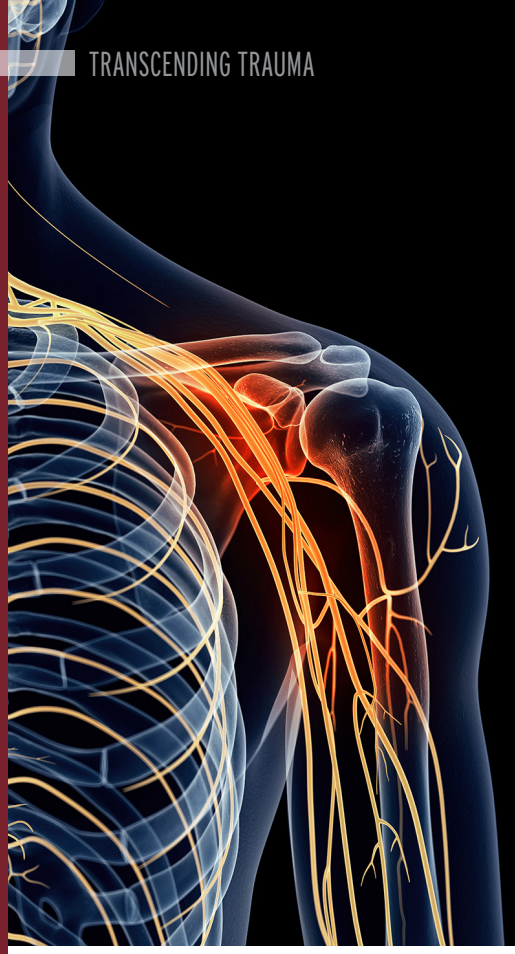

NEUROTRAUMA: BRACHIAL PLEXUS INJURIES

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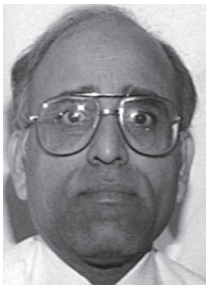

The practice of medicine is indeed a humbling experience. Decades ago, after having gained experience in various aspects of neurology, I used to believe that I could diagnose even the most complex disorders and manage most of them. One case that brought me down to the ground and taught humility was that of a 20-year-old who sustained a serious injury to his right brachial plexus after being thrown off from his motorcycle. His parents brought him for detailed evaluation a month after the injury. From clinical and electromyographic assessment, it appeared that the injury involved all three trunks of the brachial plexus. My neurosurgical colleague agreed to go ahead with the time-consuming procedure of using the patient's sural nerve as graft to reconnect the injured portions of the brachial plexus. I saw him three months after the surgery; it was disappointing to see no reinnervation even in the most proximal muscles like the deltoid. He did not turn up for the next scheduled EMG and I was totally shaken to learn that he took his life out of despair.

In the context of neurotrauma, next to the brain and the spinal cord, brachial plexus injuries lead to most devastating morbidity. I can recall many such patients and one common scenario is pan-plexus injury causing the so-called *flail arm*; often the first statement by the patient is "I wish you could cut off this useless arm." The frustration caused by sudden loss of function, especially in the dominant upper extremity can be severe enough to trigger suicidal thoughts as in the patient I described earlier. In the next few paragraphs, I will discuss various aspects of trauma to brachial plexus and the few new treatment options.

The brachial plexus is a highly complex structure and a nightmare to the medical student and the neurology resident alike. Questions like "What muscles are innervated by the upper trunk of the brachial plexus? What are the sensory branches from the cords? What is the first motor branch? What is a prefixed brachial plexus? How do you distinguish root avulsion from injury to the trunk?" are not uncommon in board examinations. Neurology examiners almost always have vignettes pertaining to the brachial plexus, often providing glee to the examiner and causing gloom to the examinee.

What causes brachial plexus injury? During my neurology residency, almost half a century ago, the most common cause used to be perinatal injury, usually from the use of forceps during labor. The reported incidence is between 0.42-5.1 per 1,000 live births,[1] providing a gold mine for the attorneys. Statistical data indicates that 75% of infants recover completely within the first month; 25% have permanent impairment. In the later years, I saw an increasing number of injuries from motor vehicle accidents; especially being thrown from a motorcycle and landing in a way that causes forcible movement of the shoulder down and away from the neck resulting in upper plexus palsy. More recently, an alarming number of gunshot injuries to the brachial plexus occur, a sign of the epidemic

of gun violence plaguing this country. Another important cause is iatrogenic injury related to positioning, regional anesthesia and surgical procedures, especially at the shoulder.

