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Morphology and Histochemistry of Myogelosis

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Myogelosis is a common diagnosis in the case of chronic pain conditions, especially in the region of the pectoral girdle musculature, the glutei muscles, and the *erector spinae* muscle. Although such indurative areas continue to be palpable even on the cadaver, few studies concerning the morphological substrate of these areas have been undertaken. Selected biopsies as well as larger tissue samples were taken from 11 corpses and prepared for histological study. Following staining, the frozen sections were examined morphometrically. A histologically constant, significant morphological alteration was found in the areas of concern. The spaces between the individual muscle fibers of healthy muscle tissue appear relatively wide, the endomysium of the myogelotic area are clearly narrowed. Split fibers, ragged red fibers, Type II fiber atrophy, and fibers with a moth-eaten appearance have been detected. The morphometry shows considerable increase in thickness of the affected muscle fibers, suggestive of a pathological, local hypertrophy. The changes described may well represent a fixed condition, so that it should not be surprising that myogelosis therapy is difficult and protracted. Clin. Anat. 12:266–271, 1999.
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INTRODUCTION

Myogelosis is a painful change in muscle structure, used as a term in the German literature since the 1920s. In English literature (Travell and Simons, 1992), myogelosis is correlated with the myofascial triggerpoints and this term has appeared since the 1940s. Other terms e.g., "fibrositissyndrome" or "muscle indurance," have also been used.

In clinical practice, muscle areas that are painful on pressure and are strikingly resistant to therapy are very common. These myogelotic changes are even palpable postmortem. However, little research has been done on the morphology of these changes (Hackett, 1958; Bischko, 1978; Schröder, 1982; Lewit et al., 1987; Yunus and Kalyan-Raman, 1989; Eder and Tilscher, 1990; Jerusalem and Zierz, 1991; Travel and Simons, 1992; Drewes et al., 1993). Several treatment options are available, including manual therapy, injections, dry needling, and electrotherapeutic modalities.

The objective of the present study is the morphological analysis of biopsy material taken postmortem from mygelotic muscle areas.

MATERIALS AND METHODS

Based on palpatory findings, tissue samples were taken from myogelotic areas of 11 unfixed human cadavers (6 female, 5 male). In addition to the biopsies from the pathologically altered muscle areas, tissue samples from nonindurative areas of the muscle in question were taken for comparative purposes.

The average age of the corpses was 68.82 years (female: 67.5 a; male: 70.4 a). The tissue samples were taken within a time period of 4–48 hours following

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TABLE 1. Sex, Age, and Resection Period Postmortem of the 11 Corpses

Corpse No.	Sex	Age a	Resection period postmortem hr
1	male	91	24
2	male	70	24
3	female	70	48
4	female	72	24
5	female	80	48
6	female	54	24
7	male	50	24
8	female	69	15
9	male	66	9
10	male	75	18
11	female	60	4

death (see Table 1). A total of 102 biopsies were examined, with most of the samples from *trapezius* or of the *gluteus medius* muscle. In one case, myogelotic material was taken from the *gluteus maximus* muscle.

The tissue specimens were divided after sampling and shock-frozen at −80°C in melting Isopentane, or fixed in a 4% solution of formaldehyde and embedded in paraffin. Subsequently, frozen sections and paraffin sections were made. In addition, semithin microsections were prepared after Epon^{R}-embedding.

The following staining procedures were carried out on the frozen sections in accordance with the standard neuropathological techniques for muscle diagnostics (Jerusalem and Zierz, 1991): ATPases pH 4.2, 4.6 and 9.4; NADH-TR; GOMORI-tricromatic; Oil red O. The semithin microsections were stained *Azurmethylene-L Blue*. The following staining procedures were carried out on the paraffin sections: H & E; Van Gieson; PAS; MASSON trichrome.

Subsequent to histological preparation, the specimens were examined under a light microscope and subjected to computer-aided morphometry (Lucia M).

