Spinal cord trauma essay

Abstract

Loss of sensory and motor function below the injury site is caused by trauma to the spinal cord. Approximately 10, 000 people experience serious spinal cord injury each year. There are four general types of spinal cord injury, cord maceration and laceration, contusion and solid core injury. There are three phases of SCI response that occur after injury: the acute, secondary, and chronic. The most immediate concern is patient stabilization. Additionally interventions may be instituted in an effort to improve function and outcome. Through health, and future development one day there will be hope for recovery from the spinal cord injury.

Introduction

Loss of sensory and motor function below the injury site is caused by trauma to the spinal cord. As indicated by Huether & McCance (2008) normal activity of the spinal cord cells at and below the level of injury ceases due to loss of continuous tonic discharge from the brain and brain stem. Depending on the extent of the injury reflex function below the point of injury may be completely lost. This involves all skeletal muscles, bladder, bowel, sexual function and autonomic control. In the past hope for recovery has been minimal. With medical advancements and better understanding today hope for recovery is better but still limited.

Risk Factors and Incidence

According to Huether & McCance (2008) approximately 10, 000 people experience serious spinal cord injury each year. 81% of those injuries are males with an average age of 33. 4 years. As indicated by Hulsebosch (2002) the majority of injuries are divided into four separate groups; 44% of the

injuries are young people sustained through motor vehicle crashes or other high energy traumatic accident; 18% are sustained through sports activities, and 24% are sustained through violence and 22% are sustained in the elderly population either through falls or cervical spinal stenosis caused by congenital narrowing or spondylosis.

Categories of Injury

According to Hulsebosch (2002) there are four general types of spinal cord injury: 1) cord maceration 2) cord laceration 3) contusion injury, and 4) solid cord injury. In the first two injuries, the surface of the cord is lacerated and a prominent connective tissue response is invoked, whereas in the latter two the spinal cord surface is not breached and the connective tissue component is minimal. The contusion injury represents from 25 to40% of all injuries and is a progressive injury that enlarges overtime.

Cellular Level Physiology

Hulsebosch (2002) gives us three phases of response directly after the injury of the spinal cord. The acute phase begins with the moment of injury and extends for the first few days. A variety of pathophysiological processes begins. There is immediate mechanical soft tissue damage, including endothelial cells of the vasculature. Cell death, resulting from mechanical forces and ischemic consequences is instantaneous.

Over the next few minutes there are significant electrolytic shifts, intracellular concentrations of sodium increase. Extracellular concentrations of potassium increase. Intracellular levels of calcium increase to toxic levels that contribute to a failure in neural function. Electrolyte shifts cascade to a generalized demonstration of spinal shock, which is representative of a “

failure of circuitry in the spinal neural network. As indicated by Shewmon (1999) spinal shock is a transient functional depression of a structurally intact cord below the site of an acute spinal cord injury.

It does not occur with slowly progressive lesions. Limited function or loss of function typically lasts two to six weeks followed by recovery of functions. The secondary phase occurs over the next few minutes to the next few weeks. Ischemic cellular death, electrolytic shifts, and edema continue. As a result of cell lysis extracellular concentrations of glutamate and other amino acids reach toxic concentrations within the first fifteen minutes after injury.

Free-radical production amplifies. Neutrophils accumulate in the spinal parenchyma within 24 hours. Lymphocytes follow the neutrophils and reach their peak numbers within forty eight hours. Local concentrations of cytokines and chemokines increase as part of the inflammation process. As inflammation and ischemia proceed the injury site grows in size from the initial mechanical force response site into the area around the site, encompassing a larger region of cell death.

Regeneration is inhibited by factors expressed within the dominos of responsive reactions. The chronic phase occurs over a time course of days to years. Cell death continues. The cord becomes scarred and tethered. Conduction deficits result from demyelination of the cord. Regeneration and emergence of axons is exhibited but inhibitory factors suppress any resultant growth. Alteration of neural circuits often results in chronic pain syndromes for many spinal cord injury patients.

Therapeutic Management

Spinal cord injury is diagnosed by physical examination, radiological exam, CT scans, MRI scans, and myelography. The most immediate concern in the management of an acute spinal cord injury is patient stabilization. The vertebral column is subject to surgical stabilization using variety of surgical rods, pins, and wires.

