

Skeletal muscle of trained and untrained paraplegics and tetraplegics

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ABSTRACT

The effect of physical conditioning on skeletal muscle of individuals with spinal cord injuries (SCI) has been investigated. The anterior portion of the deltoid muscle (active in wheel-chair propulsion) of untrained and endurance-trained paraplegics and tetraplegics, as well as that of untrained able-bodied subjects, was studied. The characterization involved fibre type distribution, capillarization, fibre areas and also oxidative and glycolytic enzyme levels. A general trend towards a successively higher proportion of type I fibres and lower proportion of type IIB fibres was noted in the order of able-bodied subjects (type I, 42%; type IIB, 41%, $n = 8$), paraplegics (type I, 57%; type IIB, 13%, $n = 13$) and tetraplegics (type I, 74%; type IIB, 4.5%, $n = 11$). The trained SCI groups had significantly higher levels of the citric acid cycle marker enzyme citrate synthase (34% and 63%) than the untrained SCI groups and able-bodied subjects, respectively. The glycolytic marker enzyme 6-phosphofructokinase was 32% lower in the tetraplegic groups than in the other groups. In contrast, the fatty acid oxidation marker enzyme 3-hydroxyacyl-CoA dehydrogenase was markedly higher in the tetraplegic group than in the able-bodied subjects (58%) and tended to be higher (21%, $P < 0.1$) than in the paraplegic group. The trained SCI groups displayed significantly higher (28%) levels of capillaries per fibre than the untrained SCI groups, which had about the same levels as the untrained able-bodied subjects. It is concluded that several of the findings are in line with normal muscular adaptation, whereas others are unexpected and support a hypothesis that some of the findings might be due to differences between the groups in, for instance, hormone levels or in types of muscular load.

Keywords capillaries, fibre types, glycolytic enzymes, human skeletal muscle, oxidative enzymes, paraplegia, physical conditioning, tetraplegia.

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The adaptation of human skeletal muscle to endurance training has been described fairly extensively with regard to healthy able-bodied individuals (see e.g. Saltin & Gollnick 1983, Schantz 1986). It is known from these studies that physical training may result in a major adaptation regarding, for instance, oxidative enzyme levels, capillary supply, fibre areas and the isoform composition of contractile and regulatory proteins. The functional importance of this adaptation is considered to be of great significance.

Considerably less is known regarding the adaptability in many abnormal states, including para- and tetraplegia. Taylor *et al.* (1979) studied m. triceps brachii in five paraplegic wheelchair athletes and compared the results obtained with data on highly trained able-bodied athletes in the literature. It was suggested that the trained paraplegics had greater

fibre areas, but lower levels of glycolytic and oxidative marker enzymes. Differences in experimental procedures between the studies make their interpretation less reliable, however. Grimby *et al.* (1976) studied the middle portion of the deltoid muscle in a mixed group of seven para- and tetraplegic subjects with old injuries but unspecified physical activity levels. Based on comparisons with the literature distribution of fibre types, fibre areas as well as oxidative and glycolytic enzyme levels were interpreted as being normal. This could be due to the fact that the studied muscle portion is not of prime importance for wheelchair ambulation.

The active muscle mass in wheelchair propulsion and control of the autonomic nervous system vary greatly between paraplegics and tetraplegics. This results in differences in the muscular efforts made. For

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example, both the normal daily and the maximal propulsion velocities (which reflect the contraction velocities) of the tetraplegics are about half that of the paraplegics (Schantz *et al.* unpublished results).

Against this background, the aim of this study was to illuminate whether physical training of individuals with spinal cord injuries may result in a muscular adaptation, and in such a case, whether any differences can be observed between paraplegics and tetraplegics. For this purpose, biopsy specimens were taken from the anterior deltoid muscle, which is active in wheelchair propulsion, of untrained and endurance-trained paraplegics and tetraplegics as well as of untrained able-bodied subjects. Standard biochemical and histochemical methods were used to study glycolytic and oxidative enzyme levels, capillarization and fibre types and areas.

