tion, with the myofibrils and mitochondria comprising only 32% of the cell volume. Similarly, the fractional cell volumes of the myofibrils and mitochondria in rat (82%) and calf (69%) ventricles are greater than in guinea-pig atrium (55%), being associated with their ability to develop greater contractile tension. The fractional cell volumes occupied by the sarcotubular systems in atrial and ventricular muscle are appreciably less than in skeletal muscle (13%)[13]. This implies a decreased storage capacity which is consistent with the rapid effect of altered extracellular calcium concentration on developed tension in the myocardium[18].

Mitochondrial cell volume determined by morphometry exhibits reasonable correlation to volumes calculated by measuring copper content[19]. Guinea-pig left ventricular wall and left atrium possessed copper contents of 0.33 and 0.19 μmoles Cu/g dry weight, respectively. Stereological examination of these cells provided mitochondrial volumes of 0.35 and 0.144 $\mu m^3/\mu m^3$ cell volume (Table), respectively. For amphibian atrial trabeculae, GREEN et al.[20] determined the mitochondrial volume as approximately 15%. The lower mitochondrial volume observed in atrial muscle, as well as the lower myofibrillar content per unit dry weight[21], may reflect a lower total consumption of ATP by the contractile apparatus of the cells.

A_i/V_{cell} for atrial sarcolemmal membrane, determined on randomly oriented sections, was 0.24 ± 0.03 $\mu m^2/\mu m^3$ which was in reasonable agreement with values determined in ventricular cells[9,10], 0.30 ± 0.02 $\mu m^2/\mu m^3$. Our inability to orient the tissue necessitated determination of the sarcoplasmic reticulum surface to volume ratio by the relationship $(A_{sr}/A_{sl})\ (A_{sl}/V_{cell})$, where A_{sr}/A_{sl} is independent of the sectioning angle [12,17]. The ratio determined for the sarcoplasmic reticulum was 0.36 ± 0.04 as compared to 1.2 $\mu m^2/\mu m^3$ for ventricular muscle[9,10]. The lower A_{sr}/V_{cell} determined for atrial muscle, as compared to ventricular muscle, is associated with a smaller sarcoplasmic reticulum fractional cell volume as well as a decreased dependence of atrial muscle on intracellular calcium stores[22].

Zusammenfassung. Mit morphometrischen Methoden wurde beim Meerschweinchen eine quantitative Analyse von Volumen und Oberflächenareal der Ultrastrukturkomponenten der Herzvorhof-Muskulatur durchgeführt, die mit dem Erregungs-Kontraktions-Kopplungsvorgang und der Erschlaffung verknüpft sind.

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Influence of Antihistamine and Compound 48/80 on Healing

The conception of the histamine-forming capacity (HFC) of tissues and the possibility of altering the HFC experimentally, more specifically of elevating it, has provided the possibility of accelerating the rate of wound healing[1–3]. In the tissues from skin wounds of the rat, the HFC has been found conspicuously elevated compared to that of intact skin, whereas the histamine content of wound tissue was low. The HFC of the skin can be experimentally increased: Repeated injections of compound 48/80 or polymyxin depletes the skin of histamine, whereby the histidine decarboxylase activity is concomitantly increased. In rats treated for a 3-day period with daily injections of 48/80 or polymyxin, wound healing has been found to be accelerated; both the tensile strength of the experimental wound and the collagen content in the granulation tissue were significantly increased. This increase persisted throughout the whole period of fibroplasia of the healing wound. The stimulation of healing was tentatively accounted for by an earlier formation of fibroblasts and thereby an earlier onset of collagen formation[4].

The pharmacologically liberated histamine, which reaches the skin via the blood-stream, i.e. extracellular histamine, does not enhance healing, since, on injecting long-acting histamine, the tensile strength of the wound was the same as the control wound in the untreated rat, as shown by KAHLSON et al.[5].

Material and methods. In order to strengthen further the evidence of the ineffectiveness of extracellular histamine in enhancing healing, rats were treated with the antihistamine shortly before receiving the histamine liberating drug. The tensile strength of 5-day-old healing skin incisions was measured with a tensiometer; the technique and procedures involved have been described in detail[4,6]. A control wound was first tested on each animal. The antihistamine was then injected followed by 48/80 15–20 min later. The antihistamine mepyramine (neoantergan®) was given i.p. in a dose of 25 mg/kg and 48/80 i.p. 1 mg/kg. This treatment was given 3 days before the experimental wound was inflicted.

Results. The dose of antihistamine was effective against extracellular histamine, because 48/80 alone produced signs of histamine shock; within 15–20 min the rats were lying still in their cages with stridor, cyanosis, signs of itching and sometimes oedema of nose and paws. The mortality in histamine shock in our previous studies was 20%[4]. When the rats had antihistamine prior to 48/80, no signs of shock were seen; the rats moved about in the normal way, looked unaffected and no deaths occurred.

