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Paravertebral muscles in experimental scoliosis: a light and electron microscopic study

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Summary. Experimental structural dextroconvex scoliosis was produced in rabbits by costotransversolisis with transversectomy and releasing of paravertebral muscles between TVII and TX on the right side. Two compensatory curves developed on the upper dorsal and lumbar levels. Biopsies of paravertebral muscles in experimental animals included, besides areas of normal tissue, a considerable derangement of the cell contractile apparatus with sarcoplasmic dilation and eventual cell disintegration and necrosis. Histological changes varied along levels, the convexity being more affected. The severity of changes and reduction in body weight and length were correlated with the degree of scoliosis. A selective atrophy of slow-twitch fibers was observed in experimental animals, especially at the level of the main curve, whereas fast-twitch fiber atrophy was more important caudally. Control animal biopsies always appeared normal. Our experimental model shows an overt participation of paravertebral muscles in the establishment of compensatory processes following scoliosis, although the role that paravertebral muscles play in the etiopathogenesis of human idiopathic scoliosis requires further investigation.

Key words: Experimental idiopathic scoliosis, Paravertebral muscle

Introduction

The etiology of idiopathic scoliosis remains unknown in up to 80% of cases, although different factors are considered to participate in its etiopathogenesis (Ponseti, 1967; Wynne-Davis, 1975; Yamada et al., 1984; Dickson, 1992; Skaggs and Bassett, 1996). Among other theories, we could mention asymmetrical vertebral growth at the paired neurocentral junctions (Knutsson, 1966), developmental instability (Goldberg et al., 1995), abnormalities in the skeletal muscle (Spencer and Zorab, 1976; Sahgal et al., 1983; Karski, 1996), melatonin deficiency (Machida et al., 1995), as well as other neurological (Machida et al., 1994; Woods et al., 1995), neurohormonal (Machida et al., 1993), histological (Hadley-Miller et al., 1994), metabolic (Worthington and Shambaugh, 1993), genetic (Cowell et al., 1972) and developmental (Karaharju, 1967) factors that may produce or favor the deformity. Nevertheless, scarce progress has been made to elucidate which processes are primary and which are secondary.

Paravertebral muscles, as active elements basic to maintain spinal dynamic equilibrium, have been studied in scoliosis associated to neuro- (Schneider et al., 1991; Maguire et al., 1993) and myopathies (Fitzsimons and Tyer, 1980; Sahgal et al., 1983). Likewise, the electromyographic activity in paravertebral muscles (Riddle and Roaf, 1953; Shimada, 1989), as well as the histopathological features present in idiopathic scoliosis have been reported (Hoppenfeldt and Spiro, 1973; Spencer and Eccles, 1976, Spencer and Zorab, 1976; Sahgal et al., 1983; Zetterberg et al., 1983). The most outstanding feature is an increase of type I fibers on the convex side (Zetterberg et al., 1983). An increase in the number of capillaries on the same side as well as disruption of myofilaments, Z-band streaming and subsarcolemmal accumulation of glycogen, lipids and mitochondria have also been reported (Sahgal et al., 1983). Other papers have stressed particular aspects, such as sarcolemma abnormalities at the myotendinous junction (Khosla et al., 1980), membranous bodies in nerve fibers and presence of leptofibrils in intrafusal muscle fibers (Low et al., 1983), and increased level of calcium in paraspinal muscles (Yarom et al., 1981).

Although scoliosis can be considered practically a specific deformity of human species, some authors have reported this affliction in animals (Taylor, 1971). Different attempts have been designed to establish experimental models of scoliosis (Spencer and Zorab, 1976; Suk et al., 1989; Herrera-Marschitz et al., 1990; Pal et al., 1991; Willers et al., 1995) which resemble as much as possible human idiopathic scoliosis regarding

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its morphology, structure and evolution. Surgical approaches producing localized lesions on muscle, ligaments, ribs or vertebrae during postnatal development offer the best results (Arnd, 1903; Langeskiöld and Michelsson, 1961; Stilwell, 1962; Karaharju, 1967; Cañadell et al., 1978; Hakkarainen, 1981; Kasuga, 1994).

On animal models, several authors have found muscle atrophy (Suk et al., 1989), variations in proportion of fiber types (Barrios et al., 1989) and other abnormalities in paravertebral muscles. These variations between reports are due to different techniques used to provoke scoliosis. Some other workers, however, found no difference between muscles of normal and scoliotic rabbits (Spencer and Zorab, 1976).