MATERIALS AND METHODS

Subjects

The study consisted of five different groups of male subjects: Untrained (UT, $n = 6$) and endurance trained tetraplegics (TT, $n = 5$), untrained (UP, $n = 8$) and endurance trained paraplegics (TP, $n = 5$) and untrained able-bodied subjects (UA, $n = 8$). Their age, height, weight, years of persisting injury and injury levels have been listed in Table 1. All cases of tetra- and paraplegia were due to traumatic spinal cord injuries. The injuries were complete in all cases but in two out of six UT, two out of five TT and one out of eight UP subjects. All spinal cord injury (SCI) subjects relied solely on wheelchairs for the ambulation. The TT subjects had been involved in wheelchair-training for an average of 6 ± 3 years, whereas the corresponding figure for TP was 12 ± 7 years. During the two months prior to the biopsy sampling the average time devoted to wheelchair training was 4×50 min per week for TT and 4×65 min per week for TP. The training regimen during this period also included one strength training session per week for three tetraplegics and three sessions per week for one paraplegic subject. The subjects were informed about the procedure and risks involved in the experiments before they volunteered. The study was approved by the Committee on Ethics of the Karolinska Institute.

Biopsy procedure

Muscle biopsy specimens were taken from the anterior portion of the deltoid muscle by means of the biopsy technique employing a conchotome (Henriksson 1979). All samples were from the non-dominant arm. No training of the deltoid muscle had been undertaken

during a period of 24 h before the biopsy sampling. Specimens for enzyme assays were frozen immediately in liquid nitrogen and stored at -80°C until analysed. Specimens for histochemical analyses were mounted in an embedding medium (Tissue-Tek II, OCT compound, Lab-Tek Products, Naperville, IL), frozen in isopentane cooled to its freezing point with liquid nitrogen and stored at -80°C for subsequent analyses.

Histochemical analysis

Serial transverse sections ($10\ \mu\text{m}$) were cut with a microtome at -20°C and stained for myofibrillar adenosine triphosphatase (ATPase) to identify fibre types (Brooke & Kaiser 1969, 1970; Dubowitz & Brooke 1973) and by the amylose-periodic acid-Schiff (PAS) method to visualize capillaries (Andersen 1975). The myofibrillar ATPase stainings of the tetraplegics were subjected to a histopathological examination (see Discussion). Capillary counts were performed as described by Andersen & Henriksson (1977a). Determinations of fibre type distribution and fibre type areas were made using a computerized image analyser (Comfas, SB 1024-SA, Scan Beam A/S Hadsund, Denmark). Capillary counts were based on an average area of 1.64 (0.24–5.56) (mean and range) mm^2 comprising 217 (22–608) fibres. The determination of fibre type distribution was based on 780 (185–1739) fibres. Fibre area determinations were based on 54 (17–105) type I fibres, 26 (9–56) type IIA fibres and 24 (3–87) type IIB fibres. Mean fibre areas were calculated on the basis of the percentages of each fibre type and the corresponding fibre type area.

Enzyme assays

Muscle specimens were weighed at -20°C and homogenized (1:100 wt/vol) by hand in Potter-Elvehjem homogenizers containing ice-cooled potassium phosphate buffer (0.3 M, pH 7.7) with 0.05% (wt/vol) bovine serum albumin. The homogenate was stored at -80°C until analysed. Activities of the following enzymes were analysed spectrophotometrically according to the references cited: 6-phosphofructokinase (PFK, EC 2.7.1.11; Opie & Newsholme 1967), citrate synthase (CS, EC 4.1.3.7; Alp *et al.* 1976), 3-hydroxyacyl-CoA-dehydrogenase (HAD, EC 1.1.1.35; Bass *et al.* 1969) and malate dehydrogenase (MDH, EC 1.1.1.37; Bücher *et al.* 1964). The cytoplasmic and mitochondrial isozymes of malate dehydrogenase (cMDH, mMDH) were assayed as described in detail by Schantz (1986). All enzymes were assayed in duplicate at 25°C under conditions in which enzyme concentrations were proportional to enzyme activities.

