

Neurotechnology

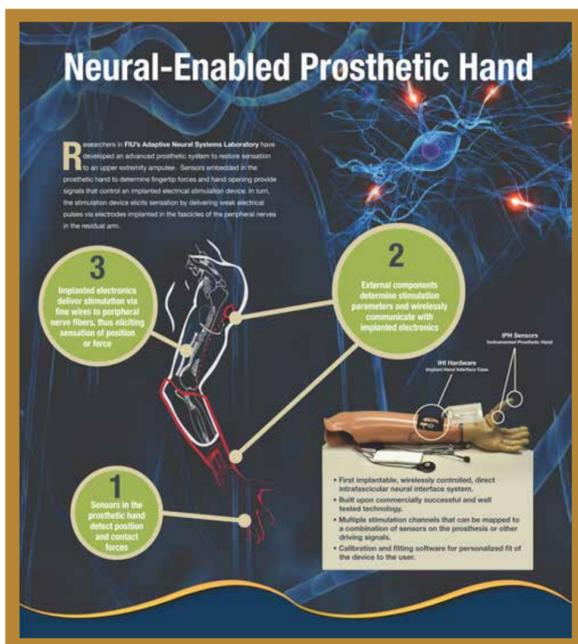
for repair and restoration of function lost to trauma

FIU's College of Engineering and Computing is at the forefront of creating neurotechnology to restore function lost to trauma. The Department of Biomedical Engineering currently has the following projects underway:

Neural-Enabled Prosthetic Hand System

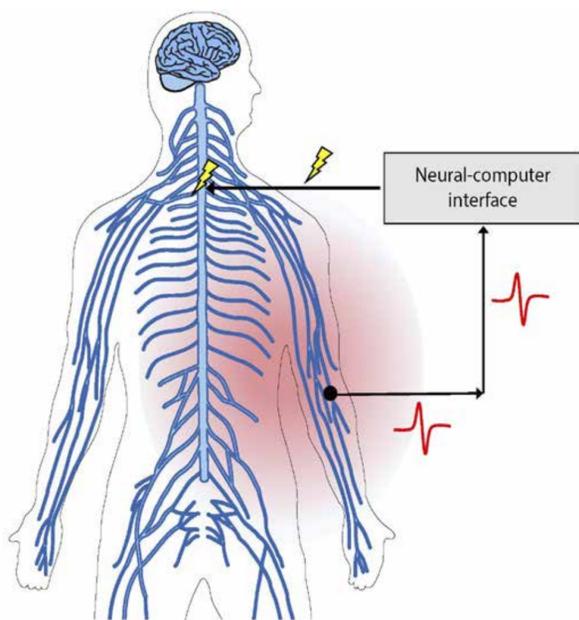
For individuals with upper extremity amputation, even though a motorized prosthesis can enable active grasping capabilities, many individuals choose not to use such devices. One of the primary deficiencies of current myoelectric prostheses is that they do not provide the user with sensation that can convey information about grasp force or hand opening. This limits the user's ability to manipulate objects and places high demands on attention from the user.

Professor Ranu Jung and her Adaptive Neural Systems Laboratory team and industrial partners have developed an advanced prosthetic system to restore sensation to upper extremity amputees. Sensors embedded in the prosthetic hand detect hand opening position and grip forces when coming in contact with an object. Signals from the sensors control an implanted electrical stimulation device that elicits sensation by delivering low-level electrical pulses via fine-wire electrodes thinner than human hair. The electrodes are implanted parallel to the nerve fibers in the fascicles of the peripheral nerves in the residual arm. The neural-enabled prosthetic hand system is intended to improve the user's ability to manipulate objects and to detect object size and firmness. This is the first implantable, wirelessly controlled, direct intrafascicular neural interface system. Recently, the group received approval from the FDA to initiate a first-in-human study with this neural-enabled prosthesis.



Alleviation of Pain by Facilitating Neural Plasticity

Neuropathic pain resulting from spinal cord injury (SCI) impacts up to 80 percent of individuals with SCI. In addition to its direct functional, psychosocial, and economic consequences, SCI-related neuropathic pain also contributes to decline in functional mobility through its negative impact on overall physical activity level and participation in motor rehabilitation. Pressure ulcers, cardiovascular decline, and metabolic changes are leading causes of morbidity in SCI that can all be caused or exacerbated by limited activity.



