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Fine Structural Study of Human Skeletal Muscle Injuries Due to Blunt Trauma

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Summary. Fine structure changes of human skeletal muscle were studied by mechanical injury. In the zone of the primary point of impact complete disruption of the muscle cells could be noticed. The degree of disorganization of the contractile elements was so extreme that the I and A bands and the H zone were homogenized, and only the Z line could be identified. In the zone around the point of impact, muscle fibers with ruptured filaments, disintegration of sarcomeres, and hypercontration were found in the early period. About 2 hours after the injury the myofilamental alterations reached a peak, and no progression of the damage could be demonstrated later. In the late period the alterations of mitochondria, sarcoplasmic reticulum, and cell nuclei were dominant versus the myofibrillar changes. In the macroscopically uninjured region of the muscle, intact muscle fibers as well as muscle fibers with hypercontraction, ruptured filaments, zig-zag formed Z lines were found. In the author's opinion the myofilamental changes were caused by the direct traumatic effect and/or by the hypercontraction. Damage of the other cell structures developed only later, based on other mechanisms (e.g., hypoxia, circulatory demages, hemorrhage).

Key words: Muscle injury – Fine structural changes.

Zusammenfassung. Bei 40 Patienten wurden die durch mechanisches Trauma bewirkten feinstrukturellen Skelettmuskelveränderungen analysiert. In der Zone des primären Anpralls wird 1 Stunde nach der Verletzung die Desorganisation der Myofilamente nachweisbar, aber es ist noch keine Schädigung anderer Zellbestandteile zu beobachten. An der Durchtrennungsstelle vom Muskelgewebe sind I- und A-Bänder sowie die H-Zone homogenisiert und nur die Z-Linie ist noch zu identifizieren. In der Randzone der Verletzung wird anfangs eine Desintegration der Sarkomere und eine starke Kontraktion festgestellt. Zwei Stunden nach der Verletzung erreicht die Myofilamentschädigung ihren Höhepunkt, ohne daß später eine Verschlimmerung nachgewiesen werden konnte. Die Schädigung der Mitochondrien dagegen und des sarkoplasmatischen Retikulums ist frühestens 2 Stunden nach der Verletzung nachweisbar und wird dann noch stärker. In der makroskopisch unauffälligen Randzone der Verletzung werden sowohl unveränderte, als auch stark kontrahierte Muskelfasern, zerrissene Filamente und zick-

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zackförmige Z-Linien gefunden. Die Myofilamentveränderungen werden als Folge des Traumas und der hierdurch ausgelösten Überkontraktion betrachtet, die Mitochondrien- und sarkoplasmatischen Retikulum-Veränderungen als Folgen anderer Faktoren (z. B. Ischämie, Blutung, Hypoxie) angesehen.

Schlüsselwörter: Skelettmuskelverletzungen – Feinstrukturänderungen.

Examination of skeletal muscle injuries is a fairly neglected field both in pathology and clinical medicine. Altough a lesion or destruction of some muscular area might cause – due to its mass – no major functional loss, the study of the traumatic change of the tissue mainly subject to injury should be considered important. Therefore, a study of skeletal muscle injuries due to blunt trauma have been carried out since their occurrence alone or in association with bone and visceral injuries are most common (compression, conquassation), and also since their consequences are least known. Fine structural changes of the human skeletal muscle caused by mechanical injury are mostly unknown.

Material and Methods

The studies were carried out from injured 40 patients (31 male, 9 female). All of them had been subject to traffic accidents, with a primary point of impact on the lower extremity, or on thoraco-

Fig. 1. Complete disruption of the contractile elements, only the Z line could be identified. Mitochondria were not alterated. M. gastrocnemius. 1 h after injury, point of impact. 5800 x

Fig. 2. Hypercontraction and relaxation of sarcomeres, disrupted myofilaments. Streaming and zik-zak formation of Z lines. Intracellular edema. M. tibialis anterior. 1 h after injury. 3 cm to point of impact. 4300 x

abdominal region. The patients aged 18 to 42 years. The muscle samples were excided by surgical intervention 1 to 22 h after the injury. Muscle samples have been removed by biopsy from three zones:

1. Primary point of impact,
2. Zone around of a point of impact (1 to 3 cm),
3. Macroscopically intact muscles near the injury.

