Secondary Injury	Description	Pharmaceutical Agent/Treatment Used to Counteract Injury
Inflammation	Swelling at the injury site. Dead cells attract inflammatory cells such as macrophages, neutrophils, and microglia, which in turn release pro-inflammatory cytokines at the site of injury.	 Methylprednisolone Dexamethasone Minocycline Erythropoietin Granulocyte-colony stimulating factor Cethrin®
Hemorrhage	Initial injury results in bleeding within the grey matter, which leads to hemorrhagic death of afflicted cells.	Methylprednisolone
Ischemia	Blood flow is restricted from the spinal cord and surrounding tissues. Hypoxia results in cell death.	 Methylprednisolone Naloxone Nimodipine Erythropoietin Thyrotropin-releasing hormone
Edema	Swelling and fluid build-up around the spinal cord. Can be the result of initial trauma, ischemia, and excitotoxicity.	MethylprednisoloneRiluzole
Excitotoxicity	Neuronal damage caused by overstimulation, produced by high levels of calcium ions and glutamate.	 Riluzole Minocycline Erythropoietin GM-1 ganglioside Thyrotropin-releasing hormone
Lipid peroxidation	Reactive oxygen species steal electrons from neuron cell membranes, resulting in membrane lysis and cell death.	 Methylprednisolone Tirilazad mesylate Erythropoietin Minocycline Riluzole
Apoptosis	Programmed cell death of neurons due to presence of cytokines and reactive oxygen species.	 Methylprednisolone Erythropoietin GM-1 ganglioside Granulocyte-colony stimulating factor Minocycline
Axon demyelination	Damaged oligodendrocytes cause demyelination of neurons. Exposed axons are susceptible to damage from reactive oxygen species.	 Granulocyte-colony stimulating factor GM-1 ganglioside Cethrin® Erythropoietin
Neurogenic shock	Normal sympathetic nervous system functioning is disrupted, leading to hypotension and bradycardia.	Established treatments for bradycardia, hypotension, and hypothermia