from chondrocytes into chondroclasts. The latter prepare the conditions for reparative regeneration of the cartilage. Such catabolic reactions are perhaps to a certain extent characteristic of the bradytrophic cartilaginous tissue under normal conditions, and they thus fall into the general biological category.

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POST-TRAUMATIC MICROANGIOPATHIES

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KEY WORDS: post-traumatic microangiopathies; endothelium; hemolysis; microthrombus; emboli; hyalinosis

In the late stages after injury to the limbs foci of recent destructive changes appear in the muscle fibers and nerve structures [2]. Since the basic processes of activity of tissue components are largely determined by the state of the microcirculation [1, 3] it was decided to assess the role of the vascular factor in progression of these dystrophic changes. This paper describes the results of an ultrastructural study of the microvessels of a skeletal muscle in the late stages after trauma.

EXPERIMENTAL METHOD

Muscles of the injured limb served as the experimental material. Mechanical trauma was inflicted on the sural muscles of five noninbred male rats by Cannon's method. An operation to replant the hind limb was performed on three rats. A particular feature of the model was the absence of prolonged thermal ischemia (during the operation the blood flow along the main vessels was preserved), and the effect of mechanical injury was excluded by studying the muscles below the level of replantation. Pieces of muscles were fixed consecutively in a cold solution of formol-sucrose and a 1% buffered solution of OSO_4, and embedded in Araldite. Ultrathin sections were examined in the JEM 7A electron microscope.

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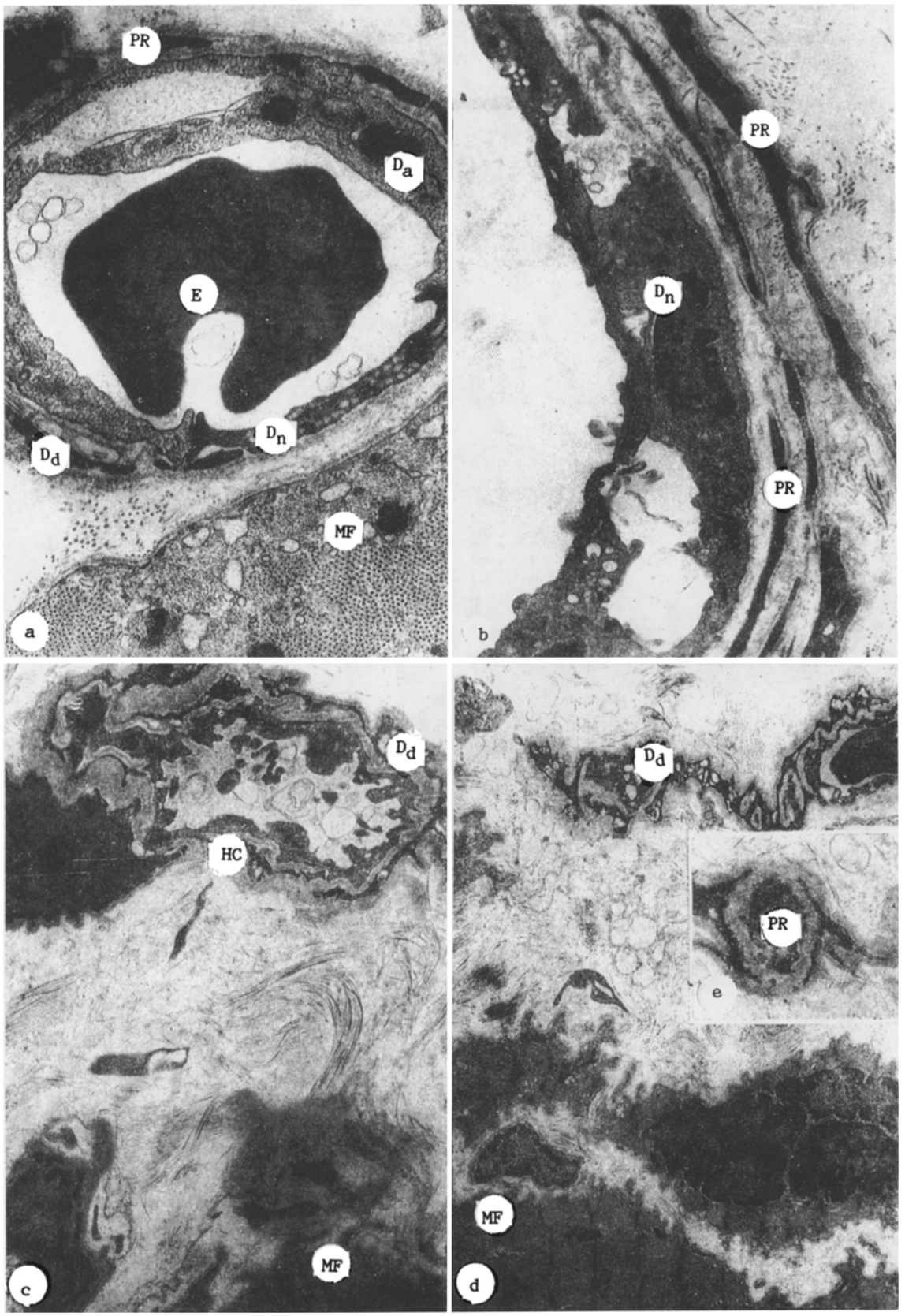