Furthermore, Maffulli (1990) reported no evidence of myopathic changes in idiopathic scoliosis although confirmed a prevalence of slow-twitch fibers. However, studies on children (Wright et al., 1992) with implantable muscle stimulators seem to conclude that the increased percentage of type I fibers is not the cause of scoliosis. Thus, no conclusive results are given to fully understand the participation of paravertebral muscles in the mechanism of production of idiopathic scoliosis.

In the present paper we provoked surgical experimental scoliosis in rabbits with the aim of clarifying the role of paravertebral muscles in the development and maintenance of scoliotic curves. This data would be of interest for the better understanding of the etiopathogenesis of idiopathic scoliosis.

Materials and methods

Subjects

A total number of 35 one month-old Californian rabbits *Oryctolagus cuniculus* of both sexes were used. Twenty-five received surgery consisting in liberation of paravertebral muscles along with costotransversolisis and transversectomy on the right side, while the remaining 10 were not operated and served as control group. In all the cases the National Research Council's guide for the care and use of laboratory animals was followed. Body parameters and ages of the animals used are shown in table1.

Surgery

Animals undergoing surgery were induced to anesthesia with Ketamine sulfate 5mg/Kg i.m. and a second injection of the same anesthetic was administered in the contralateral side to get a deep anesthesia which was reached after 15 min and prolonged during 30 min more.

After a 5-cm-long incision on the back, the skin was opened and cutaneous muscle was sectioned. On the muscle-aponeurotic plane exposed, a conspicuous venous vessel could be noted above the spinous process, this vessel running along the whole length of the rabbit's dorsum had to be electrocoagulated between the levels of interest: from TVII to TX. The fascia of the rabbit large m. spinalis dorsi was then sectioned in the midline on spinous processes. Subsequently, on the right side, between levels TVII and TX, a muscle-tendinous subperiostic release following the spinous process tract was performed. This muscle release was carried out first on the large m. spinalis dorsi and secondly on the common paravertebral mass, in this case following the tract of the vertebral laminae. After skeletization of spinous processes and laminae, two small Sem retractors were introduced in a wet gauze to laterally drive the deinserted paravertebral muscles. Then, the tendinous insertions of the large m. spinalis dorsi were subperiostically sectioned on the transverse processes and on the TX mammilary process. TX vertebra was then resected and the dissection and muscle release was finished by sectioning small intertransverse muscles. Special care was taken to maintain the viability of the deinserted muscle, laterally driven and constantly covered by gauzes impregnated by temperate saline solution.

The next step in the technique involved sectioning of ligaments and bones which was crucial for success. Between the same levels, TVII to TX, the posterior costo-transverse, the anterior costo-transverse, the intertransverse and the lamino-costal ligaments were sectioned. A small curved spatula had to be introduced into the articular space between the rib and the transverse process, moving inside without resistence to prove complete sectioning. Articular cartilage and small articular menisci of these joints also had to be removed. Finally, the transverse processes of the referred to levels on the same side were resected in about two millimeters by means of a Steven's scissors.

In all steps, maximal care had to be paid to the pleura and to intercostal neurovascular packages. Pieces of interscapular brown adipose tissue were introduced into the four costo-transverse spaces resected to avoid further cicatricial fibrosis. Furthermore, both the proximal and distal limits of the muscles involved in the operation were sutured with a wire lace. After a new irrigation of the field with temperate saline, surgery was finished with a discontinuous suture, single separate stitches of reabsorbible material on the aponeurosis over

Table 1. Ages and body parameters of control and experimental groups of animals at the beginning and end of the experiment.

PARAMETER	CONTROL GROUP		EXPERIMENTAL GROUP	
	INITIAL	FINAL	INITIAL	FINAL
Age (weeks)	5±0.5	17.6±0.54	4.4±0.16	18.4±0.16
Length (cm)	31.6±1.5	62.6±1.67*	29.7±0.57	58.2±0.35*
Weight (g)	910±15.8	3,470±75.8*	924±5.95	2,900±57.7*

Figures are mean ± SD of each group. *: significant difference between groups ($p<0.01$).

the large m. spinalis dorsi and continuous suture on the skin. Radiological control was done and the animal was placed in the postoperative cage. Usually, 15 min later the animal recovered from the anesthesia.

