A- und I-Filamenten (Figur 2a) konnten nie klar als abgewinkelte Anteile (HMM) der Subfilamente (LMM) dargestellt werden – wie es für Myosinmoleküle zu erwarten wäre –, sondern erscheinen diffuser, in ihrer Elektronendichte etwa dem Matrixprotein vergleichbar. Eine Auflagerung von Plasmaproteinen auf die Myosinbrücken als Folge des Fixierungsprozesses könnte eine Erklärung für dieses Erscheinungsbild sein.

Es ist vorerst noch nicht möglich, den Befunden eine sichere Deutung zu geben. Unter der Voraussetzung, dass es sich bei den A-Filamenten um Myosinfilamente handelt, darf man wohl annehmen, dass neben Myosin noch andere Eiweisse, eventuell Paramyosin (Tropomyosin), wesentlich an ihrem Aufbau beteiligt sind. Wahrscheinlich entsprechen die Subfilamente den LMM-Anteilen der Myosine, während die Matrixsubstanz aus einem Paramyosin besteht. Bemerkenswert erscheint die geringe Dicke der A-Filamente verglichen mit den dicken Filamenten anderer typischer Paramyosinmuskeln (ABRM von Mytilus) und die regelmässige Anordnung der Myosinmoleküle sowie der spiralige Verlauf ihrer LMM-Anteile. Die Zahl der Myosinmoleküle in einem Umgang entspricht etwa der Zahl der I-Filamente pro A-Filament.

Der Kontraktionsprozess dieser Muskelzellen dürfte mit Sicherheit nach dem Modell des HUXLEYschen Gleitmechanismus ablaufen.

Die hier mitgeteilten Befunde konnten an Hand einer grossen Zahl elektronenmikroskopischer Aufnahmen bei Primärvergrösserungen von 20000 und 40000 und 6–8 facher Nachvergrösserung gesichert werden.[14]

Summary. Thick A-Filaments (myosin filaments) of entoproctan muscle cells each consist of 9–11 fibrillar subunits, ca. 30 Å in diameter, embedded in a protein matrix of lower electron density (tropomyosin ?). Unlike hitherto described paramyosin filaments, these subunits are regularly arranged in a single circle near the outer edge of each filament. They seem to run in spiral windings around the filaments axis. The protein matrix shows a faint banding along the filament, resembling to the tropomyosin-A pattern but with a much shorter periodicity (ca. 60 Å).

P. EMSCHERMANN

Anatomisches Institut der Universität, D-78 Freiburg i. Br. (Deutschland), 21. April 1969.

[14] Nach Abschluss des Manuskripts erschien eine Arbeit über die Organisation der Stielmuskulatur von *Barentsia gracilis* (J. F. REGER, J. Cell Sci. *4*, 305–325 (1969). Der Verfasser kommt darin hinsichtlich der helikoidalen Struktur der Kamptozoenmuskulatur zu gleichen Ergebnissen wie oben geschildert.

High Structural Stability of Vascular and Glial Basement Membranes in Areas of Total Brain Tissue Necrosis

A cone-shaped hollow copper rod, filled with dry carbon dioxide and kept at −70 °C, was applied to the exposed frontoparietal bones of adult Syrian hamsters in order to obtain a focal area of cortical softening. In the course of studying the electron microscopic appearance of the cold induced tissue damage, an interesting observation was made on the basement membranes of necrotic intracerebral blood vessels.

In the normal animal, the cerebral capillary is visualized as an endothelial tube whose outer surface is coated by a continuous basement membrane. On the latter, the astrocytic foot-plates abut directly without interposition of any perivascular space[1]. In some places, however, the capillary basement membrane passes from a simple sheath-like structure to a multicameral system of closed compartments in which are found the pericytes or their cytoplasmic processes[1]. In the larger intracerebral vessels (veins and arteries), there exists a true perivascular space which normally contains adventitial cells and scattered bundles of collagen fibrils. This perivascular space is seen to be bounded internally by the basement membrane proper of the vascular wall (basement membrane of the endothelium or of the tunica media), and externally by the basement membrane of the surrounding astrocytes, i.e. by the so-called glial basement membrane[2,3].

In areas of cold induced total cortical necrosis, dissociation and disintegration of almost all constituents of the neuropile occur together with severe structural alterations of the nerve and glial cells. The intracerebral blood vessels can then be seen floating freely on a sea of cellular debris consisting of shattered neuronal and glial cell processes as well as altered organelles, e.g. mitochondria, released from the disrupted tissue components. In the center of such cold induced softening, the parietal cells of the blood vessels (endothelial cells, pericytes, smooth muscle cells) undergo, as a rule, also rapid disintegration. In this case, however, more often than not, the basement membranes are the single structural elements of the necrotic blood vessels, able to hold fast to their usual morphological features for quite some time after all other constituents of the vascular wall have wasted away (Figure). The remnants of the necrotic endothelial cells can project into, partially obliterate, and actually empty out into the vessel lumen, since there is no longer an intact cytoplasmic membrane holding up the cellular debris inwardly. No such an event can occur with the necrotic pericytes, since these are completely surrounded by and lodged within the meshes of the basement membrane lattice-work. As for the larger intracerebral blood vessels, the glial basement membrane bounding externally the perivascular space, appears to lie completely bare of its astrocytic foot-plates, after these have followed the rest of the tissue into total wastage. Notwithstanding this denudation, the glial basement membrane no less than the vascular basement membrane proper generally fails to show any tendency to loosen up and dissolve away. Often the perivascular spaces of the necrotic veins and arteries contain a few extravasated red blood cells. Occasionally the hemorrhagic collection tightly fills up

