NERVE TRANSFERS IN BRACHIAL PLEXUS INJURIES SURGERY

Essay

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By

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Aim of the study

The aim of this essay is to review the literature regarding brachial plexus injuries so as to realize the current trends of using nerve transfers in brachial plexus injuries surgery.

Contents

Introduction

■ Nerve injury:

In 1943, Seddon classified nerve injuries into 1 of 3 types: neurapraxia, axonotmesis, and neurotmesis. [(1)]

In 1951, Sunderland reorganized Seddon's classification to emphasize 2 intermediate nerve injury patterns, redefining them as 5 degrees of nerve injury. [(2)]

Mackinnon added a surgically relevant sixth degree injury pattern that combined varying degrees of Sunderland's injuries within a single nerve. [(3)] (Table 1-1). [(4)]

(Table 1-1). Classification of nerve injury. [(4)]

Nerve injury	Degree
Conduction block, resolves spontaneously.	I
Axonal rupture with intact basal lamina.	II
Some scar, axonal and basal lamina rupture; perineurium intact.	III
Complete scar; perineurium ruptured; epineurium intact; ‘‘neuroma in continuity’’.	IV
Complete transaction.	V
Combination of normal nerve and type I-V injuries.	VI

First degree injuries recover spontaneously while second degree injuries recover with the rate of 1 to 1.5 mm/day with function that is far better than that achievable with surgical reconstruction; third degree injuries also recover spontaneously to a more variable degree, although, generally, without the need for surgery; degree 4 and 5 injuries will not

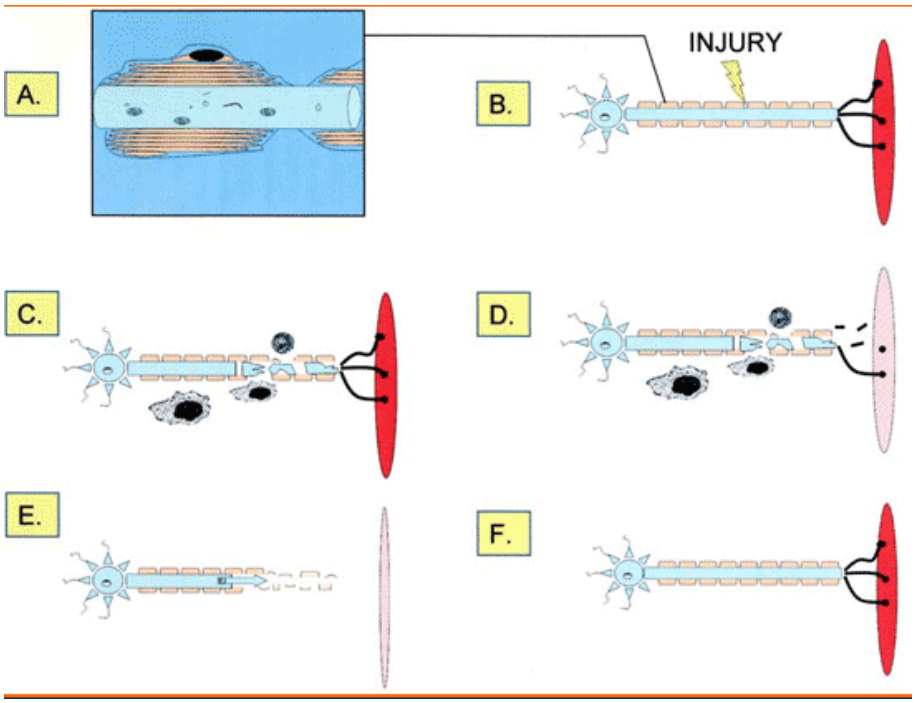
recover without repair; and degree 6 injuries recover to variable degrees, depending on the relative degrees of injury involved. [5]

The mixed injury pattern is often seen in cases of traction. Traction forces on the nerve initially lengthen the epineurium and perineurium. The fasciculi are then stretched, reducing the nerves' cross-sectional area, which raises intrafascicular pressure. Before axonal rupture, the epineurium and perineurium rupture and strip off the nerve. Axons tend to rupture over several centimeters, with the larger fascicles breaking first. [4]

■ Nerve regeneration:

Before regeneration of nerve fibers can occur, a series of degenerative processes must take place, many of which are direct preludes to regeneration. The success of regeneration depends largely on the severity of the initial injury and resultant degenerative changes. [6]

These degenerative changes are seen in (Fig. 1-1). [6]