Radiological control

Radiological studies to evaluate spine deformities were performed by means of mobile General Electric X-ray apparatus (AM XII model). Postero-anterior and lateral radiographs were taken from anesthetized animals of the experimental group just before surgery, in the early postoperative period and 3 months after surgery in the experimental group. For control animals similar radiographs were taken except for the postoperative period. X-rays were centered on the spine at a focal distance of 1.10 m, except for the last period in which the focal distance was lowered to 1 m. Intensity varied between 4 mA and 6 mA and the power between 50 KW and 55 KW. High sensibility Agfa-Gevaert films were employed.

Histological Analysis

Sampling was performed 3 months after surgery. First, the animal was anesthetized with 5 mg/Kg i.m. ketamine sulfate followed by 90 mg/Kg sodium pentobarbital in order to leave 10-15 min for the extraction before dying. Once the animal was placed on the surgery table, a 20 cm-long incision was made on its dorsum. Following the spinous process line, first on the right side and then on the left, a subperiostic incision was made to release the muscles. Then samples of approximately 2x0.5 cm of the proximal level of the right side, between TII and TVI were taken. This piece was named R1 and was composed mainly of the large m. spinalis dorsi; this muscle corresponds to a part of the homologous muscle of the human latissimus dorsi, a part of the intrinsic muscles of the dorsum and even a part of the external and internal intercostal muscles. The same was repeated to get the sample at the same level on the left side (L1). The second level, between TVII and TX, was sampled in an analogous way. Muscles corresponding to these samples, R2 and L2, were the same as in the preceding level. Finally, the third level, TXI to LII, was sampled after having enlarged the incision caudally, and releasing subperiostically both sides of the line marked by the spinous processes. Biopsies were taken from the right (R3) and left sides (L3). These samples corresponded to the most distal fibers of the large m. spinalis dorsi, the homologues of human quadratus lumborum and iliolumbaris, the external and internal intercostals, and also part of the paravertebral canal muscles. Every piece was transfixed at both ends by means of a thick 0 silk suture and attached to small wooden tablets to prevent retraction and help synchronization of muscle fiber contraction.

Samples from 10 experimental and 3 control animals were fixed in buffered formalin and processed for paraffin embedding. Sectioning of paraffin blocks was carried out with a Minot microtome at 7 μm and they were stained with hematoxylin and eosin, Masson trichrome and alcian blue-PAS. Pieces corresponding to another 10 experimental and 4 controls were frozen at -175 °C by immersion in the liquid phase of isopentane previously frozen in liquid nitrogen. Once frozen, the pieces were stored at -80 °C until being sectioned in a cryostat at 7 μm in thickness and stained for succinic dehydrogenase (SDH), ATPase and Sudan III. Finally, from 5 experimental animals, with well developed kyphotic and scoliotic curves, and from the remaining 3 control animals, R2 and L2 levels were taken and fixed in 2.5% glutaraldehyde in 0.1M phosphate buffer at pH 7.4 for conventional transmission electron microscopy.

For light microscopy three slices of each technique per piece were examined. Student t-test was applied to the quantitative data after square root and arcsine transformations. Non parametric statistics by means of Spearman correlation were also applied to the different variables.

Results

Radiology and gross examination

At the end of the experiment (3 months after surgery) a conspicuous kyphoscoliosis was found in every experimental animal, the most affected showing an evident gibbus at the level of surgery. The scoliosis was always directed to the right. In 60% of the cases end vertebrae were TVII and TX with TVIII as apical vertebra while in 40% the ends were TVIII and TXI and apical vertebra TIX. The mean of the scoliotic curve (Cobb angle) was 49.6°±8.1 and of kyphosis 61.5°±4.9 (Fig. 1). Most of the cases (80%) of scoliosis were between 41° and 60° and were considered severe while the remaining 20% were between 21° and 40° and considered mild. Kyphosis were severe (61°-70°) in 60% of the experimental animals, mild (56°-60°) in 20% and light (50°-55°) in 20%. In no case control animals showed any valuable radiological sign of scoliosis or kyphosis. Weight and length of experimental animals were reduced around 30% with respect to controls, these decreases being correlated with the degree of the scoliotic and kyphotic deformities.

For experimental animals radiology showed that the curves were structural, that is showing wedge-shaped vertebrae on the concavity, rotation of spinous processes towards the concavity and lateral deviation. Two compensatory curves with inverse inclination to the main one with a lordotic component were developed at upper dorsal and lumbar levels, without rotation or wedging in these cases. Ribs appeared markedly skew on the convexity, showing deformities in their costotransversal joints. Finally, from the macroscopical point of view a conspicuous atrophy, fibrosis and occasional calcifications of the soft tissue were noted on the convexity, mainly at the level of surgery (R2), while on

the concavity fibrosis was mild and affecting paravertebral and intercostal muscles.