Table 1 Age, height, weight, years of injury (mean ± SD) and injury levels of the subjects

	Able-bodied untrained (UA) <i>n</i> = 8	Paraplegics		Tetraplegics	
		Untrained (UP) <i>n</i> = 8	Trained (TP) <i>n</i> = 5	Untrained (UT) <i>n</i> = 6	Trained (TT) <i>n</i> = 5
Age (years)	28 ± 6	32 ± 9	33 ± 1	31 ± 9	30 ± 9
Height (m)	1.85 ± 0.08	1.77 ± 0.06	1.77 ± 0.08	1.82 ± 0.05	1.85 ± 0.05
Weight (kg)	84 ± 24	71 ± 5	64 ± 9	62 ± 7	61 ± 12
Years of injury	—	12 ± 10	16 ± 8	10 ± 8	11 ± 10
Injury levels	—	T3–T12	T4–T10	C4–C6	C5–C8

Statistics

Values are presented as means ± 1 standard deviation or range. Differences between the untrained able-bodied subjects and the four spinal cord injury groups were tested for significance using a one-factor ANOVA and Dunnett's test for comparisons involving a control mean. A two-factor ANOVA was applied for comparison between the untrained and trained paraplegics and tetraplegics, respectively, as well as between the paraplegics and tetraplegics. When there was a tendency ($P < 0.1$) to significant interaction between the two ways (training status and injury status), a one-way ANOVA and Scheffé's post hoc test were used to localize differences between the groups. Only significant differences ($P < 0.05$) are stated in the text, unless otherwise indicated.

RESULTS

Muscle fibre type distribution

Able-bodied vs. spinal cord injury groups. The percentage of type I fibres was significantly higher in all spinal

cord injury groups (59, 66 and 82%), with the exception of the trained paraplegics (55%), than in the untrained able-bodied subjects (42%). No significant differences were observed in type IIA percentage, whereas significantly lower type IIB percentages were noted in all spinal cord injury groups (4, 5, 11 and 15%) compared to the untrained able-bodied subjects (41%). No difference existed in the percentages of myofibrillar ATPase intermediate (IM) fibre types (i.e. types IB, IB-IIC and IIC which contain a mixture of slow and fast contractile and regulatory proteins, see Schantz 1986) (Table 2, Fig. 1).

Comparisons between the spinal cord injury groups. There were no significant differences in type I percentages between the trained and untrained states, whereas the trained tetraplegic group displayed a higher value (82%) than the trained paraplegics (55%). No significant difference in type IIA percentages was observed, whereas the tetraplegic groups exhibited lower type IIB percentages (4% and 5%) than the paraplegic groups (11% and 15%). There were no differences in type IM percentages (Table 2, Fig. 1).

Table 2 Fibre type distribution in the anterior deltoid muscle of able-bodied subjects and spinal cord injury subjects (mean ± SD)

	Able-bodied untrained (UA)	Paraplegics		Tetraplegics		Trained vs. Untrained	Paraplegics vs. Tetraplegics
		Untrained (UP)	Trained (TP)	Untrained (UT)	Trained (TT)		
Type I %	42 ± 7	59 ± 7	55 ± 13	66 ± 17	82 ± 18	*	*
Type IIA %	17 ± 8	25 ± 12	32 ± 13	26 ± 14	13 ± 14	*	*
Type IIB %	41 ± 9	15 ± 6	11 ± 9	5 ± 6	4 ± 5	n.s.	$P < 0.05$
Type IM %	0.1 ± 0.15	0.6 ± 1.0	2.0 ± 3.4	3.1 ± 5.7	0.8 ± 1.4	n.s.	n.s.

Brackets denote (with one exception, see below) significant differences ($P < 0.05$) between untrained able-bodied subjects and other groups. Results of statistical analyses between trained and untrained spinal cord injured groups as well as between paraplegics and tetraplegics are indicated separately. Asterisks denote a tendency ($P < 0.1$) to a significant interaction term between the factors studied (training and injury) in the two-factor ANOVA. The statistical evaluation undertaken in that situation is described in Methods. It resulted in a significant difference in type I % between TP and TT as designated by a bracket.

