## ELECTRICAL STIMULATION OF ARTIFICIAL OSSIFICATION OF A MUSCLE FLAP DURING PLASTIC REPAIR OF A BONE DEFECT

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Experiments on rabbits showed that during plastic repair of a bone defect in the tibia by means of a muscle flap on a central vascular pedicle and electrical stimulation by a direct current of  $18-20~\mu\text{A}$  with change of polarity reorganization of the muscle flap was accelerated and bone tissue formed in its substance. Electrical stimulation of osteogenesis after muscle grafting promotes restoration of anatomic integrity of the bone.

KEY WORDS: muscle grafting; electrical stimulation; osteogenesis.

During the plastic repair of bone defects by means of a muscle flap the graft gradually atrophies and is converted into scar tissue [3, 4]. Replacement of the graft by bone tissue and restoration of the anatomic integrity of the bone do not take place [2, 5]. Investigations have shown that osteogenesis can be stimulated after fractures by application of a very weak electric current [1, 6, 8], through the intervention of electrochemical processes [7, 9, 10]. Investigation of the oriented reconstruction of the muscle graft and the formation of bone tissue is thus particularly important.

The object of this investigation was to study the possibility and particular features of electrical stimulation of osteogenesis in the substance of a muscle flap during plastic repair of cavities in bone.

## EXPERIMENTAL METHOD

Experiments were carried out on 42 chinchilla rabbits weighing 1.6-2.4 kg. Under aseptic conditions plastic repair of a bone cavity in the upper third of the tibia through an oval defect measuring  $7 \times 10$  mm on the outer surface was carried out with a portion of the lateral head of the gastrocnemius muscle. The muscle graft, on a central vascular pedicle was fixed transosseously in the cavity and silver wire electrodes 0.25 mm in diameter were implanted into the substance of the flap at the level of the cortical layer. The electrode of opposite polarity, made of biologically inert electrically conducting polymer, was located on the body of a miniature device introduced under the skin in the interscapular region. The device stabilized the strength of the direct electric current at  $19-20~\mu\mathrm{A}$  irrespective of any change in interelectrode resistance in the biological tissues. The device was actuated and polarity of the electrodes changed by means of an external commutator on the skin surface.

In series I (18 experiments) no electrical stimulation was applied (control). In series II (eight experiments) the electrodes in the muscle flap were anodes 3-4 weeks after the operation. In series III (16 experiments), after the same procedure as in series II, the polarity of the electrodes was reversed and for 4-8 weeks the electrodes in the muscle flap were cathodes.

The experimental results were evaluated on the basis of clinical, x-ray, and histological data. The animals were killed by intravenous injection of air 4 and 12 weeks after the operation. Material was fixed in 10% neutral formalin and embedded in celloidin; microscopic sections were strained with hematoxylin-eosin and by Van Gieson's method.

## EXPERIMENTAL RESULTS

The operation was followed by some worsening of the animals' general condition and they became apathetic and hypodynamic. The function of the experimental limb was restored after 2-3 weeks.

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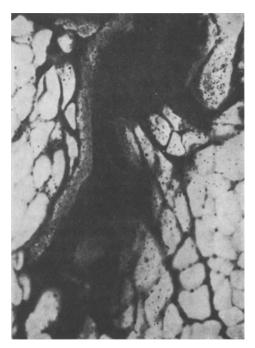


Fig. 1. Microfoci of coagulation necrosis in substance of modified muscle flap near anode on 28th day after operation. Van Gieson,  $400 \times$ .







Fig. 3

Fig. 2. Reconstruction of muscle flap on 90th day after operation during stimulation of osteogenesis: formation of new cortical layer and replacement of graft by fibrous tissue in medullary canal. Van Gieson,  $200 \times$ .

Fig. 3. Restoration of cortical layer in substance of muscle flap on 90th day after operation. Hematoxylin-eosin,  $400 \times$ .

In series I (control) emptying and obliteration of the vessels of the flap, atrophy of the muscle fibers, and changes in their staining properties were observed on the 28th day. The nuclei of the muscle cells stained only in the central part of the flap. The peripheral part of the muscle flap, located in the bone cavity, contained a mosaic of disintegrating muscle fibers, which were replaced by loose amorphous connective tissue. A thickened dense fibrous capsule surrounded the altered muscle flap in the medullary canal.

Subsequently the degeneration of the flap into scar tissue gradually progressed. On the 90th day after the operation dense fibrous tissue filled the bone cavity. The fibroblasts around the electrodes were concentric in arrangement and in the substance of the flap they were oriented along its axis. The defect in the cortical layer of the tibia remained the same size as before and its edges consisted of modified bone trabeculae with extensive foci of resorption.

In the experiments of series II during stimulation by a direct electric current coagulation of elements of muscle tissue took place in the zone of the anode. On the 28th day the electrodes were surrounded by a thin-walled connective tissue capsule and conical groups of black granules, which in some areas resembled vesicles. Immediately next to the electrodes the granules formed a continuous homogeneous mass, but away from the electrodes they were distributed mainly in the intercellular spaces and their density diminished (Fig. 1). At the perimeter of the granules numerous leukocytes and fragments of disintegrating free cells could be seen. The fibers of the muscle flap were reduced in size and shrunken, and their staining properties were uniformly altered. Individual fibers had a mummified appearance. Granulation tissue separated the flap from its bony bed.

The peripheral part of the flap 90 days after the operation was completely replaced by dense fibrous tissue. Small clusters of dark brown granules and discrete amorphous masses were found in the intercellular spaces near the electrodes. Necrotic masses were being actively phagocytosed by macrophages. The central part of the graft consisted of muscle fibers with hypertrophied fibrous interlayers. Active resorption of the cortical layer of the tibia in the region of the defect was accompanied by a marked periosteal and endosteal osteogenetic reaction. However, bone trabeculae did not invade the substance of the cicatrized muscle flap.

In series III 4 weeks after electrical stimulation (90 days after the operation) the boundaries of the former defect in the cortical layer of the tibia were difficult to determine. The newly formed bone structures closely resembled the adjacent cortical layer in the density of their x-ray shadows.

Bone trabeculae oriented circumferentially were observed in the cortical defect around the electrodes. The newly formed cortical layer restored the integrity of the bone and consisted of thickened bone trabeculae, some of which were reorganized into lamellar structures. The muscle flap in the medullary canal was replaced by dense fibrous tissue (Fig. 2) with discrete small fragments of disintegrating muscle fibers. Single yellowish brown granules in the intercellular spaces around the electrodes were surrounded by macrophages. The central part of the residual muscle flap at the level of the periosteum changed into a band of dense fibrous tissue, interweaving into the newly formed cortical layer (Fig. 3).

Stimulation by a very weak direct current thus accelerates reconstruction of the muscle tissue around the anodal electrode and stimulates osteogenesis after a change in polarity of the electric current. During plastic repair of bone cavities with a muscle flap on a central vascular pedicle electrical stimulation of osteogenesis promotes the formation of bone tissue in the substance of the flap and restores the anatomic integrity of the bone.

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