Light Microscopy

In every piece, biopsies from experimental animals showed different degrees of histological changes together with areas of normal tissue; no specifical topography being noted. Control animals always showed normal muscle fibers.

Histopathological alterations included fibrosis, chronic inflammation, cicatricial atrophy, necrosis and lipomatosis. These results, including their degree of severity are summarized in table 2. In all cases, alterations were more remarkable on the convexity, especially at R2. In every animal a significant correlation was found between the degree of scoliosis (Cobb angle) and the severity of changes. Fibrosis, varying from light to severe, was present in every one of the experimental animals. Light fibrosis appeared in R3,

Table 2. Summary of histological findings by means of conventional light microscopy for experimental animals.

LEVEL	FIBROSIS	CHRONIC INFLAMMATION	CICATRIC. ATROPHY	NECROSIS	LIPOMATOSIS
R1	-	+	+	++	+
R2	+++	+++	+++	+++	+++
R3	+	++	++	++	+
L1	-	+	+	_	+
L2	++	+	++	+	++
L3	-	+	-	-	-

Severity degree is shown by levels: negative (-); light (+); mild (++); severe (+++).

Fig. 1. Postero-anterior (**A**) and lateral (**B**) radiographs of an experimental animal at the end of the experiment showing scoliosis between TVIII and TXI levels (A) and kyphosis (B). Cuneal vertebrae and rotation of spinous processes to the concavity can be observed together with lateral bending. Two compensatory curves (arrows) of inverse inclination at upper dorsal and lumbar levels have developed without rotation or wedging. L: left side; R: right side. x 0.4