The tissue samples were fixed in Millonig's osmium tetroxide fixative for 2 h. After grading alcohol dehydration the samples were embedded in Durcupan^{R}. Semi and ultrathin sections were cut with Reichert OM U2 ultramicrotome, contrasted with uranyle acetate and lead citrate, and photogrphed with Tesla Bs 242 electron microscope.

Studied Muscles were: M. gastrocnemius (3 cases) m. tibialis anterior and m. peroneus longus (4-4 cases), m. quadriceps femoris (5 cases), m. rectus abdominis and m. transversus abdominis (5-5 cases), m. intercostalis externus and internus (4-4 cases), and other muscles (6 cases).

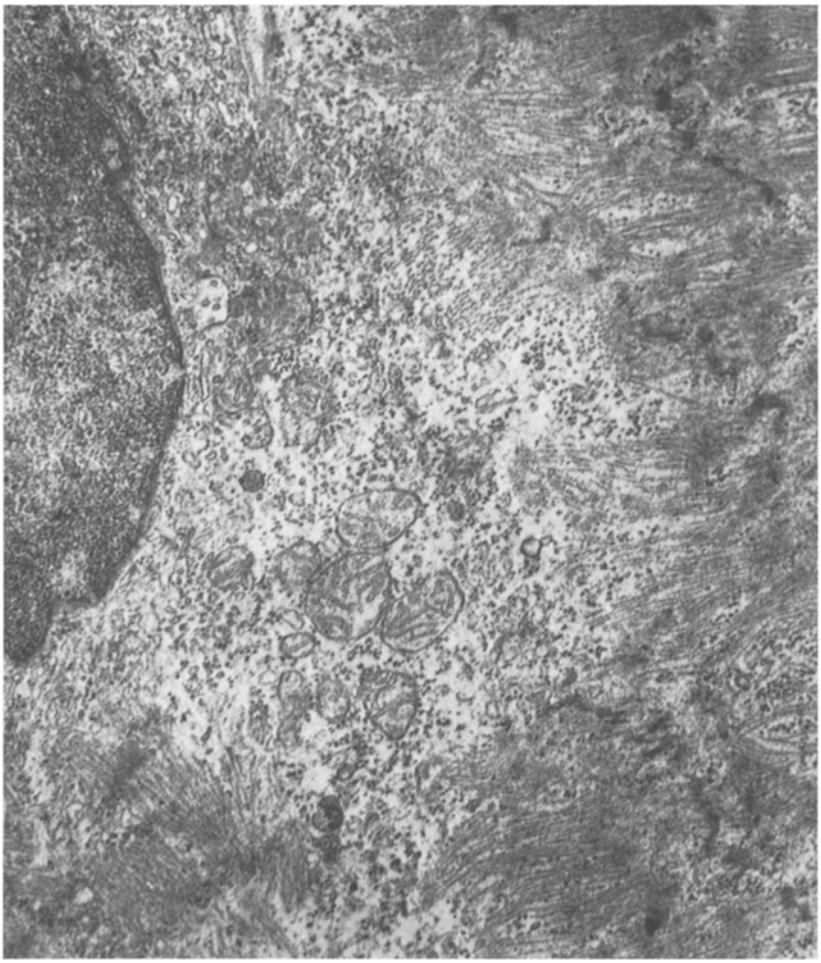

Fig. 3. Hypercontraction, zik-zak formation of Z lines, and disruption of myofibrils. No other cell organelles (mitochondria, glycogen, sarcotubular system, nucleus) showed any changes. M. rectus abdominis. 2 h after injury. 2.5 cm to point of impact. 4300 x

Results

In the zone of the primary point of impact could be seen the complete disruption of the contractile elements. The degree of disorganisation was so extreme, that the I bands and A bands, and the H zone were homogenized, and only the Z line could be identified (Fig. 1). The plasma membrane demonstrated an irregular, undulated configuration, and at a number of sites it appeared fragmented. After 22 hours in this zone complete necrosis and homogenization of the muscle cells could be observed.

