Transplantation 2020: New treatments for spinal cord injuries- Harry S Goldsmith- University of California, USA

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Abstract

Over the past half century there have been few therapeutic breakthroughs for the treatment of spinal cord injuries. It has been previously shown that following a Spinal Cord Injury (SCI), scar tissue develops at the site of injury through which axons are unable to penetrate to make appropriate neurological connections into the distal spinal cord. Over the past few years it has been reported that placing the momentum directly on a traumatized spinal cord can result in a favourable clinical result. The momentum has been shown to limit the development of scar tissue following SCI, which strongly suggests that omental transposition to an SCI now appears warranted to justify a carefully controlled evaluation of the benefits of omental application to an SCI. Spinal cord injuries are devastating events. Unfortunately, in spite of improved medical and surgical treatment of the condition, there has not been a significant improvement in the neurological results of these injuries during the past half century. The purpose of this paper is to suggest a surgical technique that might increase functional improvement in the future treatment of spinal cord injuries (SCI). A new approach to the problem seems warranted. Over a hundred years ago, Ramon y Cajal, the famous neuro-histologist, stated that axons are unable to connect to neural structures distal to an SCI based on his observations that axons cannot penetrate through connective tissue which routinely develops at the location of an SCI. A half century later, Dr. L.H. Freeman, confirmed Cajal's observations by showing that axons had the inherent capacity to grow distally within an injured spinal cord (SC), but their progress routinely encountered a scar barrier in which axons "simply form neuromata at the injury site". Based on these observations, if neuro-protection within an SCI is to be addressed, laboratory and clinical efforts should be increasingly aimed at preventing the development of scar tissue in an acute SCI and decreasing the scar barrier present during a chronic SCI.

Background:

In the late 1800's, Ramon y Cajal, Father of Neuropathology stated that the rationale patients who suffered a medulla spinalis injury (SCI) don't improve is that a scar develops at the location of the SCI which prevents axons from penetrating through the scar barrier. The aim of the study was to find out if a scar following an SCI might be surgically removed, followed by reconstruction of the medulla spinalis which could lead on to functional improvement following the injury.

Method:

Studies were administered within the laboratory to find out a way to ascertain if a bit of a medulla spinalis might be removed followed by subsequent functional improvement. It was found that when a neighbourhood of the medulla spinalis in animals might be surgically excised with reconstruction of the medulla spinalis being successfully performed followed by functional success.

Results:

It was learned in cats when a bit of medulla spinalis was removed, the medulla spinalis might be reconstructed by filling the medulla spinalis defect with collagen followed by the placement of an intact vascularized omentum directly on the underlying collagen connection. Not only was this possible, but a patient underwent excision of 1.6" of her medulla spinalis with subsequent ability to steer which was confirmed by video.

Conclusion:

It appears that chronic medulla spinalis injured patients may have within the future the power to possess the scar which is present during a chronic injury removed with expectations following a medulla spinalis reconstruction that functional return can occur.

Pathophysiology of an SC Injury it's important to know the pathophysiological events that happen following an SCI during which there's deposition of edema and blood that routinely occurs rapidly after injury within the SC. This is the results of leakage of edema and blood through the porous endothelial lining of damaged capillaries located mainly within the central gray matter of the SC. The edema fluid is rich in protein which features a high pressure that draws an increasing amount of edema fluid. As edema and blood accumulates within the injured area, it causes an in depth physical swelling of the medulla spinalis which is contained within its no yielding Dura covering and the surrounding bony spinal column , a condition that causes an increased interstitial pressure at the location of the SCI. As the edema expansion intensifies, the interstitial pressure within the medulla spinalis continues to extend, causing compression of veins within the area. This action leads to an elevated blood pressure which further enhances the capillary extrusion of edema fluid and blood elements from the porous blood vessels at the injury site. Under normal conditions, extracellular edema fluid within the medulla spinalis drains into perivascular spaces with eventual drainage into spinal fluid. This flow pattern is essential for edema fluid elimination since there are no lymphatic’s in the spinal cord. As the volume of edema accumulates in an injured SC, there is eventual blockage of the central canal, subarachnoid and subdural spaces so that the normal edema drainage system within the spinal cord is compromised. Compensatory hydraulics develops within the medulla spinalis following injury within the plan to decrease expanding edema accumulation by displacing edema fluid up and down the medulla spinals. This longitudinal fluid movement, however, cannot catch up on the increasing edema volume that develops within the injury area. As edema accumulation increases, it causes the interstitial pressure at the SC injury site to become excessive, causing capillary compression which eventually can diminish capillary perfusion to the purpose of total vascular occlusion. When this happens, there's irreversible damage to neural tissue unless circulation are often restored within hours of injury. The blood-spinal cord barrier normally prevents leakage of blood into uninjured medulla spinals. Following a medulla spinals injury, however, the blood-spinal cord barrier is broken and blood enters the medulla spinals. It becomes critical at this point to use a way to eliminate, or a minimum of lessen, the deleterious effect of the free blood within the SC since blood is known to be injurious to neural tissue. Blood which leaks from porous SC blood vessels after injury incorporates fibrinogen which may be a normal component of the blood mixture. Fibrinogen is a soluble plasma protein synthesized in the liver that is converted into fibrin in the presence of blood coagulation. The key to eliminating medulla spinals scarring could rest with the absorption of fibrinogen. The evidence for this is that fibrinogen is deposited in the spinal cord following injury, which in turn inhibits neuritis outgrowth by triggering an inhibitory signal transduction pathway to neurons. This inhibition of neuritis outgrowth by the presence of fibrinogen in the SC occurs as early as one day after injury, peaks at seven days, and decreases in the following weeks. These activities indicate the importance of absorbing fibrinogen as early after injury as possible since the neuritis

inhibition by fibrinogen may begin the process of spinal cord scarring. Absorbing fibrinogen within the blood of an SC may eventually convince be crucial to limiting scar formation within the injured SC through which axons cannot penetrate. The question is how blood with its fibrinogen component is often absorbed within the SC following injury.

Biography

Harry S Goldsmith is Clinical Professor of Neurological Surgery at the University of California in Sacramento. He has been a Full Professor of Surgery and Neurosurgery since 1970. He has written 260 published papers, has edited four surgical texts, and was the Editor of Goldsmith's Practice of Surgery in twelve volumes from 1976-1988. His main interest at present is in the treatment of Alzheimer's disease and in new treatment for acute and chronic spinal cord injuries using the omentum.

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