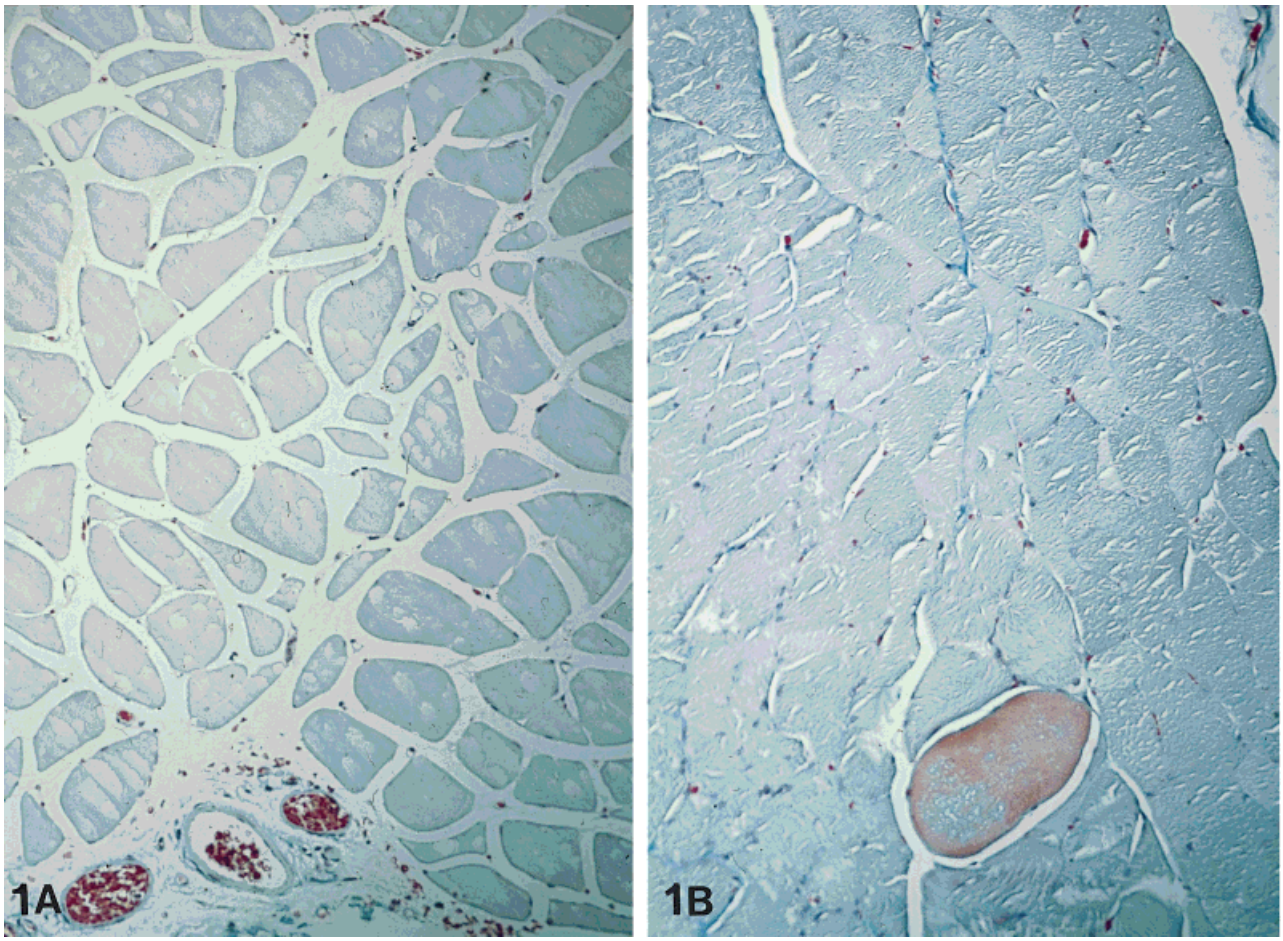

Fig. 1. A. Unchanged musculature (broad endomysial spaces). Paraffin section, trichromatic staining according to MASSON, ×225. **B.** Myogelosis, narrow endomysium. Hypertrophic fibers (orange-red). Paraffin section, trichromatic staining according to MASSON, ×225.