(Fig. 1-1). *Wallerian degeneration of the peripheral nerve.* In the normal (A) and injury state (B), Schwann cells align along the length of the axon forming multiple layers of myelin. The characteristic spaces between neighboring Schwann cells are known as nodes of Ranvier. After significant injury (C), the nerve will begin to degrade in an anterograde fashion. The axon and surrounding myelin break down during this process. The round mast cells can be seen as can the phagocytic macrophages that interact with Schwann cells to remove the injured tissue debris. As the degradation of the distal nerve segment continues (D), connection with the target muscle is lost, leading to muscle atrophy and fibrosis. Once the degenerative events are complete (E), all that remains is a column of collapsed Schwann cells (bands of Büngner). Axon sprouts with a fingerlike growth cone advance using the Schwann cells as guides. After reinnervation (F), the newly connected axon matures and the preinjury cytoarchitecture and function are restored. [6]

The regeneration and repair phase following nerve injury may last for many months. The earliest signs of this phase are visible changes in the cell body that mark the reversal of chromatolysis. The nucleus returns to the cell center and nucleoproteins reorganize into the compact Nissl granules. [6]

A complex interaction occurs between the cell body and the regenerating axon tip. Axoplasm, which serves to regenerate the axon tip, arises from the proximal axon segment and cell body. Both fast and slow components of axoplasmic transport supply materials from the cell body to the sites of axonal regeneration. [6]

The resistance an axon meets at the injury site results in formation of multiple smaller axon sprouts. Axonal sprouts form at the proximal stump and grow until they enter the distal stump. The growth of the sprouts is governed by chemotactic factors secreted from Schwann cells. [6]

Axons that successfully enter the endoneurial tubes in the segment distal to the injury site stand a good chance of reaching the end organ, given reasonable growth conditions. The distal regeneration rate is slower if endoneurial tubes have been disrupted because axon sprouts must first find their way into the tubes before advancing. The specialized growth cone at the tip of each axon sprout contains multiple filopodia that adhere to the basal lamina of the Schwann cell and use it as a guide. [6]

Regenerating axonal sprouts follow the original Schwann cells to the denervated motor end plates to reform neuromuscular junctions. Collateral sprouting also occurs, resulting in groups of reinnervated muscle fibers, all of the same fast or slow types. This is a characteristic finding in reinnervated muscle, contrasting sharply with the random pattern observed in normal muscle. Appropriate physical therapy can help maintain the denervated muscles in an optimal condition to receive the regenerating axon terminals. [6]

The rate of axonal regeneration has been assumed to be constant and is generally estimated to be 1 mm per day and is often followed by an advancing Tinel sign. Regeneration after surgical nerve repair is slower than uncomplicated regeneration reflecting the severity of original injury. Aging has been shown to retard the rate of axonal regrowth. [6]

The poor prognosis associated with peripheral nerve injuries generally is related to motor nerve injuries because of the time-dependent nature of motor end plate survival after denervation. In an adult, the motor end plates and muscle undergo irreversible fibrosis and atrophy approximately 12 to 18 months after denervation. On the other hand, the potential for sensory recovery is under no apparent time constraint, because the sensory end organs tend to remain capable of responding to reinnervation. The long distance of regeneration in high nerve injuries or the lack of a viable proximal nerve for repair are the situations that stand to gain the most from nerve reconstruction with nerve transfer. [7]

■Neurotization:

Nerve transfers, also referred to as “neurotization”, involve the repair of a distal denervated nerve element using a proximal foreign nerve as the donor of neurons and their axons, which will reinnervate the distal targets. [8]

A healthy donor nerve is separated from its territory and its proximal stump is then connected directly or via grafts, usually to a healthy postlesional distal portion of a non-functioning nerve or implanted directly into denervated muscle or insensitive skin. [8]

Nerve sprouts will grow from the transferred into the denervated elements and establish contact between them and the neurons that formerly commanded another territory. These neurons assume a new end-organ specificity, which means that a given peripheral function now will be controlled or monitored by medullary or cerebral centers that heretofore performed other functions. [9]

■ Concept of nerve transfer:

The concept is to sacrifice the function of a (lesser valued) donor muscle to revive function in the recipient nerve and muscle that will undergo reinnervation. [8]

■ Advantages of nerve transfer:

The major advantages of a nerve transfer over that of a tendon transfer are those: [4]

(1) Nerve transfer is available for both sensory and motor nerves. [4]

(2) Multiple muscle groups can be re-innervated with a single nerve transfer. [4]