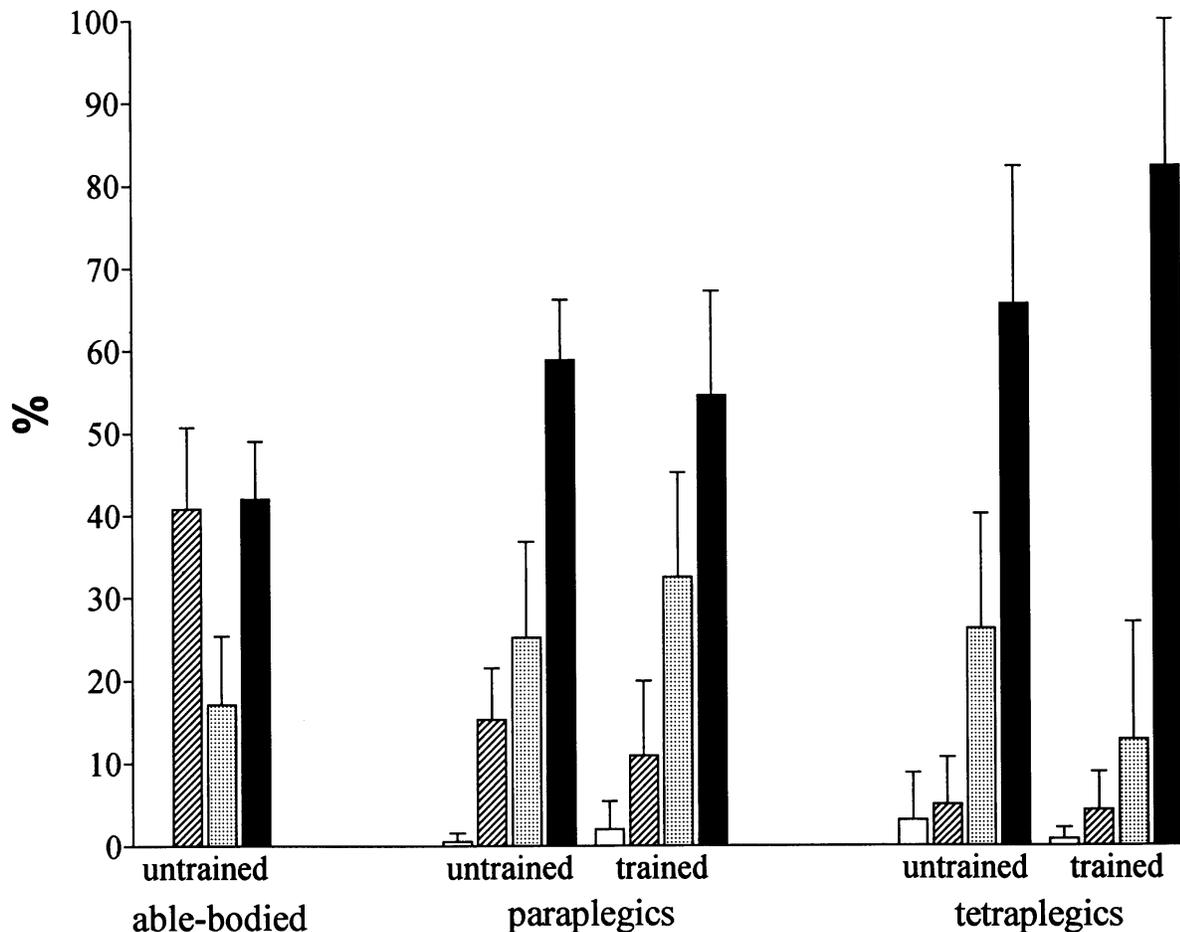


Figure 1 Fibre type distribution in the anterior deltoid muscle of able-bodied subjects and spinal cord injured subjects (mean + 1 SD). Key: (□), type IM; (▨), type IIB; (▩), type IIA; (■), type I. Type IM stands for myofibrillar ATPase intermediate fibres (types IB, IB-IIC and IIC). For further explanations see the text.

