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ORIGINAL PAPER

Decreased Creatine Kinase Activity Caused by Electroconvulsive Shock

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Abstract Although several advances have occurred over the past 20 years concerning the use and administration of electroconvulsive therapy to minimize side effects of this treatment, little progress has been made in understanding its mechanism of action. Creatine kinase is a crucial enzyme for brain energy homeostasis, and a decrease of its activity has been associated with neuronal death. This work was performed in order to evaluate creatine kinase activity from rat brain after acute and chronic electroconvulsive shock. Results showed an inhibition of creatine kinase activity in hippocampus, striatum and cortex, after acute and chronic electroconvulsive shock. Our findings demonstrated that creatine kinase activity is altered by electroconvulsive shock.

Keywords Electroconvulsive shock · Electroconvulsive therapy · Creatine kinase · Brain

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Introduction

Induction of seizure in the form of electroconvulsive therapy (ECT) has been used in the treatment of psychiatric disorders for more than 60 years [1]. This therapy is defined as a medical procedure in which a brief electrical stimulus is used to induce a cerebral seizure under controlled conditions [2]. The main diagnostic indications for ECT include depression, mania, catatonia, bipolar and schizophrenia disorders, especially in the treatment of drug-resistant depression, probably more effective than drug therapy, and in the cases that cannot wait to pharmacological interventions. Particularly in the treatment of severe major depression, evidences for the effectiveness and superiority of ECT over other treatments are clear and convincing [3, 4]. There are no doubts about its clinical importance but the adverse effects are focus of much research. The principal adverse effect of ECT, which has been the root of much controversy, is memory impairment [5].

The science of ECT has progressed rapidly over the last 20 years, providing new insights into the mechanisms of action, improving both the acute and long-term efficacy and decreasing cognitive problems associated with the treatment. In this context, some studies do not show credible evidences that ECT causes structural brain damage [6–8]. However, the precise mechanisms of its amnesic, anesthetic and therapeutic effects still remain unknown [2].

Electroconvulsive shock (ECS) seizure provides an animal analog of the ECT used in psychiatry [9]. The procedure involves a series of electrically induced generalized convulsive seizures. Typically, eight convulsions are administered at 48 h intervals over the