1 Nerve Surgery: Historical Perspectives

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Abstract

Nerves have always mystified and intrigued scientists. Ancient theories revolved around identifying the "divine substance" that flowed within nerves, and early healers believed that repairing nerves could result in "cruel death." The first major paradigm shift occurred when this nihilistic view of nerve repair gave way to earnest attempts at restoring nerve continuity. Refinements in nerve repair techniques paralleled our understanding of nerve regeneration. Advancements in nerve reconstruction were then propelled by an unprecedented number of wounded soldiers treated during the American Civil War and two World Wars. Early attempts were focused on restoring the physical continuity of the nerve at all costs. These included flexed limb posturing, shortening bones, use of nerve flaps, and tensioned repairs, which largely resulted in poor outcomes. The second major paradigm shift was the use of nerve grafts in the late 1960s in managing these critical nerve gaps. However, observations that longer nerve grafts led to increasingly worse results highlighted the fact that the "distance problem" still remained unsolved. With the introduction of the microscope and pivotal advancements in our understanding of intraneural topography, an elegant solution arose in the form of nerve transfers. The widespread adoption of nerve transfers and new ability to address a multitude of complex peripheral nerve issues marked the third major paradigm shift in nerve surgery, bringing us to the modern era.

Keywords: nerve repair, nerve grafting, nerve transfer, brachial plexus, nerve injury

1.1 Introduction

The history of nerve surgery spans centuries of scientific, technological, and medical innovation. This chapter highlights the three major paradigm shifts that have revolutionized nerve surgery. Primary repair, nerve grafts, and nerve transfers all still have a place in the modern toolbox of complex nerve reconstruction, and the subsequent chapters in this textbook describe the current state-of-the-art techniques in each. This chapter focuses the lens on the past, outlining the key events and people that have built the foundation of nerve surgery.

1.2 Ancient Theories: From Animal Spirits to Electricity

During a time when diseases were attributed to evil forces, all living things were thought to have an internal "spirit" or "soul" that was essential for life.[1,2,3] Our earliest understandings of the anatomy and physiology of the nervous system came from *Erasistratus of Chios*, the father of physiology. He described the body as being made of three distinct channels: veins, arteries, and nerves.[3] Veins contained blood produced by the liver; arteries carried the *vital spirit*; and nerves carried *animal spirit*, which was produced from the vital spirit.[4] Based on knowledge gained from animal dissections, Erasistratus proposed that sensory and motor controls were mediated by different nerves. *Galen of Pergamon* (▶ Fig. 1.1) referred to them as soft and hard nerves, respectively. Galen hypothesized that the substance