Table 3 Fibre areas (μm^2) in the anterior deltoid muscle of able-bodied subjects and spinal cord injury subjects (mean \pm SD)

	Able-bodied untrained (UA)	Paraplegic		Tetraplegics		Trained vs. Untrained	Paraplegics vs. Tetraplegics
		Untrained (UP)	Trained (TP)	Untrained (UT)	Trained (TT)		
Type I	5980 (1710)	6770 (1650)	8340 (2410)	6070 (1120)	6180 (850)	n.s.	$P < 0.05$
Type IIA	7840 (1720)	8080 (2070)	10840 (3340)	8000 (2720)	9630 ($n = 4$) (2320)	$P < 0.1$	n.s.
Type IIB	7530 (2960)	7750 (2280)	10840 (4280)	6610 ($n = 3$) (1220)	10200 ($n = 2$) (1110)	$P < 0.1$	n.s.
Mean fibre area	6920 (2140)	7200 (1760)	9320 (2770)	6870 (1550)	6850 (1450)	n.s.	$P < 0.1$

No significant differences were noted between untrained able-bodied subjects and the other groups. Results of statistical analyses between trained and untrained spinal cord injured groups as well as between paraplegics and tetraplegics are indicated to the right.

Fibre areas

Able-bodied vs. spinal cord injury groups. No significant differences were observed (Table 3).

Comparisons between the spinal cord injury groups. The only significant difference was that type I fibre areas were significantly larger (10–37%) in the paraplegic group than in the tetraplegic group (Table 3).

Table 4 Capillary supply in the anterior deltoid muscle of able-bodied subjects and spinal cord injury subjects (mean \pm SD)

	Able-bodied untrained (UA)	Paraplegics		Tetraplegics		Trained vs. Untrained	Paraplegics vs. Tetraplegics
		Untrained (UP)	Trained (TP)	Untrained (UT)	Trained (TT)		
cap \times fibre ⁻¹	1.55 \pm 0.26	1.61 \pm 0.27	2.21 \pm 0.52	1.70 \pm 0.36	2.01 \pm 0.35	<i>P</i> <0.05	n.s.
cap \times mm ⁻²	228 \pm 29	219 \pm 41	236 \pm 68	226 \pm 48	265 \pm 11	n.s.	n.s.

Brackets denote significant differences (*P*<0.05) between untrained able-bodied subjects and other groups. Results of statistical analyses between trained and untrained spinal cord injured groups as well as between paraplegics and tetraplegics are indicated separately.

Table 5 Enzyme activities (μ mol g wet wt⁻¹ min⁻¹) in the anterior deltoid muscle of able-bodied subjects and spinal cord injury subjects (mean \pm SD)

	Able-bodied untrained (UA)	Paraplegics		Tetraplegics		Trained vs. Untrained	Paraplegics vs. Tetraplegics
		Untrained (UP)	Trained (TP)	Untrained (UT)	Trained (TT)		
PFK	31.4 \pm 2.0	26.5 \pm 4.0	30.9 \pm 6.4	21.0 \pm 5.6	19.4 \pm 5.5	n.s.	<i>P</i> <0.05
cMDH	170 \pm 26	187 \pm 46	199 \pm 28	198 \pm 44	253 \pm 43	<i>P</i> <0.1	<i>P</i> <0.1
mMDH	65.6 \pm 10.7	73.6 \pm 14.4	85.0 \pm 23.3	82.2 \pm 4.8	89.2 \pm 24.4	n.s.	n.s.
HAD	4.6 \pm 0.7	5.9 \pm 1.1	6.1 \pm 0.8	6.7 \pm 2.0	7.8 \pm 1.8	n.s.	<i>P</i> <0.1
CS	8.9 \pm 1.0	10.1 \pm 1.8	13.5 \pm 2.6	11.6 \pm 1.4	15.5 \pm 3.5	<i>P</i> <0.05	n.s.

Brackets denote significant differences (*P*<0.05) between untrained able-bodied subjects and other groups. Results of statistical analyses between trained and untrained spinal cord injured groups as well as between paraplegics and tetraplegics are indicated separately.

Capillary supply

Able-bodied vs. spinal cord injury groups. The only significant difference was a 43% higher level of capillaries *per fibre* in the trained paraplegics than in the untrained able-bodied subjects (Table 4).