Hardware must be meticulously placed. Surgical intervention has the potential to instigate additional spinal trauma. Hemostatic body systems must be supported through fluid resuscitation, medication management and electrolyte support. Additionally the following interventions may be instituted in an effort to improve function and outcome:

Edema Reduction

Reduction of the inflammatory response is one intervention of concentrating in the treatment of the acute spinal cord injury. Steroids have provided a primary tool to reduce edema and inflammation, the most successful of which is methylprednisolone (MP). According to Bracken (1993) the administration of a high dose of MP, if given within eight hours of the insult in patients with both complete and incomplete SCI, as proposed by the National Acute Spinal Cord Injury Study (NASCIS-2), has been promising with respect to improved clinical outcome. The cellular and molecular mechanisms by which MP improves function may involve antioxidant properties, the inhibition of inflammatory response, and/or a role in immunosuppression.

Inhibition of Inflammation: by use of Anti-Inflammatory Agents

Although inflammation is generally held to be are pair mechanism that is restorative in nature, recent work has demonstrated that the inflammatory

cascade produces several pathways that are degradative in nature, such as the prostagland in pathways.

Anti-inflammatory agents have been administered with successful limitation of the inflammatory process. As indicated by Hains, Yucra and Hulsebosch (2001) selective cyclooxygenase (COX)-2inhibitors given systemically to spinal card injury patients have demonstrated significant improvements. Provision of inhibition of the enzyme activation sequence appears to be the safest medication action at this time.

Application of either whole body hypothermia or local cord cooling appears to hold promise for those suffering from neuro trauma. Application of hypothermia, either spinally or systemically, is thought to provide protection for neural cells and to reduce secondary inflammation, decreasing immediate mortality. According to Hayes, Hsieh, Potter, Wolfe, Delaney, and Blight (1993) local spinal cord cooling within eight and a half hours of injury in ten patients produced a better-than-expected rate of recovery of sensory and motor function.

Rescue from Neural Cell Death

Cells die due to a programmed cell death after SCI. An excellent opportunity is present for intervention with factors that could rescue the cells at risk. As presented by Eldadah and Faden (2000) one approach to cell rescue is the inhibition of caspases. Caspases are regulated signalling proteases that that accomplish a primary role in mediating cell apoptosis thru division at specific sites within proteins. These proteins inhibit programmed cell death and are a part of the bcl-2 oncogene products. According to Shibata, Murray,

Tessler, Ljubetic, Connors and Saavedra (2000) recent work has demonstrated prevention of retrograde cell loss and atrophy reduction by direct intra-spinal administration of the Bcl-2 protein into the damaged site.

Another group of proteins with potential cell death inhibition properties are calpains. Calpains are calcium-activated proteases that assist in degradation of cytoskeletal demolition of injured cells. Substances with calpain inhibitor properties could prove of benefit in reduction of cell death.

Demyelination and Conduction

According to Waxman (2001) the strategy of inhibiting the neural injury induced by the increased barrage of action potentials early in the injury phase or by inhibiting the voltage-dependent sodium channels, which provide the ionic basis for the action potential may be beneficial. In addition, neural injury and disease may introduce altered ionic channel function on nerve processes that would result in impaired conduction properties, which produces persistent hyperexcitability leading to the basis for chronic pain after CNS neural trauma.

As a result of secondary injury to the spinal cord many axons are demylinated. Infusion of a fast, voltage-sensitive potassium channel blocker may provide partial restoration of conduction properties to demylinated axons. As presented by Guest, Hiester and Bunge (2005) another strategy for the improvement in demyelinationis the transplantation of Schwann cells which may contribute to the restoration of myelin sheaths around some spinal axons.

Promotion of Axonal Regeneration

During development of the central nervous system, an assortment of axonal growth promoting proteins are present in the extracellular environment. The environment stimulates axon growth and neural development. Once the central nervous system is established the growth stimulating agents decline. The adult central nervous system shifts toward inhibition of axonal growth permitting a stable and circuitry. These inhibition and stimulatory factors provide an opportunity for research that will promote axonal growth after a spinal cord injury perhaps rebuilding a neural communication network.