Fig. 1. Ultrastructure of wall of microvessels in muscles after injury (late stages). a) Different physiological state of endotheliocytes: D_a) "dark" activated cell; D_n) "dark" necrobiotic cell, D_d) "dark"" degenerative cell; PR) pycnotic remains of endotheliocytes; MF) here and in Fig. 2, myofibrils; E) erythrocyte. 16,000×; b) Remains of pycnotic endotheliocytes (PR) between separated basal membranes. Vesicles and vacuoles in endotheliocyte. D_n) "Dark" cell in a state of necrobiosis. 7500×; c) Hyalinosis of capillary (HC). Widened condensed basal membranes, pycnotic remains of endotheliocytes, cytoclasmatosis and membrane complexes in lumen of vessel. 4000×; e) Degenerative remains of wall of a microvessel. Hyalinosis. Perivascular sclerosis. 2800×.

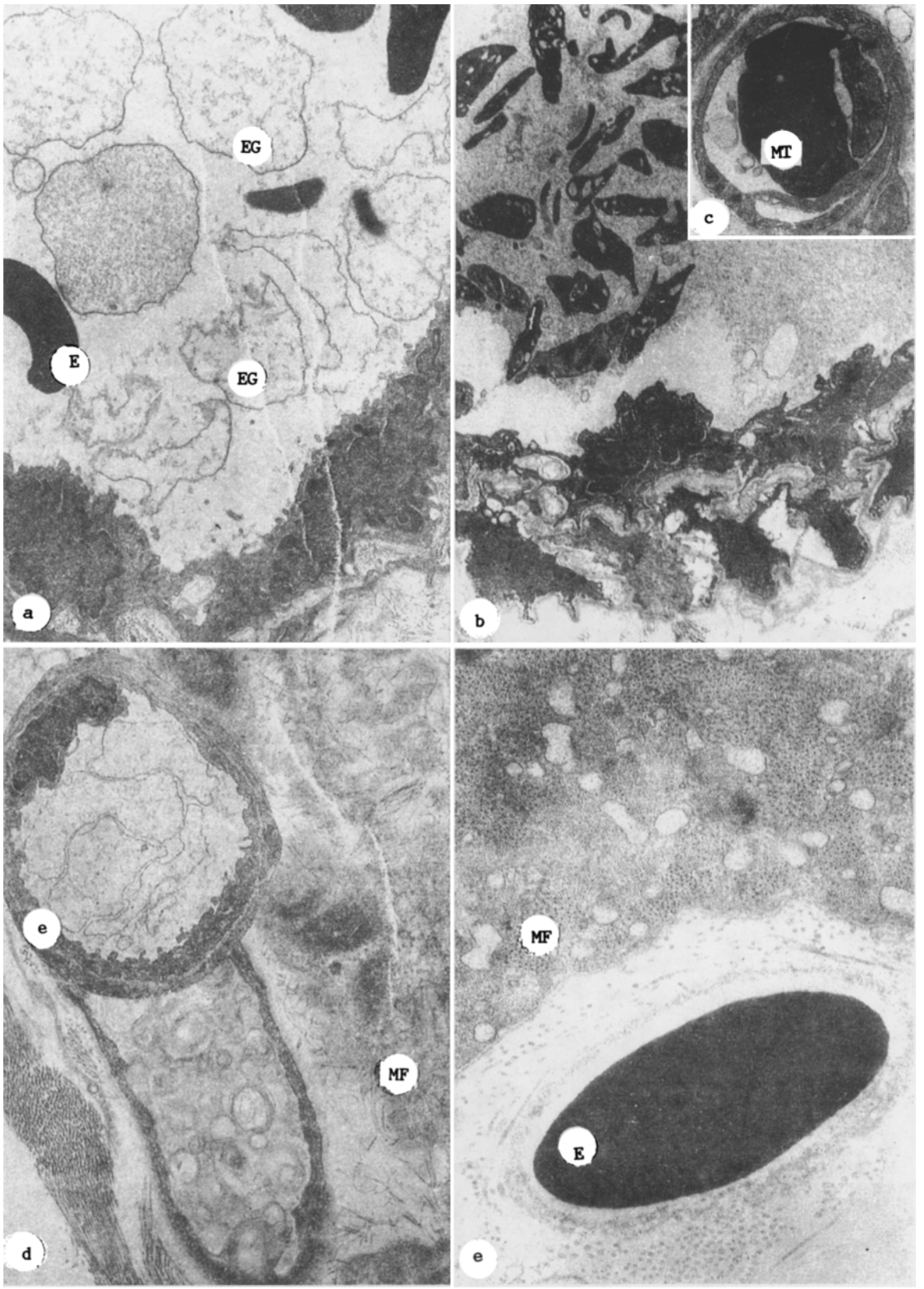

Fig. 2. Features of disturbance of rheologic properties of the blood: a) various stages of hemolysis of erythrocytes: E) erythrocyte; EG) "ghost" erythrocyte; b) cluster of degranulated platelets in lumen of arteriole; P) platelets. 3500×; c) Microthrombus (MT) in capillary. 7500×; d) Occlusion of lumen of venule by membrane complexes, on right — necrosis of muscle fiber. 10,500×; e) Occlusion of capillary lumen by membranes of hemolyzed erythrocytes. 3500×; e) Erythrocyte (E) in lumen of acellular capillary. 16,000×.

EXPERIMENTAL RESULTS

Changes in the microvessels in the crushed skeletal muscle and the replanted limb were similar in direction, evidence of the stereotyped nature of the response of the microcirculatory bed to trauma. Meanwhile the number of injured vessels and the severity of the destructive and degenerative changes in them were greater than in the case of replantation.