(3) The insertion and attachments of muscles are not disrupted. [4]

The advantage over nerve grafting or primary repair is the ability to convert a proximal high-level nerve injury to a low-level nerve injury. [4]

■ Principle criteria for nerve transfer:

Nerve transfers for both motor and sensory nerves have similar criteria and are listed in (Tables 1-2). [4]

(TABLE 1-2). Principle criteria for donor nerves transfer. [4]

Principles of sensory nerve transfers	Principles of motor nerve transfers
Donor nerve near target sensory receptors.	Donor nerve near target motor end plates.
Expendable less critical donor nerve.	Expendable donor nerve.
Pure sensory donor nerve.	Pure motor donor nerve.
Donor-recipient size match.	Donor-recipient size match.
Side-to-end (terminolateral) repair if necessary; end-to-end repair is preferable.	Donor function synergy with recipient function.
Reeducation will improve function.	Reeducation will improve function.

■ Indications of nerve transfer:

Although there is no absolute guideline for when a nerve transfer should be used, it is preferred to use a nerve transfer in the following conditions: [4]

(1) No nerve is available for grafting. [4]

(2) High proximal injuries with long distance for regeneration. [4]

(3) Avoidance of scarred areas in critical locations with potential for injury to critical structures. [4]

(4) Major limb trauma with segmental loss of nerve tissue. [4]

(5) As an alternative to nerve grafting when time from injury to reconstruction is prolonged. [4]

(6) Partial nerve injuries with a defined functional loss. [4]

(7) Spinal cord root avulsion injuries. [4]

(8) Nerve injuries in which the level of injury is uncertain, such as radiation trauma and nerve injuries with multiple levels of injury. [4]

■ Contraindications of nerve transfer:

Contraindications generally include those situations in which other methods of treatment are expected to provide better results or equivalent results with less morbidity. Traction injuries of degree 1 or 2 that will recover completely should not undergo reconstruction. Neuritis should be identified, and early surgery should be avoided for the same reason, because most will recover spontaneously. [(5)]

■ Classification of nerve transfer:

Chuang classified the nerve transfer into four methods in the brachial plexus reconstruction: extraplexus neurotization, intraplexus neurotization, close-target neurotization, and end-to-side neurotization. These four types of neurotization are all of nerve transfer. [(10)]

Extraplexus nerve transfer means transfer of a non–brachial plexus component nerve to the avulsed brachial plexus for neurotization of a denervated nerve. The reported donor nerves in common use are mostly for motor reinnervation, and they include the phrenic nerve, spinal accessory (XI) nerve, deep motor branches of the cervical plexus, intercostal nerves, hypoglossal nerve, and contralateral C7 spinal (CC7) nerve. [(10)]

Intraplexus nerve transfer is used in cases of non–global root avulsion, in which one of the spinal nerves is still available for transfer, not to its original pathway but to other more important nerves. [(10)]

Close-target nerve transfer is a procedure of distal nerve transfer, providing a direct coaptation at a more distal site closer to the end organs targets, muscle or skin, thus achieving faster recovery of the motor and sensory outcome. [(10)]

■ End-to-side nerve transfer:

Traditionally, nerve transfers are performed in an end-to-end manner with complete transection of the donor nerve then coapted to the recipient nerve. This repair is effective and preferred with motor nerve transfers when regeneration of the recipient nerve is not expected. An alternative coaptation technique is the end-to-side coaptation. This involves the coaptation of the distal end of the denervated recipient nerve to the lateral aspect of the intact donor nerve, which serves as a source of regenerating proximal axons by axonal sprouting. [(11)]