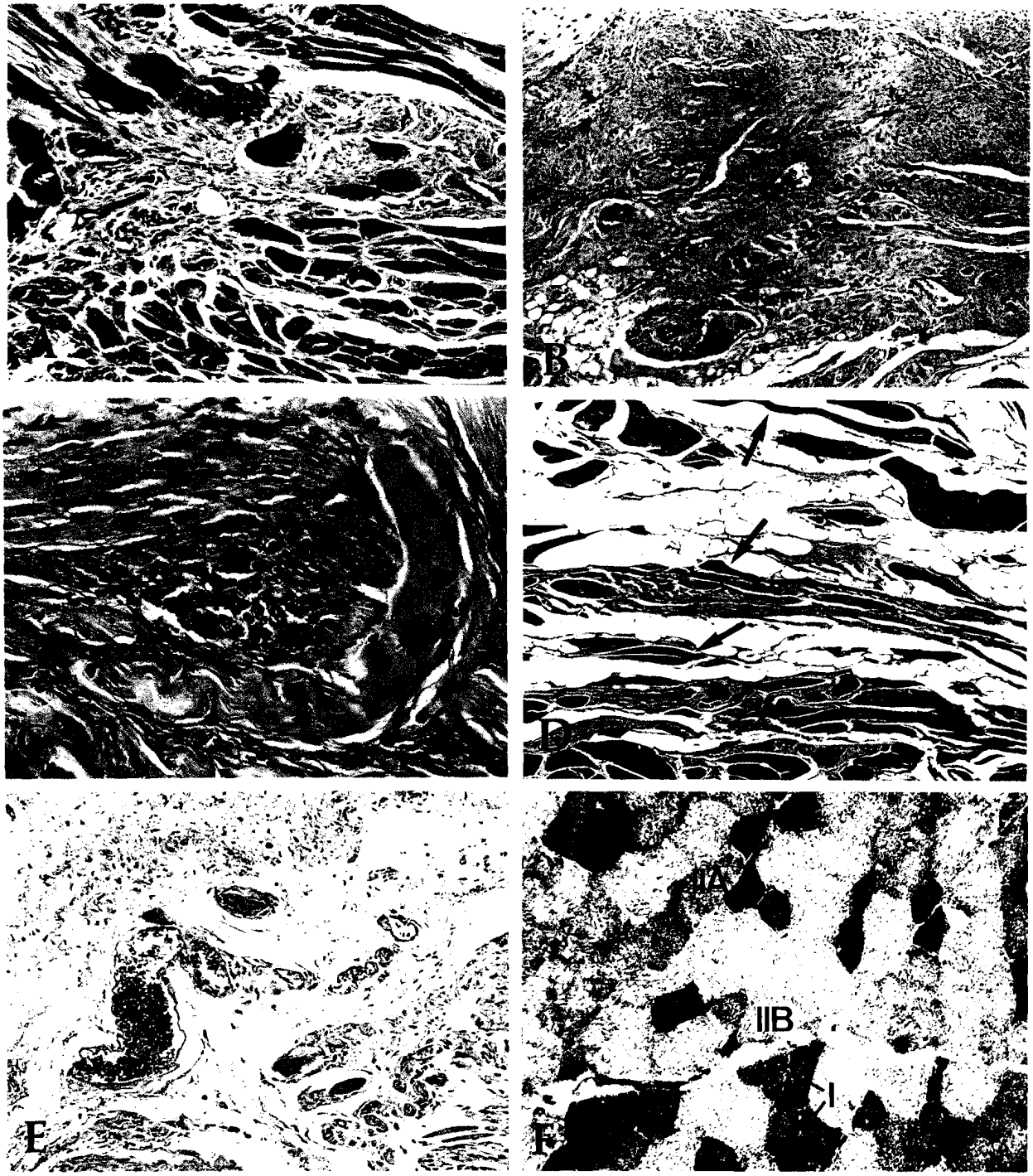

Fig. 2. Light microscopic alterations in the paravertebral muscles of the experimental animals. **A.** Mild fibrosis at level L2. Masson trichrome. x 130. **B.** Severe fibrosis and whorl-like arrangements of muscle fibers. Hematoxylin and eosin. x 50. **C.** Intense chronic inflammatory infiltrate at R2. Hematoxylin and eosin. x 250. **D.** Atrophic muscle fibers (arrows) among adipose infiltrates (lipomatosis) at R2 level. Masson trichrome. x 80. **E.** Severe necrosis at level R2. Hematoxylin and eosin. x 80. **F.** Clear predominance of fast-twitch fibers (IIA and IIB) as well as a tendency of slow-twitch fiber (I) to cluster are signs of type I fiber selective atrophy in level R2. SDH histochemistry. x 125

while mild fibrosis was found at L2 (Fig. 2A). Severe fibrosis was detected exclusively in R2 (Fig. 2B), with muscle fibers appearing in a whorl-like arrangement. A moderate chronic inflammatory infiltrate was also observed in everyone of the experimental animals, more conspicuously on the convexity followed by R3 (Fig. 2C). Meanwhile, the left side showed light inflammation. Occasionally an eosinophil infiltrate was also observed. In every experimental animal atrophy of muscle fibers was observed at some level of both sides of the scoliotic curve, R2 being more severely affected (Fig. 2D) followed by L2 and R3. A mild degree of necrosis was observed in 100% of the animals at some level, while severe signs of necrosis were found at R2 (Fig. 2E). Lipomatosis was also present to some extent in everyone of the experimental animals. The degree and extension of the adipose infiltrate was variable, R2 being most affected (Fig. 2D) and L2 to a lesser degree. Some muscle fibers showed lipid droplets included in their cytoplasms reaching occasionally the degree of fatty change.

Signs of denervation were also observed, but in this case only in 40% of the experimental animals and exclusively at the level where surgery was performed (R2). Hypertrophic muscle fibers were seldom present, without any systematic or regular distribution. Other features observed with light microscopy were loss of striation, vacuolation, central nuclei and macrophage activation.

According to the average count, both on ATPase and SDH techniques, the distribution and proportion of fiber types by levels for control and experimental animals are shown in table 3. Slow-twitch fibers (type I) were less frequently observed than fast-twitch ones (type II) along all the levels (Fig. 2F). The proportion of type I fibers decreased from proximal to distal in control animals, but in experimental animals this rule was broken at L3, level in which the proportion of type I fibers was highest.

A significant decrease in the ratio of type I/type II fibers was observed in the experimental group at every level except for R3 and L3. In R3 a decrease was still observed but was not statistically significant ($p<0.1$), whereas in L3 the situation was reverse since the proportion of slow-twitch fibers increased. When comparing the proportion of slow-twitch fibers on each side -including all levels- with the correspondent side in control animals, a global decrease ($p<0.001$) of the slow-twitch type could be noted both on the right (30.6±1.2% vs. 35.3±0.2%) and on the left side (29.3±4.9% vs. 33.6±7.6%). Besides, slow-twitch fibers appeared more frequently in clusters (Fig. 2F).

Table 3. Mean percentage ± SD of slow-twitch (type I) and fast-twitch (type II) fibers at each level in both control and experimental animals.

