Seddon´s Classification

In 1943, Seddon described three basic types of peripheral nerve injury that include:

Neurapraxia (Class I)

Neurapraxia It is a temporary interruption of conduction without loss of axonal continuity.

In neurapraxia, there is a physiologic block of nerve conduction in the affected axons.

Other characteristics:

It is the mildest type of peripheral nerve injury. There are sensory-motor problems distal to the site of injury. The endoneurium, perineurium, and the epineurium are intact. There is no wallerian degeneration. Conduction is intact in the distal segment and proximal segment, but no conduction occurs across the area of injury.

Recovery of nerve conduction deficit is full,and requires days to weeks. EMG shows lack of fibrillation potentials (FP) and positive sharp waves.

Axonotmesis (Class II) Main article: Axonotmesis It involves loss of the relative continuity of the axon and its covering of myelin, but preservation of the connective tissue framework of the nerve (the encapsulating tissue, the epineurium and perineurium, are preserved).

Other characteristics:

Wallerian degeneration occurs distal to the site of injury. There are sensory and motor deficits distal to the site of lesion. There is no nerve conduction distal to the site of injury (3 to 4 days after injury). EMG shows fibrillation potentials (FP),and positive sharp waves (2 to 3 weeks postinjury). Axon regeneration occurs and recovery is possible without surgical treatment.Sometimes surgical intervention because of scar tissue formation is required.

Neurotmesis (Class III) Main article: Neurotmesis It is a total severance or disruption of the entire nerve fiber.

A peripheral nerve fiber contains an axon (Or long dendrite), myelin sheath (if existence), their schwann cells, and the endoneurium. Neurotmesis may be partial or complete.

Other characteristics:

Wallerian degeneration occurs distal to the site of injury. There is connective tissue lesion that may be partial or complete. Sensory-motor problems and autonomic function defect are severe. There is no nerve conduction distal to the site of injury (3 to 4 days after lesion). EMG and NCV findings are as axonotmesis. Because of lack of nerve repair, surgical intervention is necessary.