Do we know how the spinal interneuronal network interacts with descending motor commands?

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Much research into human motor control is biased towards understanding the complex functions of motor areas of the cerebral cortex and decoding the patterns of output that are produced during different types of voluntary movement. However the vast majority of descending connections to spinal cord terminate on interneurones that may be one or more synapses distant from motoneurones. We have surprisingly little information about the detailed anatomical connectivity of this network of neurones and of the functions that it can perform. A long history of reflex studies has tended to focus on the phasic response to transient inputs and their modulation during different types or phases of movement. More recent work has highlighted the unexpected properties of the network in response to continuous inputs. The net result is that we now see the spinal interneuronal circuitry as a complex network of connections that can transform the inputs it receives from brain and peripheral nerve into patterns of motor output.

In the context of regenerative strategies to restore function after spinal injury, two major questions emerge. First, how will the intact interneuronal circuitry below the lesion adapt to the changed patterns of input (both peripheral and central) that it receives? Reflex studies have shown that there are large changes in sensitivity of phasic responses which give some clues as to the shifts in network function that occur. The second question is how this changed network will respond to any remaining (or new, following spinal cord repair) inputs from the brain? For example, even if perfect connectivity were re-established would function return to normal, or would reorganisation have gone too far to be reversed? Indeed, any connections that are restored after treatment will be only a fraction of those that were damaged, and their connections may be distributed aberrantly in the cord. What types of output will such inputs be able to recruit from the spinal circuits, how stable will these patterns be, and what varieties of physical training will be required to shift patterns of network organisation to optimise function?

Some insight into the operations provided by spinal circuits comes from examining the computational transformations that have to be applied to recordings from cortical neurones when brain-machine interfaces activate muscles to produce limb movements. Other insights come from studying how the spinal circuits respond to sustained sensory inputs (as opposed to the transient inputs traditionally used to test reflex function). Finally, if therapy does restore some functional connections from brain to cord it is highly unlikely that they will originate from their original sources. At present it is unknown how well the brain will be able to reorganise in humans to adapt to new patterns of connectivity. However, recent brain mapping studies in spinal injury suggest that it may be much less widespread than in rodent models. I will review these questions with particular emphasis on what is known in intact and lesioned humans.