RESULTS

Myogelotic foci appear significantly in certain areas of the musculature of the trunk. In the upper half of the body, we found and examined these changes, particularly in the *trapezius* muscle. In the lower half of the body, we concentrated on the *gluteus medius* muscle.

The mygelotic areas are commonly palpable postmortem, which was one of the most surprising macroscopic findings. On the basis of these palpatory findings, it was subsequently possible to take selected muscle biopsies as well as control samples. The most striking finding of the histological preparation is a distinctly wider endomysium in the control group in comparison to the myogelotic areas (see Fig. 1A, B, trichromatization according to MASSON).

In addition to the narrowing of the endomysial space to its almost complete disappearance, enlarged, rounded muscle fibers were noted repeatedly (Fig. 1B). Their intense red-orange staining (Acid-Fuchsin component of GOLDNER-Trichrome staining) indicates an increased number of mitochondria.

This finding is also apparent in the frozen sections (unfixed material). The occurrence of shrinking after formaldehyde fixation can, therefore, be ruled out. These sections also show distinct endomysial clefts in "normal" muscle tissue (Fig. 2A), which disappears in the myogelotic areas (Fig. 2B). A conspicuous vacuolization was also noted in the enlarged and rounded muscle fibers.

At the same time, degenerative processes were ascertained in the hypertrophied fibers, such as localized coarse fiber solidification. Irregular textural disturbances (ragged red fibers) and the sector specific-loss of myofibrils (see Fig. 1B). Fiber-splitting occasionally

