Public Abstract
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The Effects Of Axotomy On The Biophysical Properties Of Reticulospinal Neurons In Larval Lamprey
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In all vertebrates, neurons in the brain project descending processes (axons) to the spinal cord to initiate motor activities, such as locomotion. Immediately after a severe spinal cord injury (SCI), such as a complete transection, these descending axons are injured (axotomy), and animals are paralyzed below the lesion. Adult higher vertebrates, such as birds and mammals, exhibit little to no axonal regeneration of descending axons, and there is minimal recovery of voluntary motor function below the lesion (Schwab and Bartholdi, 1996). In contrast, following SCI in lower vertebrates, such as the lamprey, brain neurons, including reticulospinal (RS) neurons, regenerate their descending axons through the lesion site, and locomotor function recovers in a few weeks (McClellan, 1992, 1994; also see Rovainen, 1976).

Following axotomy (axonal injury) of large, identified RS neurons (Müller cells) in the lamprey, most studies examined anatomical or molecular changes in cell properties. Following SCI in larval lamprey, RS neurons undergo a number of changes in their electrical properties (McClellan et al. 2002; McClellan, 2003). Based on the previous information from our laboratory, we hypothesize that transections in the upper spinal cord will alter the electrical properties of axotomized RS neurons to a greater degree than transections in the lower spinal cord. Furthermore, we were interested in whether some of the changes in electrical properties of injured RS neurons allows these neurons to regenerate their axons.

Following transections in the upper spinal cord, the majority of injured RS neurons displayed a number of changes in electrical properties that are typical of injury. In contrast, following transections in the lower spinal cord, the majority of injured RS neurons displayed electrical properties typical of normal, uninjured RS neurons. Therefore, the present study suggests that transections in the upper spinal cord are not only a stronger stimulus for axonal regeneration of RS neurons than transections in the lower cord but also alter the electrical properties to a greater degree. Furthermore, in injured RS neurons, the amplitude and shape of the main part of the action potentials were largely unchanged, but a delayed component, called the fast (slow) afterhyperpolarization (fAHP, sAHP) was significantly larger (smaller) than in uninjured RS neurons. The sAHP is produced by calcium entering RS neurons during the action potential. Our laboratory has shown that low intracellular calcium concentrations are necessary for axonal growth. Therefore, injured RS neurons may undergo a number of changes in their electrical properties to maintain relatively low intracellular calcium concentrations and to promote axonal regeneration. If the specific changes in electrical properties of RS neurons that promote axonal regeneration can be identified in lamprey, this information may be helpful in understanding why neurons in higher vertebrates, including humans, are unable to regenerate following SCI.