Comparisons between the spinal cord injury groups. No difference was seen in the number of capillaries per mm², whereas the number of capillaries per fibre was 18 and 37% higher in the trained paraplegics and tetraplegics than in the untrained para- and tetraplegics, respectively (Table 4).

Enzyme activities

Able-bodied vs. spinal cord injury groups. On the average the activity of PFK was 36% lower in the tetraplegics than in the untrained able-bodied subjects. On the other hand, about 50–75% higher activities of cMDH, HAD and CS were noted in the trained tetraplegics compared with untrained able-bodied subjects. Significantly higher activities of HAD (45%) were also noted in the untrained tetraplegics, and a 52% higher CS activity was noted in the trained paraplegics compared with untrained able-bodied subjects (Table 5, Fig. 2).

Comparisons between the spinal cord injury groups. On the average, the activity of PFK was 42% higher in the paraplegics than in the tetraplegics, and on the average the trained tetra- and paraplegics displayed 34% higher CS activities than the corresponding untrained groups (Table 5, Fig. 2).

DISCUSSION

The results provide evidence of a substantial adaptation following spinal cord injury (SCI). Some of the findings are in line with what is normally seen with endurance training, whereas other are unexpected.

The results focus attention on the considerably lower levels of adrenaline and noradrenaline in tetraplegics at rest and during physical exercise (Bloomfield *et al.* 1994, Kjaer *et al.* 1996). The possible importance of this, as well as the qualitative differences in physical work performed, will be discussed.

Fibre types

A general trend towards successively lower type IIB and higher type I percentages was noted when the untrained able-bodied subjects were compared with the paraplegics and tetraplegics.