The clinical features of brachial plexus trauma depend upon the part of the plexus that has sustained the injury. It may affect the upper plexus (Erb's palsy) or the lower plexus (Klumpke's palsy) or the entire plexus. From the surgical point of view, it is crucial to know if the injury is at the nerve root level (root avulsion), which is proximal to the dorsal root ganglion (preganglionic injury), carries a poor prognosis for spontaneous recovery and tends to be non-operative. In such lesions, since the cell body of the sensory neuron is in continuity with the nerve, although the patient has no perception of sensations, paradoxically the sensory nerve action potential (SNAP) is intact on nerve conduction studies. On the other hand, in an injury distal to the dorsal root ganglion (postganglionic), the axons in the nerve are disconnected from the cell body and hence the SNAPS are absent. Root avulsions can be further substantiated by the use of sensory evoked potentials and by imaging studies.

There are different grades of injuries with differing prognosis. Injuries to the nerves can cause loss of the insulating myelin sheath (demyelination) leading to conduction block (neuropraxia) or disruption of axonal continuity (axonotmesis) or severance and discontinuity of the nerve (neurotmesis). Both axonal and nerve discontinuity set in motion a cascade of events resulting in Wallerian degeneration (named after Augustus Waller)[2] and eventually denervation atrophy of muscles. Reinnervation of muscles may occur by a process of axonal regrowth (1mm/day) in cases of complete injury or by axon terminal sprouting in partial injury. The existing intact axon terminal tends to reinnervate the denervated muscle fibers (much faster than axonal regrowth) leading to quicker restoration of function. The traditional surgical repair consisted of reconnecting the severed ends in cases of neurotmesis or use of nerve grafts from sural nerve of the patient or by the use of collagen tubes. These procedures took long hours and often the results were not impressive except in the very young.

Let us see what is in the horizon in the field of nerve repair. There are three areas of translational research that hold promise.

Since Wallerian degeneration is the underlying process after axonal injury, leading to denervation atrophy of muscles, can it be postponed or avoided? Recent discovery of genetic mutations that delay Wallerian degeneration (slow Wallerian degeneration in mice in which transected axons do not degenerate for weeks[3]) has provided unique insight into the process and holds promise for significant advances in treatment. It appears that a chemical, nicotinamide mononucleotide adenyl transferase 2 (NMAT2) is essential for axon growth and survival; loss of it may be the trigger for Wallerian degeneration.

Unlike the central nervous system, the peripheral nervous system possesses intrinsic capability to regenerate, as axons can regrow over long distances to reach their final target and Schwann cells are able to remyelinate them. But the rate of growth is slow. Can we hasten this process and also avoid misdirection of axonal growth? An interesting observation is that brief low frequency electric stimulation has been found to be capable of accelerating axonal regrowth.[4] This is a topic of active research and may provide future guidelines for intra and postoperative electric stimulation during nerve repair.

There is growing enthusiasm about the use of nerve transfers to provide quicker reinnervation of denervated muscles. The slow regrowth of axons often leads to unsuccessful reinnervation as the muscle may be fibrotic by the time the regenerating axons reach the target. Transfer of an undamaged nerve to the injured nerve close the motor end plate zone may lead to fast reinnervation. This was initially popularized by Oberlin[5] who used a branch of ulnar nerve to connect to the musculocutaneous nerve to quickly reinnervate the biceps in upper plexus injuries including C6 root avulsion. The success paved way to the same technique being used in many other situations: use of a branch of radial nerve to innervate deltoid via axillary nerve; terminal branch of anterior interosseous nerve to reinnervate ulnar nerve-innervated intrinsic hand muscles.

There is hope. What nature has been hiding within the injured nerve, now we can see by combining electrical studies with ultrasound and magnetic resonance neurography and furthermore, recent advances in surgical techniques are promising better outcomes.

"There is no medicine like hope, no incentive so great, and no tonic so powerful as expectation of something tomorrow." (Orison Swett Marden)

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