently associated to microlymphatic changes of the skin of the paralyzed leg and it is an important cause of thromboembolic disease in paraplegics. In the patients, the increase of the deep venous resistance and the venous stasis determine microangiopathic alterations in the skin, in-

<table>
<thead>
<tr>
<th>Untrained paraplegics</th>
<th>26.0 ± 4.2</th>
<th>47.0 ± 4.0</th>
<th>1.10 ± 0.12</th>
<th>340 ± 8</th>
<th>2.70 ± 0.1</th>
</tr>
</thead>
<tbody>
<tr>
<td>Parawalker users</td>
<td>33.0 ± 5.4*</td>
<td>50.0 ± 5.6</td>
<td>1.46 ± 0.6*</td>
<td>300 ± 8</td>
<td>3.60 ± 0.4*</td>
</tr>
<tr>
<td>Normal adults</td>
<td>53.3 ± 7.4</td>
<td>52.0 ± 4.2</td>
<td>1.80 ± 0.1</td>
<td>280 ± 18</td>
<td>4.00 ± 0.2</td>
</tr>
</tbody>
</table>

Table 4. Overall capillary and fibre morphometry in rectus femoris muscle from healthy control subjects, untrained paraplegics and Parawalker trained paraplegics. Mean ± standard error of the mean (SEM). P < 0.0001.
Figure 4. A. Disorganized myofibrils with smearing and streaming of the Z line. Rectus femoris muscle. 7 months post SCI. Electron micrograph. X 7,000. B. Extensive myofibrillar degeneration and vacuolation with presence of lysosomal bodies in the cytoplasm of two adjacent muscle fibres. Rectus femoris muscle. 17 months post SCI. Electron micrograph. X 6,000. C. Two adjacent muscle fibres with subsarcolemmal lysosome accumulation and two small vessels with thickening of the basal lamina. 4 months post SCI. Soleus muscle. Electron micrograph. X 6,000. D. Thickened intramuscular capillary with reduplication of the basal lamina. Soleus muscle. 10 months post SCI. Electron micrograph. X 10,000.
volving blood and lymphatic vessels and eventually leading to skin dystrophic changes and oedema (phlegmasia alba dolens). The morphological identification of lymphatic vessel in the skeletal muscle is difficult. However, in a study, we analyzed lymphatic vessels in the skin of the lower extremities from young male paraplegic patients with and without thromboembolic disease (TED) [44, 46].

In paraplegics with TED of deep veins, skin lymphatic vessels of paretic legs showed dilated lumen and distended wall. The endothelial cells were attenuated and numerous channels among endothelial cells were present. Perivascular collagen and elastic fibres were dissociated by oedema and by the presence of granular material. In paraplegics without TED similar but more rare microlymphatic changes are present.

These morphological changes demonstrated a lymphatic microangiopathy in paraplegics, with lymph stasis and an increased transcapillary diffusion of the lymph material into perivascular dermic tissues, clinically resulting in oedema and reduced removal of tissue catabolites. These leg terminal lymphatic changes and the blood cutaneous microangiopathy probably determines the extent of the trophic disturbances and the ulcer formation in paraplegics. The microangiopathy is the basis for reduction of PO2 and of for destruction of the lymphatic capillary network, seen respectively after transcutaneous PO2 measurements and by fluorescent microlymphography in long term paraplegia and in patients with chronic venous insufficiency [11, 46].

Conclusions and Perspectives

From a rehabilitation perspective it is necessary to evaluate the conditions of the motor unit and the muscle fibre plastic potentialities and reversibility in paraplegics after SCI. The results from the present studies demonstrated changes in the fibre morphology and in their contractile properties, and in the muscle capillarity of paralyzed muscle fibres after SCI. There is a number of rehabilitative programs that are able to modify muscle atrophy and to prevent some clinical complications, by means of physical training, FES, aerobic exercise trainers and bio-mechanic orthoses. In numerous reports, prevention of muscle disuse and improvement in the fibre oxidative capacities after muscle electric stimulation in paraplegics were demonstrated [12, 49]. In adult paraplegics, FES induces changes of morphological, biochemical and histochemical profile, and of contractile properties of muscle fibers and improve the muscle capillarity [31], as well as the use of bio-mechanic orthoses [27, 41, 43] and of aerobic exercise trainers [29]. The muscle fibre atrophy and the fibre type transformation process in paraplegics was partially normalized after electrical induced cycle training [25]. These supports for paraplegic locomotion require high energy cost and may be utilized in patients without cardiovascular and respiratory diseases [27]. The knowledge of the muscle condition and of plastic capacities for fiber type shifting is not only important in attenuating the adverse muscle impact and complications of SCI, but it is also crucial in the choice of an appropriate rehabilitative program directed to preventing the changes associated to disuse and to retrain contractile properties associated to spasticity and microvascular damage. In fact, it is well known that the therapeutic exercise setting out to isometric eccentric or concentric contracture determines selective recruitment of fast or slow fibres which prevents selective fibre type atrophies in paretic muscle. Finally, in an attempt to restore function and regain motor control, many laboratories are now focusing a rehabilitation program based on engineering devices that substitute a motor controlled function (SCI transcutaneous FES walking). It is easily comprehendible that these recovery techniques can be utilized only in well-preserved muscle after SCI. Since it is evident that muscle atrophy and transformation in their fundamental properties occur precociously post SCI, rehabilitative interventions need to be instituted as early as safely possible.

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References


Paraplegia and muscle