Under normal conditions the endothelial lining of the microvessels consists of endotheliocytes of varied electron density, associated in most cases with the cycle of their activity. The so-called dark cells have well developed organelles and are rich in ribosomes and polysomes. Most workers rightly class them as young activated cells (Fig. 1a). With an increase in the functional activity of the organ these cells, while preserving their intracellular organization, can form microvilli, baylike invaginations, and marginal folds.

The "dark" endotheliocytes of the microvessels of the damaged muscles differed from the same cells under normal conditions in the extraordinarily high electron density of their cytoplasm, in which organelles were difficult to distinguish. Transport micropinocytotic vesicles appeared to be "immured" in the "compressed" cytoplasm (Fig. 1b). The nuclei were hyperosmic and often appeared pycnotic (Fig. 1c). To study the time course of the vascular changes we traced the fate of these cells, the successive stages of their degeneration to cloudy swelling, and as a result, the state of the "dark" cells could be assessed as necrobiotic (Fig. 1). Signs of severe vacuole and vesicle formation also appeared in the "dark" endotheliocytes of similar type, followed by the formation of transendothelial channels and ports, evidence of a severe disturbance of vascular permeability. The process of formation of microvilli ended in cytoclasmatosis (Fig. 1c), or even sequestration of sheets of endothelium with the formation of acellular capillaries. The presence of erythrocytes in such capillaries was evidence of the possible preservation of the blood flow in them. Areas of necrosis and rupture of the capillary and venular walls was accompanied by extravasation. At the same time clear signs of regeneration of the endothelium appeared (solitary mitoses in the endotheliocytes, endothelization of the acellular and necrotic areas due to migration of cytoplasmic outgrowths of neighboring cells along the exposed basal membranes, and de novo vessel formation).