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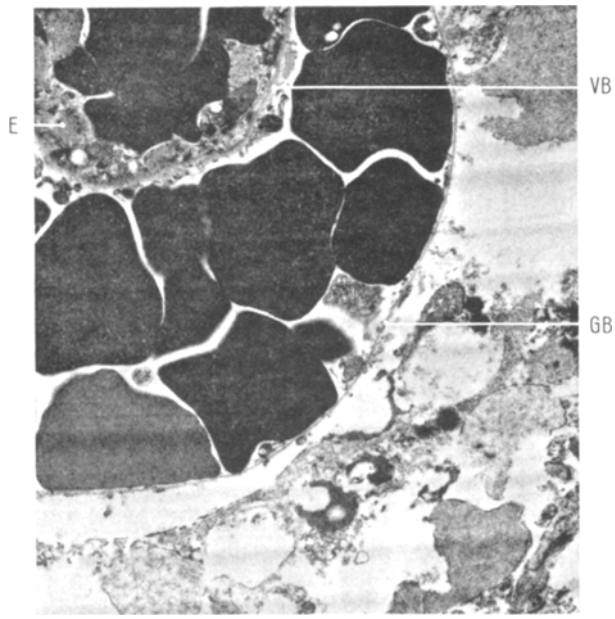

This shows a small necrotic vein within an area of cold induced cortical softening. The endothelial cells (E) are about to undergo structural disintegration but the vascular basement membrane (VB) is still preserved. The perivascular space is stuffed with extravasated erythrocytes. The distended glial basement membrane (GB) appears totally denuded from astrocytic foot-plates. Nevertheless, it is obviously able to prevent further spreading of the perivascular hemorrhage. × 5170.

the perivascular space. Under the pressure of the extravasated erythrocytes, the denuded glial basement membrane may become distended; however, even under these conditions, it usually yields no evidence of actual rupture (Figure). On the contrary, it appears to be, like the vascular basement membrane, a very resistant structure, able to prevent, at least for some time, spreading of perivascular hemorrhages into the surrounding necrotic neuropile, even after all cellular barriers have broken down.

Light microscopy has long since shown us that the blood vessels are the stablest tissue components in an area of cerebral necrosis. The electron microscope has confirmed this classical observation, and has further pointed out that the most resistant structural elements of the vessel walls themselves, are the basement membranes including the perivascular glial ones. Our present findings, which partially corroborate the results of previous electron microscopic studies on traumatic, radiation, and anoxic-ischemic brain lesions[4,5], reveal that both vascular and glial basement membranes can 'survive' for quite some time without being associated with any of the living cells from which they were originally elaborated.

Zusammenfassung. An der Grosshirnrinde von Goldhamstern wurde durch lokale Kälteeinwirkung eine umschriebene Erweichung hervorgerufen. Im Zentrum derselben wiesen nicht nur die neuronalen und gliösen Gewebskomponenten, sondern häufig auch die Blutgefässe schwerste nekrotische Strukturveränderungen auf. Bemerkenswerterweise blieben selbst nach dem völligen Untergang der Gefässwandzellen und der perivaskulären Astrozytenfußstücke die vasalen und gliösen Basalmembranen eine gewisse Zeit erhalten. Bei grösseren kortikalen Blutgefässen konnte sogar wiederholt beobachtet werden, dass die von astrozytären Zytoplasmafortsätzen bereits völlig entblössten gliösen Basalmembranen noch imstande waren, die Ausbreitung massiver perivaskulärer Erythrozytenansammlungen in das umgebende nekrotische Neuropil zu verhindern.

K. Blinzinger, A. Matsushima and A. P. Anzil

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Special Forms of Amitotic Nuclear Division in Striated Muscle and Other Insect Tissues

Numerous examples of amitotic nuclear division have been reported by different authors[1–3] in several animal tissues in normal and pathological or experimental circumstances. Up to the present we have not found any literature concerning the amitotic nuclear division in insect tissues, where it is a frequent process. The main purpose of this paper is to describe briefly some amitotic pictures found in insect tissues and to point out – as a preliminary report – the observation of a new type of complex amitotic nuclear division in insect striated muscle cells.

Adult specimens and embryos of Diptera, Hymenoptera, Lepidoptera and Coleoptera were studied in the present work. Microscopical preparations were performed by 2 different methods: (a) Fragments of the insect tissue were placed in a centrifuge tube and homogenized lightly by means of a glass rod and then the tissue pulp was suspended in 5 ml distilled water. The suspension containing the isolated cells and tissue fragments was collected and centrifuged at 1000 rpm for 3 min. The supernatant was aspirated off. Methanol-glacial acetic acid (3:1) fixative was added without disturbance of the pellet at the bottom of the centrifuge tube. After 2 h fixation the cells were suspended, centrifuged and resuspended in fresh fixative. Slides were prepared by the air-drying technique of Rothfels and Siminovitch[4]. (b) Fragments of insect

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