LEVEL	CONTROL GROUP		EXPERIMENTAL GROUP	
	Slow-twitch	Fast-twitch	Slow-twitch	Fast-twitch
R1*	44.7±0.1	55.2±0.8	38.8±4.6	61.1±4.6
R2*	36.3±0.5	63.6±0.5	30.1±1.9	69.9±1.9
R3	24.7±1.3	75.2±1.3	23.1±3.6	76.8±3.6
L1*	42.2±0.1	57.7±0.1	34.6±3.4	65.3±3.4
L2*	31.3±0.04	68.6±0.04	28.7±4.1	71.2±4.1
L3*	27.4±2.16	72.5±2.16	44.7±1.2	55.2±1.2

*: significant differences between both groups ($p<0.001$). The ratio between slow- and fast-twitch fibers decreases significantly at most of the levels (R1, R2, L1 and L2), while this decrease is also observed at R3, but is not statistically significant in this latter case. Contrastingly at L3 an increase of the mentioned ratio (type I/type II) was obtained.

Electron microscopy

Ultrastructurally, biopsies in experimental animals also showed variation from normal to considerably affected striate muscle, whereas control animals appeared consistently normal. Therefore, all the following descriptions refer to the experimental animals.

As already mentioned regarding light microscopic data, normal fibers could be seen close to alterated ones both on the concavity and on the convexity. Fibers at different degrees of atrophy as well as different degrees of cytolysis were seen. Central nuclei were also seen.

Malalignments of myofibrills appeared breaking the characteristic transverse striation, with Z-lines arranged in a stairway manner (Fig. 3A). Myofibrillar threading was also seen (Fig. 3B). A-bands lost their homogeneous density, appearing as irregular dense patches without a precise limit between I-bands. Some myofibrills showed dehiscences or even bifurcations. In muscle fibers with a more severe degree of disorganization, the microfilaments of some myofibrills were not arranged in the characteristic transverse striation.

Sarcoplasmic reticulum showed a generalized dilation, more intense in the terminal cisternae, losing its topographical relationships with sarcomeres (Fig. 4A). Finally, some cells showed a complete disorganization, where sarcomeres were no longer distinguishable and myofibrills disappeared. Dense and myelinic bodies appeared together with loss of organelles and nuclear features of necrosis (Fig. 4B). Cells in different stages of degeneration up to complete cytolysis, as well as phagocytosis by macrophages and substitution of muscle fibers by connective tissue could be seen.

Discussion

Our method for experimental scoliosis, consisting of costotransversolisis with transversectomy and the releasing of paravertebral muscles caused kyphoscoliotic curves in 100% of the cases. The resulting curves showed an S-shape with wedging and vertebral rotation, i.e., they are structural and with similar characteristics to those of human idiopathic scoliosis (Miles, 1947; Langeskiöld and Michelsson, 1962; Karaharju, 1967), thus validating our surgical method.

In all cases the curves were dextroconvex, i.e. to the same side of surgery, in agreement with that obtained with analogous techniques (Karaharju, 1967;

Fig. 3. Micrographs of paravertebral muscles in the experimental group. **A.** L2 level showing a stairway arrangement of Z-lines denotes malalignments of myofibrills x 11,000). **B.** R2 level showing thread-like arrangements of myofibrills (arrows). Sarcomeres are hard to identify and transverse striation has been lost. x 15,000