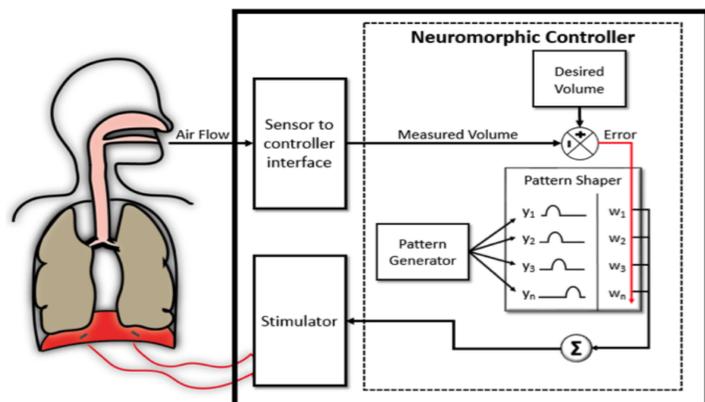
The overall goal of Jacob G. McPherson's research is to develop a fully implantable neuroprosthetic device that seamlessly integrates with natural, ongoing neural activity and improves upon the limited, temporary pain relief offered by conventional therapies. The cornerstone of his approach is that precise timing of activity amongst interconnected neurons can dramatically modify how the neural circuit functions. This phenomenon is known as activity-dependent neural plasticity, and is thought to underlie many of the clinical benefits realized by state-of-the-art physical therapy interventions. While activity-dependent neural plasticity is the basis for our nervous system's unique ability to learn, adapt, and reorganize, it can also become maladaptive after neurological injury. In fact, it is believed to play a large role in the development and persistence of neuropathic pain post-SCI.

McPherson and his team are developing closed loop neural-computer interface technologies to harness and exploit the potent effects of activity-dependent neural plasticity. They decode pain-related neural activity from the peripheral nerves and use this information to trigger focal electrical stimulation in specific regions of the spinal cord. Over time, such synchronization of neural activity can decrease the sensitivity of spinal pain-processing neurons, providing meaningful, long lasting relief from SCI-related neuropathic pain that does not rely on opioid analgesics.

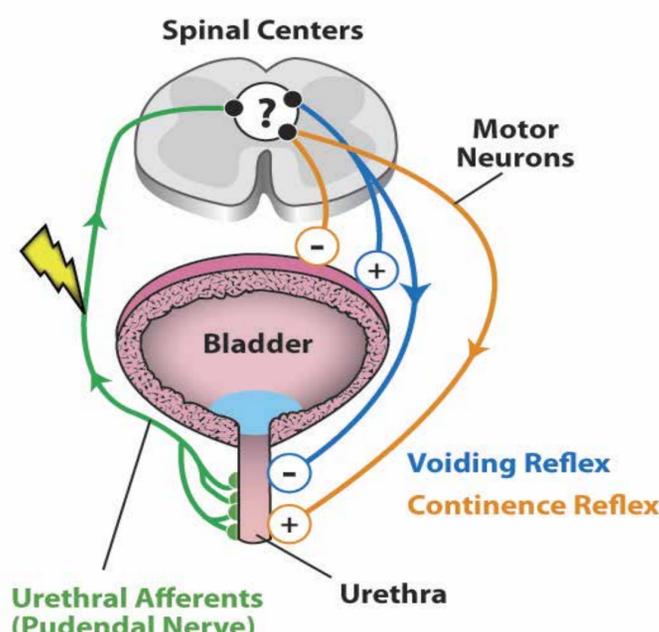
Restoration of Ventilation after Spinal Cord Injury

Spinal cord injury at the cervical level can cause damage to the descending respiratory pathways, which can lead to a significant reduction in ventilatory capabilities. Individuals with impaired ventilatory capacity are often dependent on mechanical ventilation for ventilatory support. Electrical pacing of the diaphragm has been demonstrated as an alternative viable approach for ventilatory support following spinal cord injury. Commercially available ventilatory pacing devices require initial manual specification of stimulation parameters and frequent adjustment to achieve and maintain suitable ventilation and ventilatory efficiency over long periods of time.

Ranu Jung, Brian Hillen and colleagues, have developed a closed-loop neuromorphic controller capable of meeting the ventilatory demands of the user despite diaphragmatic fatigue, changes in electrode properties, as well as other extrinsic factors. Their controller uses real-time dynamic lung volume and expired CO₂ to adapt stimulation parameters based on ventilatory need.



Restoration of Bladder Function after Spinal Cord Injury



Under typical circumstances, the urinary bladder sends information to the brain (such as how full it is), and the brain sends command signals back to the bladder about when to initiate a void. However, after a spinal cord injury, this back-and-forth communication between brain and bladder is severely disrupted. The result is that the bladder is no longer able to contract to void its contents, urine begins to accumulate, and catheterization is required to prevent damage to the rest of the urinary tract. In most cases, some form of reflex bladder contraction emerges during recovery from the initial spinal injury. However, this newly developed "compensatory reflex" is weaker than the original reflex, and it does not act constructively with a relaxation of the urethral sphincter to void the bladder effectively. The compensatory reflex also may emerge too late to prevent negative changes that occur to the bladder following the initial spinal injury, such as harmful stretching from urine accumulation, and a toughening of the bladder wall that makes it more difficult to contract. Zachary Danziger's goal is to better understand the development of the compensatory reflex in quantitative terms.

By learning when and how the new reflex emerges, he and his team hope to provide clinicians with a better way to manage the initial bladder dysfunction that follows spinal injury. Another goal is to try to increase the effectiveness, control, and safety of the compensatory reflex using therapeutic electrical stimulation of peripheral nerves following spinal cord injury. This may strengthen the new reflex, potentially reduce the required frequency of intermittent catheterization, and prevent some lasting damage to the bladder and urinary tract.