ELECTRICAL DIAGNOSIS OF A ZONE OF NECROSIS IN SKELETAL MUSCLES

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UDC 616.74-002.4-073.7

KEY WORDS: electrical diagnosis; necrosis of muscles; impedance; stimulating pulse.

No simple and accessible methods of rapid diagnosis of necrotic areas arising in skeletal muscles after mechanical trauma, prolonged ischemia, and thrombosis of the main limb arteries are yet available for use in surgical practice. The timely discovery of necrosis of muscle tissue in the crush syndrome, which is often accompanied by marked edema, interfering with diagnosis, may be particularly difficult.

The aim of this investigation was to develop an objective method for rapid detection of necrosis of skeletal muscles and to determine its boundaries.

EXPERIMENTAL METHOD

The method is based on the known property of skeletal muscles in the intact organism of changing their electrical resistance during contraction [1]. The absence of any characteristic change in resistance after application of an electric pulse is evidence of the presence of necrosis in a crushed zone and in muscles subjected ischemia for longer than 6 h. The presence of necrosis was confirmed histologically.

Rabbits, anesthetized lightly with ether, in which the crush syndrome was simulated (6-10 h), were used as the test objects. An apparatus, the block diagram of which is shown in Fig. 1, was used. It consists of a stimulator, generating square pulses 10 msec in duration, with a following frequency of 1 Hz, a high-frequency ohmmeter, and separating filters, connecting to two electrodes. The high-frequency ohmmeter incorporated a measuring bridge, combined with phase-sensitive balance detector, and a generator. The frequency of the measuring current was 120 kHz. The investigation was conducted in two stages. Electrodes were applied to an area of skeletal muscle known to be healthy, and the characteristic response was obtained by gradually increasing the voltage of the stimulating pulses. The voltage amplitude of the stimulating pulses was then doubled, and the traumatized limb investigated. Absence of response was interpreted as a sign of necrosis in the particular region. To reduce the risk of injury, especially during investigation of the sound limb, one of the needle electrodes can be replaced by any kind of plate electrode.

EXPERIMENTAL RESULTS

Investigations of various sound caudal rabbit limb muscles showed that the characteristic response is stable in shape (Fig. 2). As a rule the response appeared to a stimulating pulse with a voltage of 1.5-2 V. Any further increase in amplitude of the stimulating pulse was accompanied by a virtually proportional increase in amplitude of the muscle response. If the voltage of the stimulating pulse was much above the threshold level, the contraction process began to spread to the whole muscle, and for that reason the optimal voltage of stimulation was 10-20% above the threshold value. Parallel recording of the electrical response and mechanical contraction using a transducer connected to the needle electrode showed that the change in impedance of the probed regions was virtually synchronous with muscle contraction.

By the use of the suggested method it is thus actually possible to discover areas of muscles in the region tested that preserved their basic function, namely ability to contract ac-

A. V. Vishnevskii Institute of Surgery, Academy of Medical Sciences of the USSR, Moscow. (Presented by Academician of the Academy of Medical Sciences of the USSR M. I. Kuzin.) Translated from Byulleten' Éksperimental'noi Biologii i Meditsiny, Vol. 100, No. 9, pp. 375-376, September, 1985. Original article submitted September 14, 1984.

0007-4888/85/0009-1295$09.50

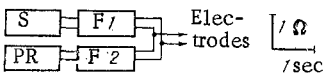

Fig. 1

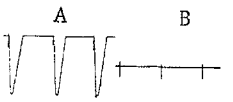

Fig. 2

Fig. 1. Block-diagram of apparatus. S — Stimulator, R) high-frequency ohmmeter, F_1, F_2) high and low frequency filters.

Fig. 2. Trace of electrical response. A) Responses of intact rabbit muscle to threshold pulse (15 V); B) absence of responses to above-threshold stimuli (3 V) in area of necrosis. Vertical lines — markers of stimulating pulses.

tively, in response to electrical stimulation. In necrotic zones of skeletal muscles, despite doubling the voltage of the stimulating pulse, no characteristic change in interelectrode impedance could be observed.

LITERATURE CITED

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MASUGI'S NEPHRITIS: PREPARATION OF AN ACTIVE NEPHROTOXIC SERUM

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UDC 616.611-002-056.43-092.9

KEY WORDS: experimental nephrotoxic syndrome; isolated glomeruli; nephrotoxic serum.

Production of experimental glomerulonephritis (GN) is necessary in order to study its pathogenesis and the metabolic disturbances which arise in this disease, as well as to evaluate new methods of treatment. In nephrology a model of GN, induced with the aid of antikidney antibodies, suggested in 1900-1901 by the Russian scientist V. K. Lindeman [1], and elaborated in more detail by the Japanese scientist Masugi [8] in 1933-1934, is widely used in nephrology. The development of Masugi's nephritis is induced by injecting an experimental animal with a specific nephrotoxic serum (NTS), obtained from another animal after its immunization with the kidney of the first animal. After intravenous injection of NTS into a rat, antikidney antibodies are quickly fixed on the basement membrane of the glomerulus, and within 1 h after the injection, marked proteinuria develops [5]. This is the first, heterologous phase of the disease. Later, starting with the 10th-14th day, the kidney lesion is maintained as a result of fixation of the animal's own antibodies, produced to rabbit antikidney γ-globulin, bound with the basement membrane, in the glomeruli (the second, autologous phase). A definite role in the pathogenesis of Masugi's nephritis may also be played by autoantibodies formed to antigens of the damaged basement membrane in the second phase [2].

If the serum has high nephrotoxicity, the GN which develops closely resembles, in its clinical picture (edema, hypertension) and its laboratory features (massive proteinuria, hypoproteinemia, hyperlipidemia, azotemia), severe human GN of nephrotic or mixed type. However, preparation of the active NTS is associated with certain difficulties. According to our own observations, when rabbits are immunized with a suspension of rat renal cortex, an NTS capable of inducing a nephrotic syndrome was produced in only one-third of rabbits. This

Laboratory for Problems in Nephrology, Department of Pathological Anatomy, First Department of Internal Medicine and Department of Preventive Medicine, and Interclinical Biochemical Laboratory, I. M. Sechenov First Moscow Medical Institute. (Presented by Academician of the Academy of Medical Sciences of the USSR E. M. Tareev.) Translated from Byulleten' Éksperimental'noi Biologii i Meditsiny, Vol. 100, No. 9, pp. 377-380, September, 1985. Original article submitted May 17, 1984.

 0007-4888/85/0009-1296$09.50