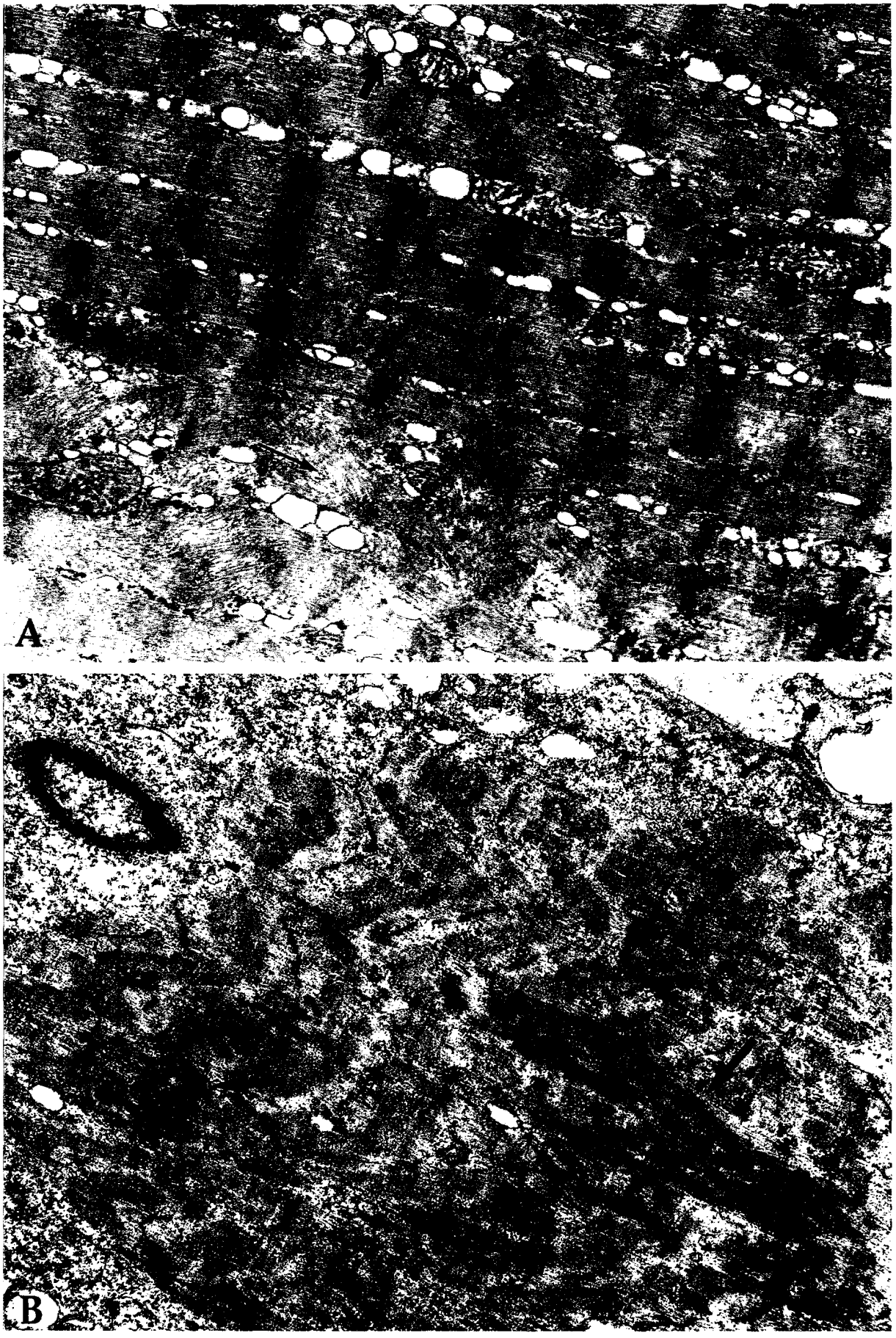

Fig. 4. Micrographs of level R2 of paravertebral muscles in the experimental group. **A.** I-bands are not distinguishable and myofibrills tend to derange. Some myofilaments appear loose (thin arrows) and sarcoplasmic reticulum shows a generalized dilation (bold arrows). x 13,500. **B.** Complete disorganization of the cell contractile apparatus and signs of necrosis. Myofilaments form dense aggregations (arrows). x 10,000

Langeskiöld, 1971; Beguiristáin, 1974; Sevastikoglou et al., 1978). However, Cañadell (1974), using a different approach consisting in unilateral periosteum release of one paravertebral canal, obtained the convexity in the contralateral side.

No significant differences between sexes were found in the experimental group, although in humans idiopathic scoliosis is more frequent in girls (Shands and Eisberg, 1955; Wynne-Davies, 1968) and a sex-linked inheritance has been implied (Cowell et al., 1972).

The experimental animals showed a reduction of weight and length in correlation with the degree of the scoliotic and kyphotic deformity. Although Stilwell (1962) noted a direct correlation between kyphosis and scoliosis, the correlation between the degree of kyphosis and loss of weight and length had not been previously reported. Thus, the formation of the compensatory curves lets the animal hold its postural equilibrium, but with negative repercussions in its growth.

Chronic inflammation produced on both sides appeared more intense in the convexity in contrast with the results obtained by other authors, who found more intense inflammation on the concavity (Tresserra, 1969; McEwen, 1973). These differences could be explained because in our experiment the lesions in the convexity are caused not only by the spine deformity but also by the muscular release, transversectomy and costotransversolisis. The eosinophil infiltrate found in some cases on our material could be interpreted as reaction to the suture material.

Atrophy, necrosis and fibrosis were also found on both sides, with the convexity being more affected. Similar descriptions have already been reported regarding human idiopathic scoliosis in classical studies (Tillmans, 1874; Virchow, 1911) as well as in previous experimental studies in animals (Stilwell, 1962; Cañadell, 1974). Contrastingly, no necrosis is referred to by Spencer and Zorab (1976). These differences could be explained by the different method used, due to the authors' utilization of the plaster cast technique (Langeskiöld, 1971) instead of our surgical approach.