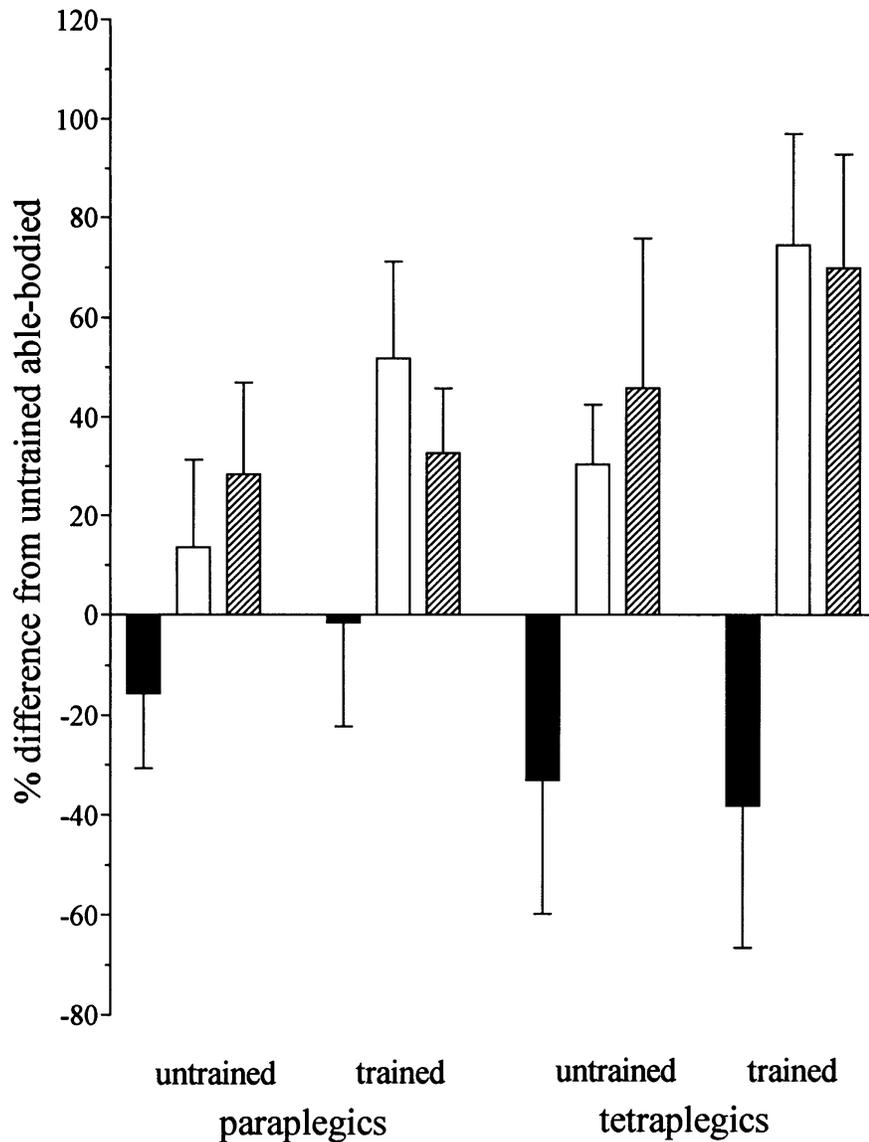


Figure 2 Enzyme activities in the anterior deltoid muscle of spinal cord injured subjects expressed in per cent (mean + or -1 SD) above or below the mean value of the untrained able-bodied subjects. Key: (■), PFK; (□), CS; (▨), HAD.

The injury levels of the tetraplegics (C4–C8) were in the same region as the segmental innervation of the deltoid muscle (C5–C6). This could have included an upper or lower motor neuron lesion. These possibilities therefore have to be discussed. In case of an upper motor neuron lesion, a type II dominance and atrophic fibres would be expected (Grimby *et al.* 1976). That was not observed. A lower motor neuron disorder could induce collateral sprouting and fibre type grouping, i.e. when a fibre is totally enclosed by fibres of its own type. This could explain the high percentage of type I fibres. However, in such cases fibre type grouping would also be observed in type II fibres. In a histopathological examination there were, however, no signs of type grouping in the type II

fibres. Neither were any atrophic fibres or other signs of neurogenic pathology observed. Thus, the high type I proportion in the tetraplegics does not appear to be due to an upper or lower motor neuron disorder.

The literature about catecholamine effects on fibre type composition provides conflicting results (see Svedenhag 1985, Zeman *et al.* 1988, Aboudrar *et al.* 1993). It is therefore difficult to evaluate their possible role for the present findings.

Fibre type transformations from type IIB to type IIA (Andersen & Henriksson 1977b) and from type II to type I (see Schantz 1986) can, however, occur in response to endurance training. The present findings could therefore be interpreted in terms of a gradually greater strain on the anterior deltoid muscle with

higher injury levels. This does not necessarily have to do with the regular physical training *per se* since no significant differences were noted between the trained and untrained subjects within the different SCI groups.

The muscular work of the para- and tetraplegics in daily life is therefore of interest. It is a common observation that tetraplegics are more dependent on the deltoid muscle for the balance of their upper body than paraplegics. Thus, the tetraplegics' deltoid muscle serves a postural function with a substantial portion of more or less isometric contractions. Furthermore, the self-chosen speed of tetraplegics during wheelchair propulsion in normal daily life, as well as their maximal speed, is about half that of the paraplegics (Schantz *et al.*, unpublished results). Since the wheel and rim diameters were identical in the two groups, the speeds of contraction in the anterior deltoid muscle are clearly lower in the deltoid muscle of the tetraplegics.

In this context the finding that hypergravity results in a greater type I percentage (rats, Martin & Romond 1975) is of interest. It is also of interest that an involvement of fast, in contrast to slow, fibres in isometric or slow motions is unfavourable in energetic terms (Suzuki 1979, Katz *et al.* 1986, Coyle *et al.* 1992). Therefore, from a teleological viewpoint a changeover from fast to slow fibres could be appropriate for the tetraplegic subjects.

Enzymes

The markedly higher levels of the citric acid cycle enzyme CS in the trained para- and tetraplegics than in the untrained able-bodied subjects and the respective untrained SCI groups are normal with endurance training. The findings with regard to the tetraplegics lend support to an adaptive mechanism that is independent of the sympathetic nervous system (Kraus *et al.* 1989), but also in this matter conflicting data exist (Svedenhag 1985).