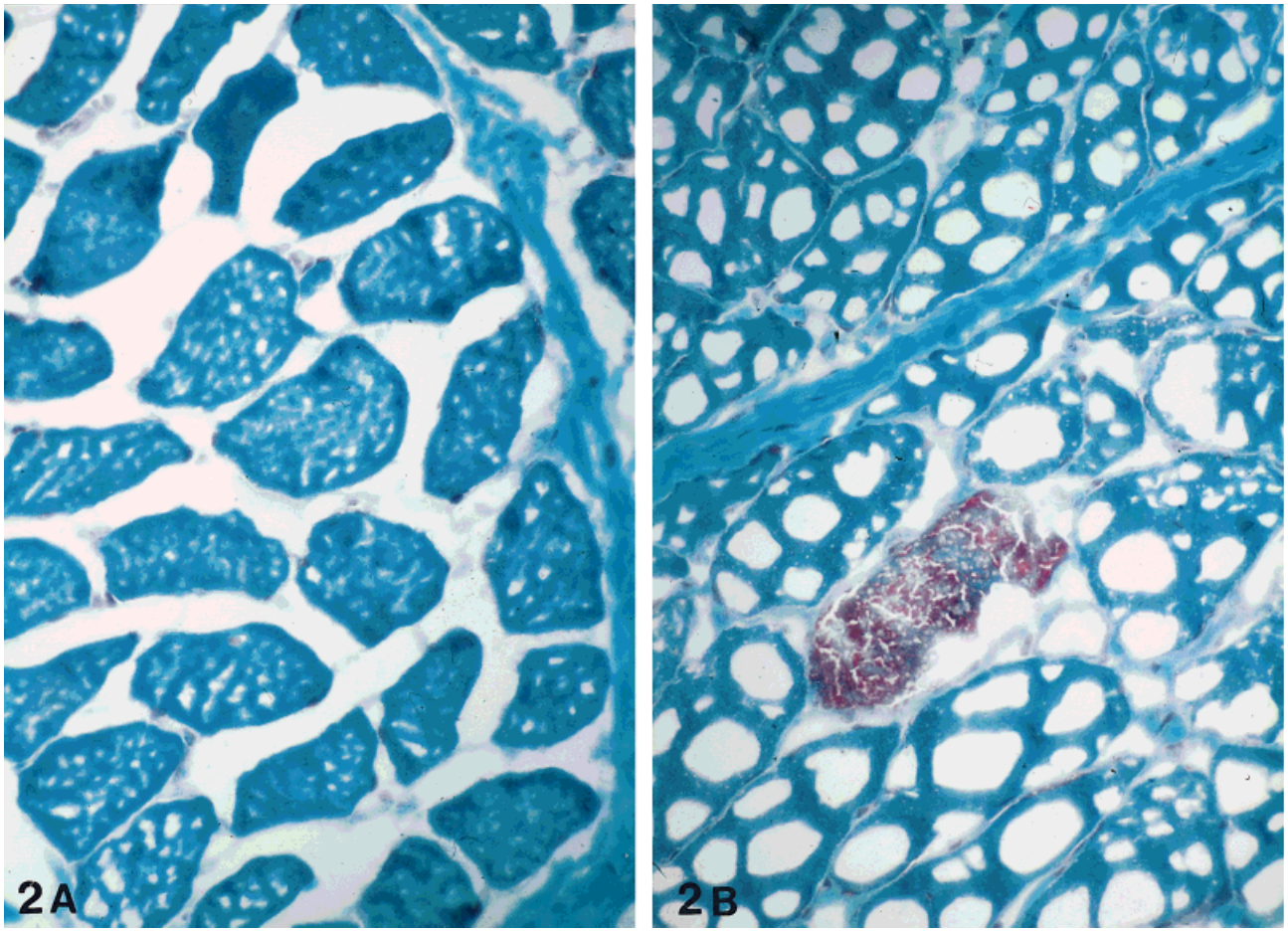

Fig. 2. **A.** Unchanged musculature, frozen section, trichromatic staining according to GOMORI, ×450. **B.** Myogelosis, vacuoles in the fibers, ragged red fibers (blue-violet). Frozen section, trichromatic staining according to GOMORI; ×450.

can be observed in greatly enlarged muscle fibers (see Fig. 5).

In comparison to unchanged muscle tissue (see Fig. 3A), mygelotic tissue samples in the ATPase-stainings reveal a selective hypertrophy of Type I fibers (Fig. 3B). In contrast, Type II fibers appear to be atrophically changed (Fig. 4A, B). Strongly hypertrophied Type I fibers commonly appear as fibers with a moth-eaten appearance (Fig. 3B, arrow).

The morphometrical preparation of the biopsies was carried out on the semithin microsections with the use of computer-aided morphometry (LUCIA M). A significant increase of the diameter in the transverse section of the fibers in myogelotic areas, from 60 μm (mean) to >100 μm (mean) could be determined (Fig. 1A, B).

DISCUSSION

Localized muscular areas that are pain-sensitive to pressure are often encountered in clinical practice. This corresponds to our observations on corpses. The few studies concerning myogelosis, including muscular trigger points published to date, describe biopsy material sampled in vivo (Hackett, 1958; Bischko, 1978; Schröder, 1982; Lewit et al., 1987; Yunus and Kalyan-Raman, 1989; Eder and Tilscher, 1990; Jerusalem and Zierz, 1991; Travell and Simons, 1992). The present study is the first to examine cadaver material. Of particular importance is the fact that myogelotic areas are palpable on the corpse. This contradicts the published view that such changes are functionally reversible (Bengtsson et al., 1986; Fassbender and