One hour after injury, in the neighborhood of the point of impact, muscle fibers with ruptured filaments, disintegration of sarcomeres and interfibrillary edema have been found. Two hours after injury, extensive rupture of myofilaments could be

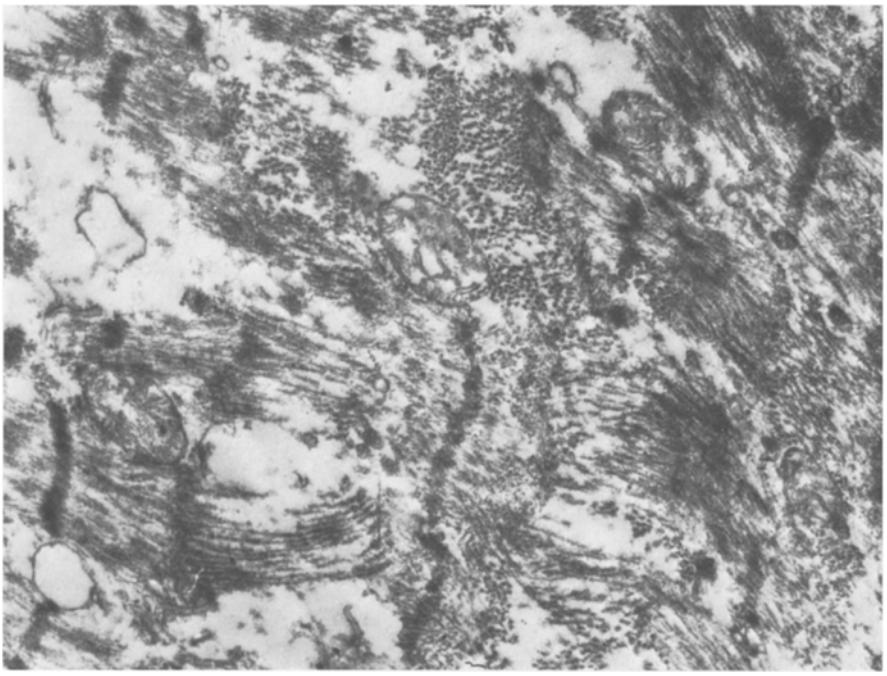

Fig. 4. Disorganisation and necrosis of contractile elements. Swelling of mitochondria, destruction, and desorganization of the inner membranes. M. rectus abdominis. 19 h after injury. Cca. 1 cm to point of impact. 5800 x

seen. These sites of fracture were usually lay near to or at Z lines. In some sarcomeres hypercontraction, in the others relaxation was found. The streaming and zik-zak formation of Z lines was commonly. At that time, no other cell organelles (mitochondria, sarcotubular system, cell membrane, etc.) showed any changes (Figs. 2 and 3). The myofibrillar changes are caused by the direct traumatic effect (Fig. 1), and/or by the consecutive hypercontraction (Figs. 2 and 3). They appear right after or shortly after the injury. Damage of the other muscular structures developed only later. About 2 h after the injury, the myofibrillar alterations reached a peak and no progression of the damage could be demonstrated later. Three hours or later we noticed swelling of mitochondria and dilatation of sarcotubular systems. The mitochondrial cristae began to manifest early signs of disintegration, seeming to be more susceptible to injury than the outer mitochondrial membrane. Matrix granules were not seen in these injured mitochondria. More than 12 h after the injury, beginning cell necrosis was recognizable also in these zones. In this period the alterations of mitochondria and of sarcoplasmic reticulum are dominant versus the myofibrillar changes. Here, too, the above-mentioned, myofibrillar changes are present. However, they are not aggravated as compared to those seen 2 to 3 h after crush injury. Enlargement of mitochondria, complete destruction, and desorgnisation of the inner membranes could be observed (Fig. 4). The limiting membranes of some mitochondria have been