The pattern of PFK levels is, however, clearly unexpected. Normally, no differences are seen in glycolytic enzyme levels in connection with endurance training (cf. Saltin & Gollnick 1983). However, the PFK activity in the tetraplegics is about 30–35% lower than in the paraplegics and able-bodied subjects. Part of this difference can be due to the fact that the glycolytic enzyme levels in type II fibres are normally about double those of type I fibres in the untrained and endurance trained state (Essén *et al.* 1975, Essén-Gustavsson & Henriksson 1984). Taking these differences into account, about half of the difference in PFK activity between the groups can be explained. The other 50% remains, however, to be explained. The lower levels of catecholamines in tetraplegics is here of interest since adrenaline has been shown to

stimulate glycogenolysis (Jansson *et al.* 1986, Spriet *et al.* 1988). Thus, a lower glycolytic flux could be reflected in lower PFK values.

In this context, the findings related to the fatty acid oxidation enzyme HAD are of particular interest. Normally, about 50% higher levels of HAD are seen in type I fibres (Billeter *et al.* 1978, Chi *et al.* 1983, Essén-Gustavsson & Henriksson 1984). Taking the fibre type area proportions into consideration, the para- and tetraplegics could be expected to have about 10% higher levels of HAD than the able-bodied subjects. Instead, they display about 30 and 60% higher activities, respectively. This is of the same order of magnitude as the differences seen in CS. However, physical training of both arm and leg muscles normally induces considerably smaller changes (< 50%) in HAD than in citric acid cycle enzymes (Jansson & Kaijser 1977, Schantz *et al.* 1983, Wibom *et al.* 1992). What may have caused the abnormally high HAD values in the tetraplegics is not known, but they can be interpreted in terms of the lower values in PFK being compensated for by higher values in HAD, reflecting a greater dependence on fatty acid oxidation.

Capillarization

Whereas no difference in capillaries per mm² was observed, the trained groups displayed 18–37% higher levels of capillaries per fibre than the corresponding untrained groups. Thus, it appears that the capillary neoformation normally seen in endurance training may also occur in para- and tetraplegia. The finding relating to the tetraplegics is in concordance with Svedenhag (1985) who noted the same degree of training-induced capillary neoformation in human skeletal muscle with and without beta-blockade.

Fibre type transformation – an independent adaptive response?

It is a well-known fact that physical conditioning in man may induce adaptation in mitochondrial enzymes and capillary supply without a simultaneous or subsequent fibre type II to type I transformation (Saltin & Gollnick 1983). But when signs of transformation have occurred, an adaptation of metabolic variables has also been noted (cf. Schantz 1986).

Electrical stimulation of animal muscles reveal an adaptive sequence with mitochondrial and capillary adaptation preceding type II to type I transformation (Salmons & Henriksson 1981). A metabolic adaptation preceding or occurring simultaneously with the fibre type transformation process has therefore been looked upon as a compulsory part of an integrated adaptive process. The present study contradicts that viewpoint

since the untrained paraplegics and tetraplegics show large differences in fibre type distribution compared with the untrained able-bodied subjects (type I: UP, 59%; UT 66%; UA 42%), but only minor and non-significant differences in capillaries per fibre (UP, 1.6; UT 1.7; UA 1.6) and CS activity (UP, 10.1; UT 11.6; UA 8.9). Corresponding results have been obtained by Borg & Henriksson (1991) in the anterior tibial muscle of prior polio patients.

Thus, it appears that type II to type I transformation can occur independently of adaptation in mitochondrial enzymes and capillaries. It is conceivable that this is due to an increased load of predominantly slow or isometric contractions with limited metabolic demands.

In conclusion, several of the present findings are in line with normal muscular adaptation, whereas others are unexpected. The low glycolytic and high fatty-acid oxidation enzyme levels in the tetraplegic groups are difficult to interpret in terms of normal adaptation to physical training or differences in fibre type distribution. Furthermore, great differences in fibre type distribution, compared with the able-bodied subjects, are also seen in the untrained SCI groups with only normal daily usage of the muscles. This is not connected with significant differences in levels of enzymes or capillary supply. The differences in fibre type distribution between the paraplegics and tetraplegics were also unexpected and support the conclusion that some of the findings might be due to differences in, for instance, hormone levels or in qualitative differences in muscular load between the groups.

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