Neuromuscular disease

Neuromuscular disease is a very broad term that encompasses many diseases and ailments that impair the functioning of the muscles, either directly, being pathologies of the voluntary muscle, or indirectly, being pathologies of nerves or neuromuscular junctions.

Neuromuscular diseases are those that affect the muscles and/or their direct nervous system control, problems with central nervous control can cause either spasticity or some degree of paralysis (from both lower and upper motor neuron disorders), depending on the location and the nature of the problem. Some examples of central disorders include cerebrovascular accident, Parkinson's disease, multiple sclerosis, Huntington's disease and Creutzfeldt–Jakob disease. Spinal muscular atrophies are disorders of lower motor neuron while amyotrophic lateral sclerosis is a mixed upper and lower motor neuron condition.

see Neuromuscular spine deformity.

Burn-induced neuromuscular dysfunction may contribute to long-term morbidity; therefore, it is imperative to develop novel treatments. The present study investigated whether erythropoietin (EPO) administration attenuates burn-induced motor neuron apoptosis and neuroinflammatory response. To validate our hypothesis, a third-degree hind paw burn rat model was developed by bringing the paw into contact with a metal surface at 75°C for 10 s. A total of 24 male Sprague-Dawley rats were randomly assigned to four groups: Group A, sham-control; Group B, burn-induced; Group C, burn + single EPO dose (5000 IU/kg i.p. at D0); and Group D, burn + daily EPO dosage (3000 IU/kg/day i.p. at D0-D6). Two treatment regimens were used to evaluate single versus multiple doses treatment effects. Before sacrifice, blood samples were collected for hematological parameter examination. The histological analyses of microglia activation, iNOS, and COX-2 in the spinal cord ventral horn were performed at week 1 post-burn. In addition, we examined autophagy changes by biomarkers of LC3B and ATG5. The expression of BCL-2, BAX, cleaved caspase-3, phospho-AKT, and mTOR was assessed simultaneously through Western blotting. EPO administration after burn injury attenuated neuroinflammation through various mechanisms, including the reduction of microglia activity as well as iNOS and COX-2 expression in the spinal cord ventral horn. In addition, the expression of phospho-AKT, mTOR and apoptotic indicators, such as BAX, BCL-2, and cleaved caspase-3, was modulated. Furthermore, the activity of burn-induced autophagy in the spinal cord ventral horn characterized by the expression of autophagic biomarkers, LC3B and ATG5, was reduced after EPO administration. The present results indicate that EPO inhibits the AKT-mTOR pathway to attenuate burn-induced motor neuron programmed cell death and microglia activation. EPO can modulate neuroinflammation and programmed cell death and may be a therapeutic candidate for neuroprotection [1)].

Rhizotomy is a neurosurgical procedure that selectively destroys problematic nerve roots, most often to relieve pain symptoms or neuromuscular diseases.

1)

Wu SH, Lu IC, Lee SS, Kwan AL, Chai CY, Huang SH. Erythropoietin attenuates motor neuron programmed cell death in a burn animal model. PLoS One. 2018 Jan 31;13(1):e0190039. doi: 10.1371/journal.pone.0190039. eCollection 2018. PubMed PMID: 29385149.