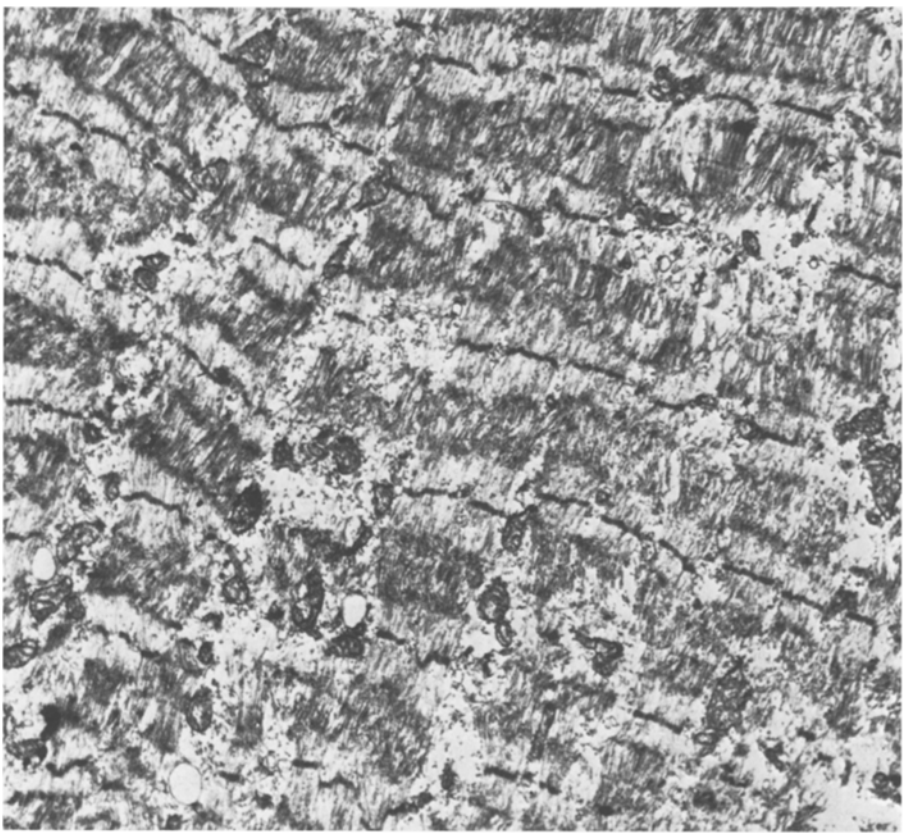

Fig. 5. Ruptured myofilaments, zik-zak formed Z lines and intracellular edema. M. tibialis ant. 1 h after injury. Macroscopically intact region. 2800 x

torn. The sarcolemma demonstrated a slight irregular, undulated configuration. The nuclei in the necrotic cells manifested degenerative changes, as early as 12 h after injury.

There was prominent clumping of the chromatin peripherally, focal swelling of the outer nuclear membrane, resulting in the formation of small blebs; and eventual fragmentation of the nucleus. The nucleolus appeared to be the nuclear component most resistant to morphologic change; its eventual fate could not be discerned however.

One hour after injury, intact muscle fibers, as well as muscle fibers with hypercontraction, ruptured filaments, zik-zak formed Z lines, and intracellular edemas were found in the macroscopically intact muscles (Fig. 5.). In this zone later the swelling of mitochondria, with formation of transversal lamellae (Fig. 6.), extra- and intracellular edema, and moderate dilatation of sarcoplasmic reticulum could also be observed. In the macroscopically intact region cell necrosis was not found.