Cell Replacement Strategies

After spinal cord injury function of nerve cells and cells that produce myelin that insulates and provides a positive impulse conduction venue has vanished. Cellular replacement to rebuild conduction properties is a promising therapy. As indicated by Normura, Tator and Shoichet (2006) there is promise that technology utilizing cellular treatment procedures including olfactory ensheathing cells, (the cells that form the myelin on olfactory nerves),

Schwann cells (the cells that form the myelin on peripheral nerves), dorsalroot ganglia, adrenal tissue, and neural stem cells can promote repair of the injured spinal cord. It is postulated that these tissues would rescue, replace, or provide a regenerative pathway for injured adult neurons, which would then integrate or promote the regeneration of the spinal cord circuitry and restore function after injury. As indicated by Nakamura (2005) there is promise that bioengineering technology utilizing cellular treatment advances

can promote repair of the injured spinal cord. Transplantation of these cells promotes functional recovery of locomotion and reflex responses.

The engineering of cells combines the therapeutic advantage of the cells along with a delivery system. For example, if delivery of neurotrophins (neuro- related to cell nerves, tropin- a turning) is desired, cells that secrete neutrophins and cells that create myelin can be engineered to stimulate axon growth and rebuild nerve function.

In an effort to further enhance beneficial effects autoimmune agents such as macrophages can be extracted from the patient's own system and inserted at the injury site. The patient's own activated macrophages will scavenge degenerating myelin debris, rich in non-permissive factors, and at the same time encourage regenerative growth without eliciting an immune response.

Retrain the Brain with Aggressive Physical Therapy

It is apparent that recovery of locomotion is dependent on sensory input that can " reawaken" spinal circuits and activate central pattern generators in the spinal cord, as demonstrated by spontaneous " stepping" in the lower limbs of one patient. According to Calancie, Alexeeva, Broton and Molano (2005) it may take six or more months for reflexes to appear following acute SCI suggesting they might be due to new synaptic interconnections.

Electrical Stimulation

Functional electrical stimulation (FES) that contributes to improved standing can improve quality of life for the individual and the caregiver. There is considerable interest in computer-controlled FES for strengthening the lower extremities and for cardiovascular conditioning, which has met with some

success in terms of physiological improvements such as increased muscle mass, improved blood flow, and better bladder and bowel function. With added benefit there are decreases in medical complications such as venous thrombosis, osteoporosis, and bone fractures. Stimulation of the phrenic nerve, which innervates the diaphragm, is used in cases where there is damage to respiratory pathways.

Chronic Central Pain

As indicated by Siddall & Cousins (1997) pain continues to be a significant problem in patients with spinal cord injuries. There is little consensus regarding the terminology, definitions and nature of the pain. Treatment studies have lacked congruence due to inaccurate identification of pain types. There has been little progress in efforts to bring an understanding of the pathophysiology of CCP to the development of therapeutic approaches for the SCI patient population.

Chronic central pain (CCP) syndromes develop in the majority of spinal cord injury patients. As indicated by Que, Siddall and Cousins (2007) chronic pain is a disturbing aspect of spinal cord injury, often interfering with basic activities, effective rehabilitation and the quality of life of the patient. Evidence that neurons in pain pathways are pathophysiologically altered after spinal cord injury comes from both clinical and animal literature. In addition, the development of the chronic pain state correlates with structural alterations such as intra-spinal sprouting of primary afferent fibres.

According to Que, Siddall and Cousins (2007) pain in the cord-injured patient is often resistant to treatment. Recognition of Chronic Central Pain has led to

utilization of non-opioid analgesics. According to Siddall and Middleton (2006) Baclofen, once used exclusively in treatment of spasticity and the anticonvulsant gabapentin originally used to treat epilepsy, have had some success with attenuating muskuloskeletal CCP syndromes. The tricyclic antidepressant amitriptyline has shown effective in treatment of dysesthetic pain.

Conclusion

Stem cell therapy will offer hope for spinal cord injury patients with opportunities for the abundance of cell replacement strategies. Advances in the field of electronic circuitry will lead to better FES and robotic devices. Pharmacological advances offer intervention direction to aid in recovery and improve patient's' quality of life every day. The re-establishment of cell, nerve and muscle communication interconnections will be potentially possible. Through tenacity, health, and future development one day victims of spinal cord injury will be told there is hope of recovery.

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