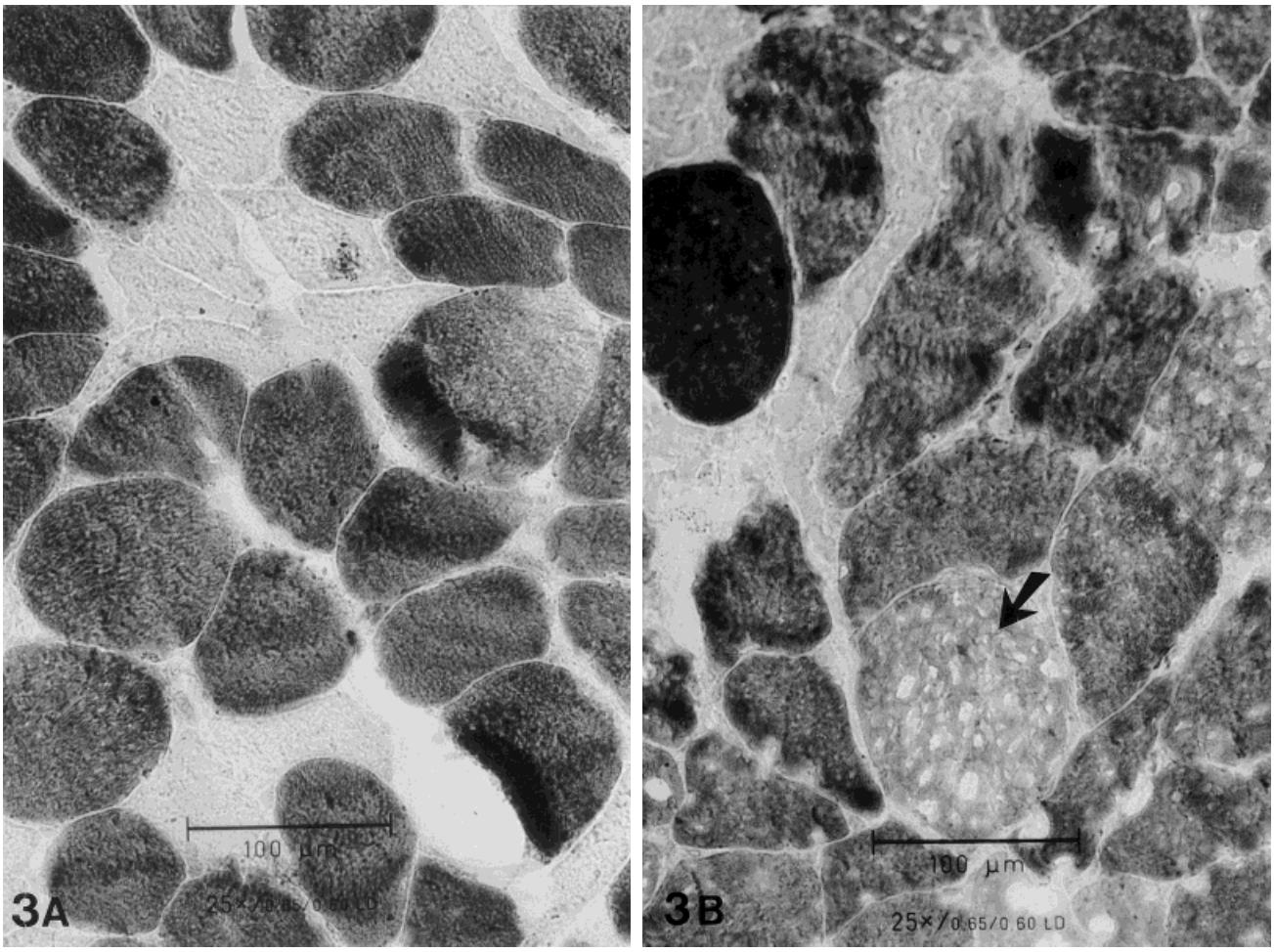

Fig. 3. **A.** Unchanged musculature, Type I fibers dark, frozen section, ATPase pH 4.3; ×250. **B.** Myogelosis, Type I hypertrophy, fibers with motheaten appearance (arrow). Frozen section, ATPase pH 4.3; ×250.

