



Peripheral Nerve Surgery: A Journey (Presidential Oration: Transcript)

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Good morning, first of all, thanks to ISPNS for this privilege and opportunity to share some of my experiences in this field.

I thank Professor Gopalakrishnan for the kind introduction.

It has always been a matter of concern (for me) that neurosurgeons are neither trained nor practicing peripheral nerve surgery. They are content to let someone else do it.

The results of surgery for nerve repair take a long time to manifest. And then they are not always as expected or sometimes not even great. The answer is not always simple.

Let us try and understand some well-known and not so well-known facts.

- A nerve is mainly made up of connective tissue and approximately 15% of it consists fascicles. So, any injury incites scar tissue.
- All injuries are broadly either complete avulsion/clean-cut or incomplete (focal, diffuse, or skip lesion).
- Following injury, both antegrade and retrograde degeneration occurs followed by regeneration (if continuity with the neuron is preserved).
- Simultaneously, transneuronal degeneration occurs, which can extend to the brain and the spinal cord.

While evaluating a nerve injury, one must look not only for motor deficit or recovery but also for sensory loss and its recovery. Once a nerve injury occurs, cortical changes start almost simultaneously; the more complete the injury, the more the chances of mosaic recovery.

In brachial plexus injuries due to traction in road traffic accidents, multiple levels of critical injury occur to the nerves at the points of fixation.

Our initial work was to try and understand the role of the grafts in the regeneration process, specifically whether pre-treated allografts would facilitate faster “reconnections” by providing a pathway for regrowth of the proximal regenerating stump. Most of our studies have been on Sprague Dawley rats, chosen for their suitability in nerve regeneration research. Their sciatic nerve demonstrates remarkable regenerative capacity, enabling the limb to regain function within 4 to 6 weeks. The nerve was cut and a 2- to 3-cm piece was removed. The test animals underwent immediate resuturing with pretreated grafts or untreated grafts or delayed suturing. The histology of the nerve across the graft showed minimal inflammation, with good regrowth of fascicles with pretreated grafts.^{1,2} Then in another set, we wrapped the anastomotic site in amniotic membrane and noted less amount of fibrous reaction.³

We used fibrin glue instead of suturing for repair and found that without stay sutures the ends do not stay together and therefore there is discontinuity.^{4,5}

In another study, we placed stem cells from dental pulp at the anastomotic site of nerve repair. We found that dental pulp is a good source of stem cells. Also, stem cells facilitated repair.^{6,7}

Once we realized that the cerebral processes dictate to a major extent the outcomes, we used functional magnetic resonance imaging (fMRI; both resting state and task based)

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to look for the changes and try to track them with diffusion tensor imaging (DTI) and then quantify the white matter changes in the corpus callosal volumetry.⁸⁻¹⁴

The studies showed that following the neurotization the initial activation occurs in the area representing the donor nerve, and with time and further improvement of the recipient nerve's function, the activation shifts to the recipient nerve's representation.^{8,9}

The birth brachial plexus injuries were studied with corpus callosum volumetry.^{11,12} This was a novel study and the birth brachial plexus injuries were studied and a brain MRI with DTI and corpus callosal volumetry was done, and the cohorts consisted of traumatic brachial plexus injuries and head injuries in children. We also studied cognitive function of all the three groups.

The study of outcomes following neurotization with intercostal nerve for brachial plexus injury showed that best results were when the time to surgery was at 5.5 months with a single intercostal nerve as the donor.^{15,16}

While studying entrapment neuropathies, we realized that many patients had post-traumatic entrapments as well as those secondary to lesions such as Hansen's disease. One interesting observation about Hansen's disease was that during its active phase, the affected nerve's distribution area was numb, and the nerve often appeared thickened due to intraneural granulomas. After treatment, the same patients presented with severe pain along the distribution. One would think that the onset of ulnar nerve neuritis may signify treated Hansen's disease.¹⁷

The work on carpal tunnel showed that when a patient presents with severe pain in the distribution of the median nerve with atrophic thenar wasting, release of the flexor retinaculum controls the pain. Hence, release is indicated even when there is wasting, if nocturnal pain is dominant.¹⁸

Among the preventable nerve injuries, a regular presentation was injection palsies.^{19,20} Initially we used to see these patients very frequently. In the last few years, the referral or incidence has come down.

Children also present with animal bites and fracture-related nerve injuries.²¹

Children with storage disorders may benefit from release of the flexor retinaculum.²²

We hypothesized that the dysesthetic pain following brachial plexus injury is due to antidromic excitation and therefore should respond to DREZotomy. The fMRI studies on patients with dysesthetic pain following brachial plexus injury showed that denervation of the limb resulted in activation of the sensory cortex with spread to the adjacent motor cortex.^{23,24} We also found that the brachial plexus injuries caused decreased sweating.^{25,26}

Our clinical and academic research in peripheral nerve studies showed that the results of peripheral nerve repair need to be understood in the light of amazing changes that occur in the cortex almost simultaneously with the injury. These central changes prevent proper equivalent results following repair.

After spending hours on end to repair the injured nerve, one needs to wait for 6 months to 1.5 years before repair and reanimation can occur.

This may be one of the reasons why neurosurgeons are not giving adequate attention to peripheral nerve surgery. In the process, it is being done by others.

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Conflict of Interest

None declared.

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