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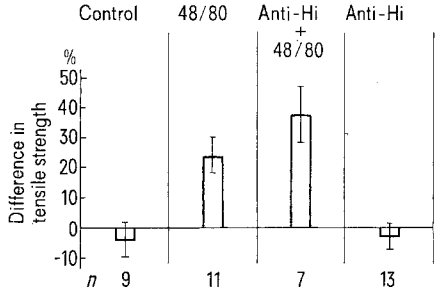
The results are shown in the Figure, which, in addition, includes 2 groups studied earlier[4] namely a control group and a group treated with injections of 48/80 only. In the control group there was no significant difference in the tensile strength between a 5-day-old healing skin incision on one half of the back, compared to an identical incision on the contralateral side ($p > 0.1$). The group pre-treated with antihistamine only did not differ significantly in tensile strength of the control wound ($p > 0.1$). However, the 2 groups pre-treated with 48/80 only or with antihistamine plus 48/80 displayed significantly increased wound tensile strength on HFC-elevating treatment ($0.01 > p > 0.001$).

Discussion. The results with the histamine-liberator show that blood-carried histamine, extracellular histamine, does not affect the healing process, whereas treatment with 48/80 which elevates HFC in the wound tissues enhances wound healing. The ineffectiveness of extracellular histamine on wound healing is also apparent from experiments with long-acting histamine[5]. Likewise, in the pregnant rat the rate of wound healing is not enhanced during the last third of the pregnancy when the histamine generated by the foetuses reach the highest levels[7,8]. It would appear that the stimulating effect on the healing processes is brought about by histamine newly formed, 'nascent histamine'[9], in the wound proper.

In connection with the present experiments, it should be recalled that in anaphylaxis histamine is not merely reduced but also newly formed[9]. Antihistamines are not always effective in certain allergic conditions. Mepyramine suppresses some histamine-stimulating effects, e.g. the contraction of the smooth muscles of the bronchi and the gut, and has been defined as an H_1-receptor antagonist[10]. An H_2-receptor antagonist (burimamide) can antagonize some responses of histamine which cannot be blocked by mepyramine[11]. The elevated rate of formation of histamine, e.g. in wound tissues of the rat is not affected by pretreatment with mepyramine (SANDBERG, unpublished). An enzyme inhibitor would restrain the elevation of HFC, as shown by SANDBERG and STEINHARDT[12] to occur in the wound tissues under the influence of cortison administration, whereby the rate of healing was found retarded.



Difference in wound tensile strength (T.S.), mean ± standard error of mean in 4 groups. Number of animals are given. Anti-Hi, antihistamine.

Zusammenfassung. Der Effekt der Vorbehandlung mit Antihistamin und Compound 48/80 wurde an heilenden Wunden bei Ratten untersucht. Da die Vorbehandlung die heilungsstimulierende Wirkung des 48/80 nicht veränderte, ist anzunehmen, dass das intrazellulär gebildete Histamin, sowie die Aktivierung der Enzyme für die Beschleunigung des Heilprozesses entscheidend sind.

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Cytoplasmic Filaments Associated with Lipid Droplets in Chondrocytes of the Rat Auricular Cartilage

The nature of the boundary between the lipid droplet and the surrounding cytoplasm is still an unsolved problem. It is also questionable whether or not the lipid-cytoplasm interface is of the same nature in all cell types which contain lipid in particulate form[1,2]. LUCKENBILL and COHEN[3] and WOOD[1] described a highly ordered complex of fine filaments surrounding the fat droplets in some of the chick adipose cells. The presence of such filaments in mammalian adipose cells could not be confirmed without reserve[2]. The present communication deals with the finding of cytoplasmic filaments around the lipid droplets in chondrocytes of the cartilage in the external ear of the rat.

Small pieces of cartilage dissected from the auricle of adult albino rats were fixed at 4 °C for 1–2 h in 1% osmium tetroxide (alone or with 6.25% glutaraldehyde) buffered in 0.06 *N* cacodylate buffer. The tissue was embedded in Durcopan Fluka, sectioned with glass knives in a Reichert's ultramikrotome, stained with lead citrate and uranil acetate and examined with the Siemens Elmiskop I.

The elastic cartilage in the external ear of the rat is of cellular type. In the adult tissue, the chondrocytes contain large lipid droplets. In all but the peripheral cells, a single large lipid droplet fills up the cytoplasm to the degree of giving the cell the appearance of a signet-ring like white adipocyte ('Fettknorpel' or adipose cartilage of SCHAFFER[4]). Near the periphery of the cartilaginous plate, the chondrocytes contain one or more lipid droplets of different size. The rest of the cytoplasm is characterized by abundant 60–70 Å thick filaments spaced at intervals of about 60–200 Å. In general, they run in different directions, but there are large areas of cytoplasm, in which all the filaments display a regular

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