The walls of many capillaries appeared to consist of one layer only on account of the pycnotic remnants of endotheliocytes trapped between the separated basal membranes (Fig. 1b), evidence of the repeated renewal of the endothelial cover, evidently due to the persistent character of the action of endogenous traumatic factors (death of muscle fibers and nerve structures of autoimmune genesis as a result of imperfect reinnervation, local hypoxia, etc.). Necrobiotic changes in the endothelium were accompanied by seepage of plasma into the walls of the microvessels, followed by haylinosis (Fig. 1c, d) and necrosis or lysis of the walls (Fig. 2d). With an increase in the periods of observation the number of damaged microvessels increased, to reach 20-25% of their total number.

Another group of changes combines features of disturbance of the rheologic properties of the blood. Besides evidence of juxtamural stasis of the erythrocytes and the formation of "rouleaux," signs of "sludging" also were observed. Where erythrocytes were concentrated, cells of different density could be seen, some of them having the appearance of "ghosts" (Fig. 2a). Membranes of hemolyzed erythrocytes under these circumstances became irregular in shape. The lumen of certain microvessels was filled with membrane complexes of dying erythrocytes, circularly coiled microvilli of the endothelium, pinched off in the course of cytoclasmatosis (Fig. 2c). Incidentally, such membrane concentrations were sometimes found in relatively undamaged vessels, reflecting the ability of these structures to be transported and for free emboli to be formed from them.

Besides evidence of precipitation of plasma proteins and adhesion of platelets on the luminal surface of the damaged microvessels, the formation of microthrombi could be seen (Fig. 2b). In such areas foci of necrosis of muscle fibers were often seen (Fig. 2c).

The degenerative-necrobiotic changes described above in endotheliocytes, accompanied by disturbance of permeability of the microvascular wall and hyalinosis, and by well-marked changes in the rheologic properties of the blood, form a group of changes which can be interpreted as post-traumatic microangiopathies, resembling those taking place in diabetes, hypertension, chronic alcohol poisoning, and other diseases, associated with the action of a constant pathogenic factor [4, 5]. In the late stages after trauma, the role of pathogenic factor may be played by high-molecular-weight compounds entering the blood stream on death of the muscle and nerve structures, which damage the endothelium and contribute to the release of biologically active substances, notably platelet activating factor (PAF), and aggregation of erythrocytes and platelets. The hypoxia accompanying these changes aggravates the dystrophic changes in the muscle tissue, evoking damage to the new muscle fibers and nerve structures, and thereby ensuring a chronic progressive course of the pathological process

in the muscle. On the basis of all these findings post-traumatic microangiopathies can be regarded as leading factors in the genesis of the late dystrophic disorders in injured muscle.

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VOLUME BLOOD FLOW IN THE POPLITEAL LYMPH NODE REGION AFTER EXPERIMENTAL MYOCARDIAL INFARCTION AND ITS TREATMENT WITH DOGROSE POLYPHENOLS PREPARATION

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KEY WORDS: experimental myocardial ischemia; lymph node; volume blood flow; correction

Myocardial infarction causes considerable disturbances of the blood and lymph circulation throughout the body. The degree of these disturbances differs at different periods of the disease. The aim of the present investigation was to study the dynamics of the blood flow in the zone of a somatic lymph node in experimentally myocardial ischemia and during its correction. The blood flow was recorded by polarography based on hydrogen clearance [1, 2].

EXPERIMENTAL METHOD

Noninbred male rats aged 2 months and weighing 160-200 g were used. The test object was the popliteal lymph node. The animals were divided into 10 groups with 10 rats in each group. Animals of group 1 served as the control. Myocardial ischemia was induced in the rats of the remaining groups under ether anesthesia by thoracotomy and ligation of the left coronary artery at a distance of 3-4 mm from its origin from the aorta, followed by closure of the operation wound in layers. The volume velocity of the blood flow in group 2 was recorded on the 1st day after the operation, in group 3 on the 3rd day, and in group 4 on the 7th day. The times of investigation of the volume blood flow corresponded to the acute period of myocardial infarction, accompanied by marked clinical manifestations. Once a day the animals of groups 5, 6, and 7 received the dogrose polyphenols preparation through a gastric tube in a dose of 1 mg/50 g body weight [4]. The volume velocity of the blood flow in group 5 was recorded on the 1st day after the operation, in group 6 on the 3rd day, and in group 7 on the 7th day after the operation.

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