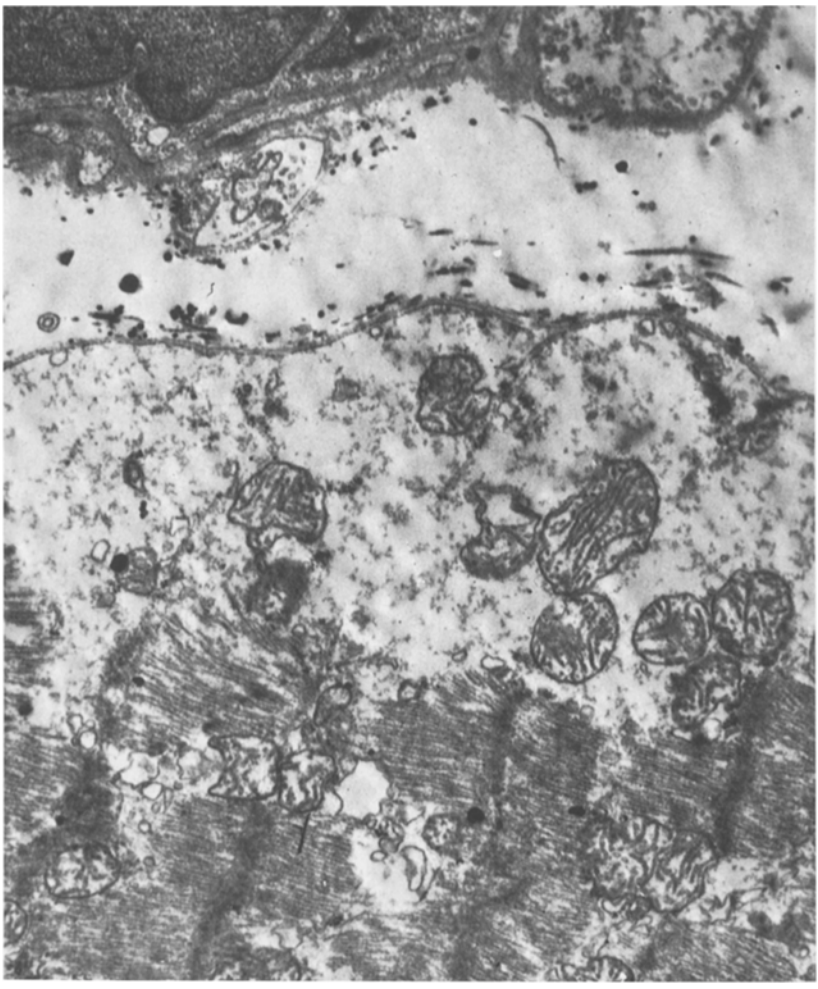

Fig. 6. Intracellular and intercellular edema, decreased glycogen content. Swelling of mitochondria with formation of transverse lamellae and moderate dilatation 19 h after injury. Macroscopically intact region. 4300 x

Discussion

The present study includes the observation of the fine structural changes of skeletal muscles, elicited by crush injury, and muscle fibers showing irreversible changes in the primary point of impact.

On the basis of the observations of the crush-injured muscle, two pathomechanical factors have been considered to be responsible. The immediate changes involve the damage of the contractile elements, even of the myocytes, and probably result in important physico-chemical changes of muscle proteins. As to the consequences of the immediate traumatic effect, contraction-hypercontraction should be stressed. Hypercontraction is most probably induced by this direct effect, followed by the recorded

changes. Obvious early muscle changes may not be explained by hypercontraction alone, but also other factors have to be considered. In our opinion the myofilamental changes are caused by direct traumatic effect and/or by the consecutive hypercontraction. They appear immediately after or a short time after the injury. Damage of the other cell structures develops only later, i.e., functional alteration of mitochondria appears 2 h after the injury (Järvinen and Sorvari, 1975; Józsa, 1974: Möttönen et al., 1972; Niwelinski et al., 1974), morphological changes become manifest even later. Similar time-dependent progress of the alteration can be observed in the tubules of the sarcoplasmic reticulum. About 2 h after the injury, the myofilamental alterations reached a peak and no progression of the damage could be demonstrated later. Conversely, based on other mechanisms (e.g., hypoxia, circulatory damages, absence of function, hemorrhage) the damage of mitochondria, sarcoplasmic reticulum progresses a long time after the injury.

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