Likewise, lipomatosis appeared to a greater degree on the convexity, but the concavity was also affected. Reports on human scoliosis have described lipomatosis but the location of the greatest degree of severity varies with authors (Yarom et al., 1981; Low et al., 1983; Von Lesser, 1988). The proliferation of adipose tissue could stop fibrosis to some extent and in this way reduce the evolution of deformities. Regarding lipid accumulation inside muscle cells, we have only occasionally found it , on both sides of the curve without a clear prevalence; however, Stilwell (1962) found in monkeys more prevalence on the convexity with signs of fatty change. In human idiopathic scoliosis, Sahgal et al. (1983) reported subsarcolemmal accumulation of lipids besides glycogen and mitochondria, but this intracellular lipid accumulation seems to be of a different kind and we consider the lipid droplets inside muscle cells appearing in our material as an occasional finding taking place in degenerating cells. The loss of muscle cells subsequent to necrosis is therefore replaced mainly by fibrous tissue and also by fat infiltrate. This connective tissue also plays an important role in the structuration of the curves.

An interesting and controversial point is the distribution and ratio of fiber types. In our study a decrease in slow-twitch fibers was found in experimental animals as compared with control animals at all levels except for R3, which was not significant and for L3 where slow-twitch fibers were increased. Thus, a selective atrophy of type I fibers took place at all levels, especially at R2, while type II atrophy was rare and unspecific except for L3 in which it appears evident.

Spencer and Zorab (1976), provoking scoliosis in rabbits by plaster cast technique (Langeskiöld, 1971), found a decrease in slow-twitch fibers at levels 1 and 2, but these were not statistically significant. Since in human idiopathic scoliosis a decrease in slow-twitch fibers has been established (Spencer and Eccles, 1976; Yarom and Robin, 1979; Ford et al., 1984; Hsu et al., 1988), our results yield statistical significance to the mentioned decrease in slow-twitch fibers in levels 1 and 2 and therefore our method appears more suitable to experimentally mimic human idiopathic scoliosis. Nonetheless, in the lumbar level, our results clearly contrast with those of Spencer and Zorab (1976) since they found a significant decrease in slow-twitch fibers in this region and we found an increase in the proportion of this type of fibers at L3. These differences could again be explained in terms of the different experimental approach.

There are atrophy and necrosis of both type I and Type II fibers at every level in our experimental animals, but this loss of fibers is more important for slow-twitch fibers at the level of the curve, whereas it affects fast-twitch fibers more severely distally. Slow-twitch fibers are considered to be more sensitive to trauma, while type II atrophy is more dependent on disuse (Dubowitz and Brooke, 1973). Our study seems to confirm this belief since slow-twitch fibers decrease more at the levels of the main scoliotic curve and the reverse situation which appears at L3 may be a consecuence of the compensatory processes taking place distally, including adaptation to new tensions required in the lumbar region.

Our ultrastructural findings are comparable to some extent with those of Sahgal et al. (1983), Fitzsimons and Tyer (1980) and Khosla et al. (1980) in human idiopathic scoliosis. However, in contrast to Khosla et al. (1980) we have not found the myotendinous disarrangement postulated by them as a primary defect in idiopathic scoliosis. Since in our experimental model we cause a lesion consisting of muscle release, it could be comparable to an abrupt and total failure of the myotendinous junction. Thus, our experimental method mimics one of the proposed etiopathogenic mechanisms of idiopathic scoliosis, supporting the reliability of this model for pathophysiological studies and for testing different treatments.

Peterson et al. (1995) did not find apparent

differences in the outcome between patients who were given electrical stimulation and those patients who were not, thus diminishing the support for muscle imbalance as a cause of idiopathic scoliosis. Nevertheless, muscle constitutes a dynamic factor adapting to changing requirements, as the modifications in the ratio of fiber types demonstrate. This fact must always be taken into account when a therapeutic method is chosen for the treatment of scoliosis.

In conclusion, after our surgical production of scoliosis a degenerative process takes place in paravertebral muscles, which is initiated by a progressive derangement of the contractile apparatus of the muscle cell, disintegration and eventual necrosis. The role that paravertebral muscles play on the etiology and pathophysiology of human idiopathic scoliosis requires further investigation but our results suggest an overt participation of paravertebral muscles in the establishment of compensatory processes.

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