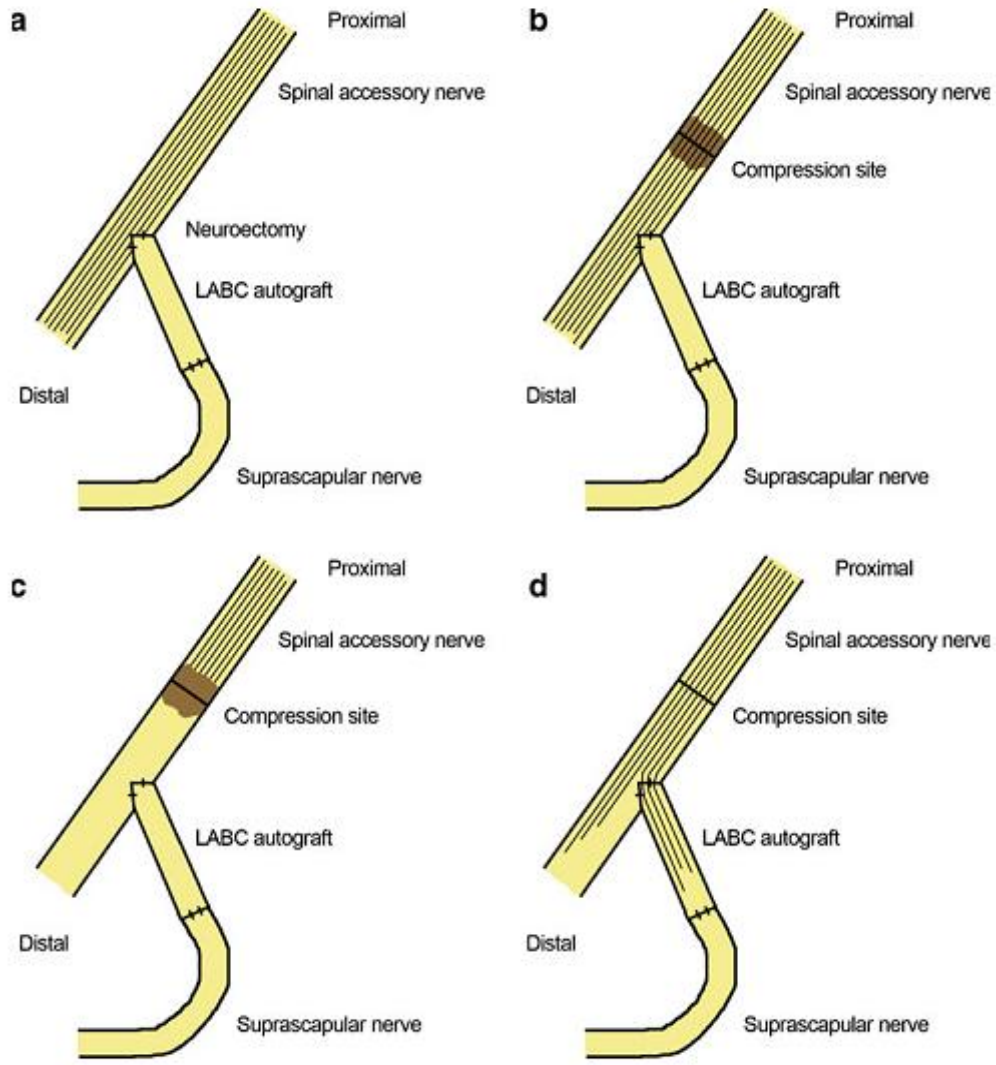
However, clear differences exist between sensory and motor regeneration across an end-to-side repair. Regeneration in sensory nerves occurs through spontaneous sprouting (collateral sprouting), while donor motor nerve requires axonotmetic injury for neuronal regeneration (regenerative sprouting). [(11)]

Collateral sprouting of axons is a branching outgrowth of new axon terminals from uninjured axons. It usually leads to an expanded innervation area of the uninjured into the adjacent denervared tissue. After peripheral nerve injury, collateral sprouting of sensory axons from the adjacent non injured nerves into the denervated skin critically depends on the availability of the nerve growth factor (NGF). It is proposed that signaling between the injured and intact neurons in the dorsal root ganglion (DRG) is an important induction mechanism for collateral sprouting. [(11)]

As regard regenerative sprouting, the motor nerve transfer is augmented by performing a proximal crush on the donor nerve or a partial neurectomy. This has been shown experimentally to improve the motor axonal sprouting and subsequent nerve regeneration into the recipient nerve. The added injury to the donor nerve is critical to

encourage donor motor sprouting. Without this donor nerve injury, spontaneous motor sprouting from the donor nerve will not occur. [11]

Reinnervation mechanism of an end-to-side neurotization by regenerative sprouting is shown in (Fig. 1-2). [12]



(Fig. 1-2). Mechanism of *accessory to the suprascapular end-to-side nerve transfer*. a, neuroectomy at the coaptation site of the spinal accessory nerve is created in order to acquire reinnervation by the motor fibers. b, an axonotmetic injury is created proximal to the end-to-side neurotization through a compression to induce reinnervation of the suprascapular nerve. c, Wallerian degeneration proceeds distal to the axonotmetic injury following the compression. d, Afterwards, axonal regeneration proceeds through the native donor and into the recipient pathway through the end-to-side repair. [11]
LABC, lateral antebrachial cutaneous nerve.