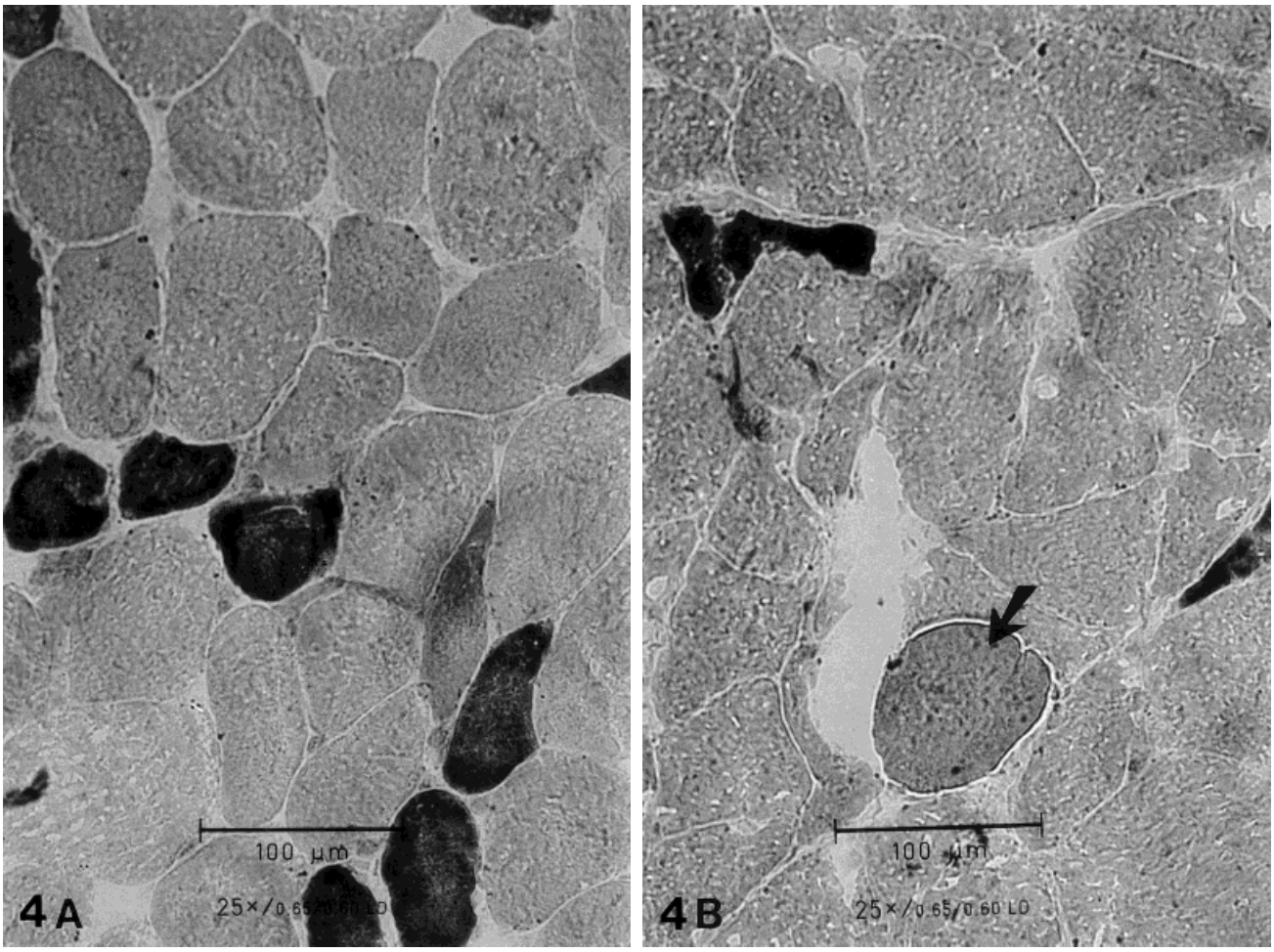

Fig. 4. **A.** Unchanged musculature, Type II fibers dark. Frozen section, ATPase pH 10.4; ×250. **B.** Myogelosis, Type II fibers changed degeneratively and atrophic (arrow). Frozen section, ATPase pH 10.4; ×250.

