Acquired Channelopathies Secondary to Repetitive High Energy Field "Low-Voltage" Electrical Injury

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ACQUIRED CHANNELOPATHIES SECONDARY TO REPETITIVE HIGH ENERGY FIELD, "LOW-VOLTAGE" ELECTRICAL INJURY

By Sarah Price Hancock, MS, CRC

Chen, W., & Lee, R. C. (1994). Evidence for Electrical Shock–induced Conformational Damage of Voltage-gated Ionic Channels. *Annals of the New York Academy of Sciences*, *720*(1), 124–135. https://doi.org/10.1111/j.1749-6632.1994.tb30440.x

Electricity with high energy force changes how the body uses sodium, potassium and other voltage-gated ions.

In cardiac muscle, high-intensity electrical shock can induce a depolarization of the membrane resting potential for minutes, resulting in dysfunction of heart cells. The current hypothesis attributes this reaction to a shock-induced microlesion of the cell membrane, resulting in nonselective ionic and enzymatic leakage.

Chen, W., Zhongsheng, Z., & Lee, R. C. (2006). Supramembrane potential-induced electroconformational changes in sodium channel proteins: A potential mechanism involved in electric injury. *Burns*, *32*(1), 52–59. https://doi.org/10.1016/j.burns.2005.08.008

Electricity with high energy force permanently alters the way the body uses sodium, causing acquired channelopathy.

Chen, R., Li, Y. J., Li, J. Q., Lv, X. X., Chen, S. Z., Li, W. Z., … Li, X. Y. (2011). Electrical injury alters ion channel expression levels and electrophysiological properties in rabbit dorsal root ganglia neurons. *Burns*, *37*(2), 304–311. https://doi.org/10.1016/j.burns.2010.08.006

Electricity with high energy force permanently alters ion channels causing electrophysiological changes of voltage-gated potassium (Kv) and sodium (Nav) channels.

Hjaeresen, M.-L., Hageman, I., Wortwein, G., Plenge, P., & Jørgensen, M. B. (2008). Chronic Electroconvulsive Stimulation but Not Chronic Restraint Stress Modulates mRNA Expression of Voltage-Dependent Potassium Channels Kv7.2 and Kv11.1 in the Rat Piriform Cortex. *Brain Research*, *1217*, 179–184. https://doi.org/10.1016/j.brainres.2007.09.071

Repeated ECT use permanently alters how potassium is used on a cellular level.

Kragh, J., Seidelin, J., & Bolwig, T. G. (1993). Seizure threshold to lidocaine is decreased following repeated ECS (electroconvulsive shock). *Psychopharmacology*, *111*, 495–498. https://doi.org/10.1007/BF02253542

The more exposure to electro-shock, the lower the seizure threshold becomes when exposed to sodium blockers. The greater the amount of treatments exposed, the easier it is to inadvertently cause a major motor seizure and kill a patient using a normal dose of a sodium-blocker.

Lee, R. C. (2005). Cell Injury by Electric Forces. Annals of the New York Academy of Sciences, 1066, 85–91. https://doi.org/10.1196/annals.1363.007

"These voltage sensors are obviously susceptible to an applied intensive electric field. Tsong and Teissie examined the effects of a strong field pulse exposure on the membrane protein Na/K ATPase in erythrocytes. After using a microsecond pulsed intense electric field to shock red blood cell membranes, they found that in a low ionic medium at least 35% of membrane pores induced by the shock pulse were linked to channels in denatured Na/K ATPase in the cell membrane. They attributed this ionic leakage to the electroporation of the Na/K ATPase. In a series of experiments performed to study the effects of field-induced large transmembrane potentials on ion channel proteins in skeletal muscle fibers, both the open channel currents from the voltage-dependent Na+ channels and the delayed rectifier K+ channels were reduced by an intense electric shock. An electric shock by a single 4-ms, −500-mV pulse may decrease about 20% of the Na+ channel currents and 30% of the delayed rectifier K+ channels currents. The channel conductance of the delayed rectifier K+ channels were also substantially reduced by the above electric shock. The decrement varies from 10 to 40% depending on the type of cells."

Leibovici, D., Shemer, J., & Shapira, S. C. (1995). Electrical injuries: current concepts. Injury, 26(9), 623–627. https://doi.org/10.1016/0020-1383(95)00130-2

"Respiratory arrest is usually a combined effect of tetanic [(Ca)] paralysis of the respiratory muscles and damage to the respiratory centers in the brain stem."

Pei, Q., Burnet, P. W., Grahame-Smith, D. G., & Zetterström, T. S. (1997). Differential effects of acute and chronic electroconvulsive shock on the abundance of messenger RNAs for voltage-dependent potassium channel subunits in the rat brain. *Neuroscience*, *78*(2), 343–350. https://doi.org/10.1016/s0306-4522(96)00574-x

ECT alters the way potassium is used at a cellular level.

Streck, E. L., Feier, G., Búrigo, M., Franzon, R., Dal-Pizzol, F., Quevedo, J., & Wyse, A. T. S. (2006). Effects of electroconvulsive seizures on Na+,K+-ATPase activity in the rat

hippocampus. *Neuroscience Letters*, *404*(3), 254–257. https://doi.org/10.1016/j.neulet.2006.06.002

"Results showed an inhibition of Na+,K+-ATPase activity in the hippocampus 48 h, 7, 30, 60 and 90 days after a single electroconvulsive shock. Chronic treatment diminished the enzyme activity in the hippocampus 7 and 30 days after electroconvulsive (ECS) sessions. Our findings demonstrated that Na+,K+-ATPase activity is altered by ECS."