Martens, 1992; Simons 1993, 1994, 1997). A neuromuscular dysfunction theory has been discussed (Simons, 1991).

The typical localization of these myogelotically changed muscle areas is in the dorsal muscles of the pectoral girdle (the *trapezius*, *rhomboidei*, *levator scapulae* muscles) as well as the *gluteus medius et maximus*, *piriformis* muscles and the *erector spinae* muscle.

In our view, this implicates statically overstrained structures that are related to the erect posture. The selective hypertrophy of the slow contracting Type I fibers supports this view. The fact that myogelotic areas are palpable on the corpse leads to the conclusion that the condition is a fixed one and that it represents the terminal stage of a possibly progressive development.

This distress of the muscles leads to local-partial muscle fiber hypertrophy in circumscribed areas. As a result of the significant increase of the diameter in the transverse section of the muscle fibers, a histologically distinct and visible narrowing of the endomysial space occurs, which brings about impairment of the trophicity of the affected area. This is particularly true of the hypertrophied fibers, which show a tendency towards so-called fiber-splitting, moth-eaten necrosis, textural disturbances, and to the formation of ragged-red-fibers (Figs. 1B, 3B, 4B).

A further visible indication of the altered intracellular environment of the myogelotic areas is apparent in the frozen sections (Fig. 2A, B). In contrast to the control group (Fig. 2A), the muscle fibers in the pathologically changed areas (Fig. 2B) reveal pronounced vacuolization.

In conclusion, morphological analysis of human postmortem biopsy material discussed in the present

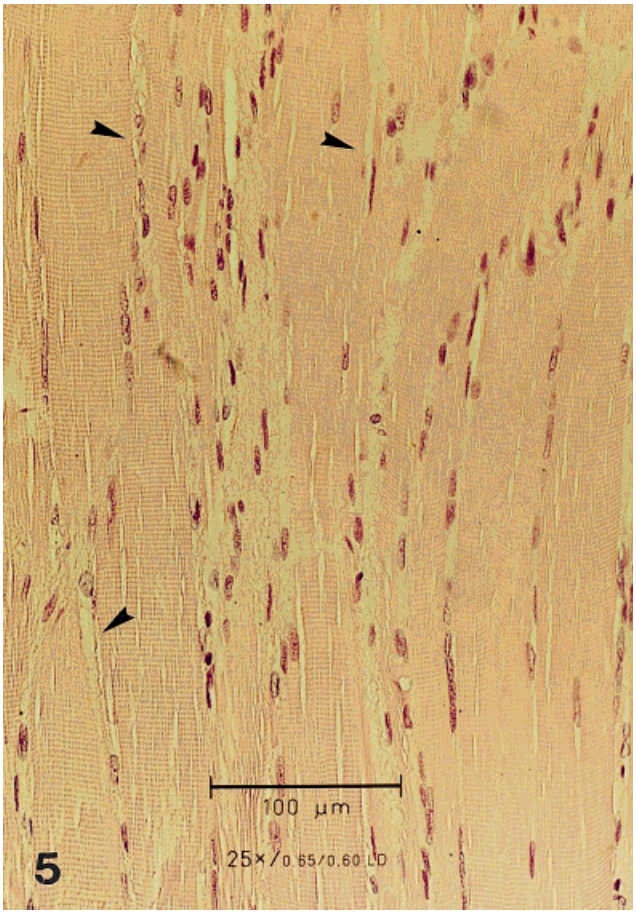

Fig. 5. Split fibers in hypertrophic muscle fibers (arrows), H & E; ×250.

study shows a constant substrate in the sense of a local-partial Type I hypertrophy connected with a distinct narrowing of the endomysial space. In our view, the fact that these changes remain palpable even after death suggests a fixed, irreversible condition, which would explain the resistance to therapy. This finding makes it necessary to carry out further studies.

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