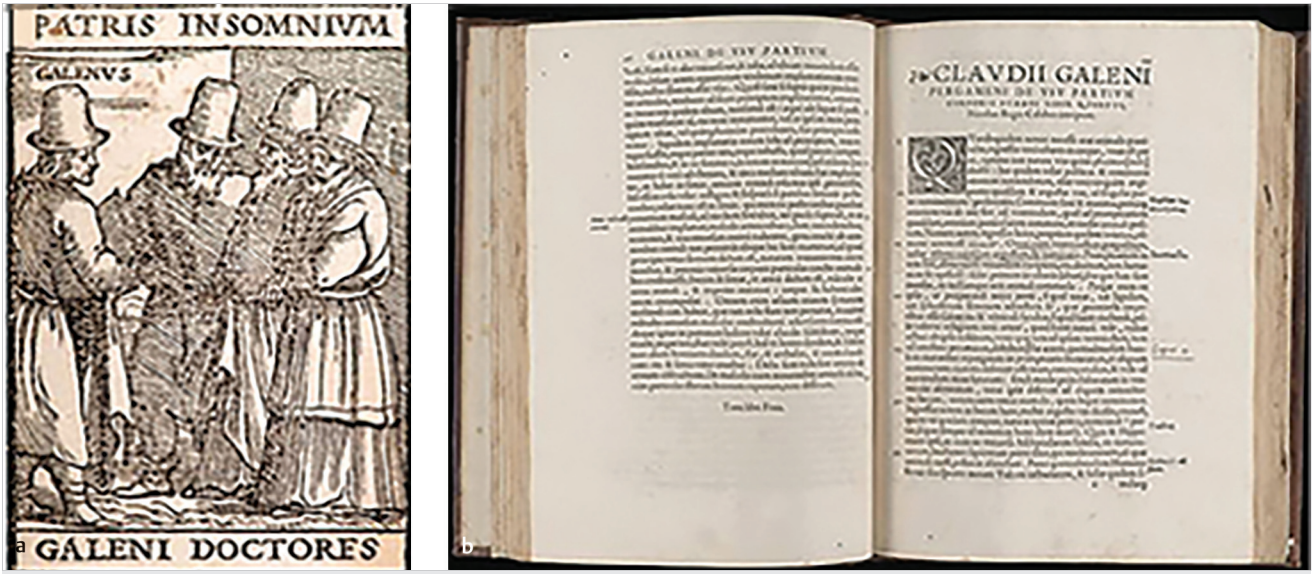


Fig. 1.1 Galen of Pergamon (c. 129–216). **(a)** Galen, illustrated in 1556, *Galeni Omnia Quae Extant in Latinum Sermonem Conversa*. Venice, Apud Luntas. He was the personal physician to emperors Marcus Aurelius and Commodus. His laborious and meticulous dissections are described in *De usu partiuum* (On the Usefulness of the Parts) shown in **(b)** and *De anatomicis administrationibus* (On Anatomical Procedures). He distinguished hard nerves (motor) from soft nerves (sensory) and believed that after passing through the brain, the animal spirits flowed in nerves. His descriptions of the spinal cord, recurrent laryngeal nerve, sympathetic ganglion, brachial plexus, and seven cranial nerves are surprisingly accurate, although he viewed the brachial plexus not as a discrete anatomic structure but a reorganization process of nerves before entering the upper arm. (Reproduced with permission from Thomas M. Brushart.)

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within hard motor nerves acted mechanistically, causing nerves to pull on the muscle. For example, the phrenic nerve would pull up on the diaphragm during expiration, and the recurrent laryngeal nerve would pull on the inferior laryngeal muscles to produce sound.[5] *William Harvey*, however, noted that "nerves are like pipes and not like reins or the sources of movement" based on their small size, fine caliber, and often oblique and multiple insertions to individual muscles.[6]

The substance that flowed through these "pipes" remained debated and elusive. *Renes Descartes* postulated that nerves were composed of a large number of small hollow tubes, which allowed animal spirits to flow into the muscles.[3] Descartes described valves arranged in such a way that animal spirits flowing into one muscle would also close off valves to its antagonistic muscle.[7] The observations that there was "electrical matter" in animals, such as the electric eel, sparked the idea that electricity was the long sought-after agent of nerve action.[3] *Luigi Galvani* described metallic objects causing muscle contractions in a frog after being in close proximity to sparks generated from a machine. Similarly, he noticed that when a metal arc completed the circuit between a muscle and rest of the frog, muscle contractions occurred. Galvani renamed Erasistratus' animal spirits "animal electricity" instead and believed in the intrinsic ability of "electricity" within animals to incite muscle contractions (▶ Fig. 1.2).[8] He proposed that nerves carried electric fluid and were surrounded externally by a substance that prevented the dissipation of this fluid. This also explained the cessation of muscle contraction if a ligature was placed around a nerve.[8] Insightfully, he felt that neuropathies could be caused by a blockage or slowing of this electrical flow.[8] Galvani's conclusions were refuted by *Alessandro Volta*,[3] who believed that the generator of muscle action was the metallic arc itself. He demonstrated that the strength of contractions was proportional to the number dissimilarly coupled metals (basic battery units) connected in series. Volta's arguments were so widely supported that it would not be until the beginning of the 20th century, when *Svante Arrhenius* demonstrated the properties of ions in electrolyte solutions, that our current understanding of the electrical nature of nerves, ion channels, and action potentials would become established.

1.3 Nerve Injury and Regeneration

Dating back to the third century BC, Hippocrates and Galen believed that the attempted repair of injured nerves would cause "unbearable pain, spasms, and cruel death." Galen even went so far as to suggest that partially injured nerves should be completely cut to prevent "convulsions."[3] Not all early healers agreed with these views, and some attempted repairs using "agglutinatives" and egg albumin to bind nerves.[9,10] These early attempts were often deemed an abysmal failure when there was no visible return of nerve function, despite healing of the overlying soft tissues. This seemed to support Galen's views on the irreparability of nerves. The one notable departure was Guy de Chauliac (1300–1370), who believed that nerves could "consolidate by first intention," in a way similar to bony healing, if the two ends were kept in proximity.[11] However, this was the exception and the galenic view on the futility of nerve repairs remained widely supported until the 18th century.

In the late 1700s, growing experimental evidence supported the ability for nerve healing. There became two main schools of thought: those who believed in "nerve reunion," where function returned after the ends of the nerve were reunited by an exudate from the medium, and those who believed in nerve regeneration.

Proponents of the nerve reunion theory cited cases where functional recovery in divided major nerves occurred too quickly to have been a result of regeneration.[12] *James Paget*, after whom Paget's disease of the breast and bone are named, was a major proponent of this theory. He was also surgeon extraordinaire to Queen Victoria and president of the Royal College of Surgeons, with significant influence on the medical establishment. The "reunionists" believed that distal axons remained viable but lost their myelin sheaths. According to their theories, the return of function occurred when the proximal and distal axons united and the myelin regenerated.

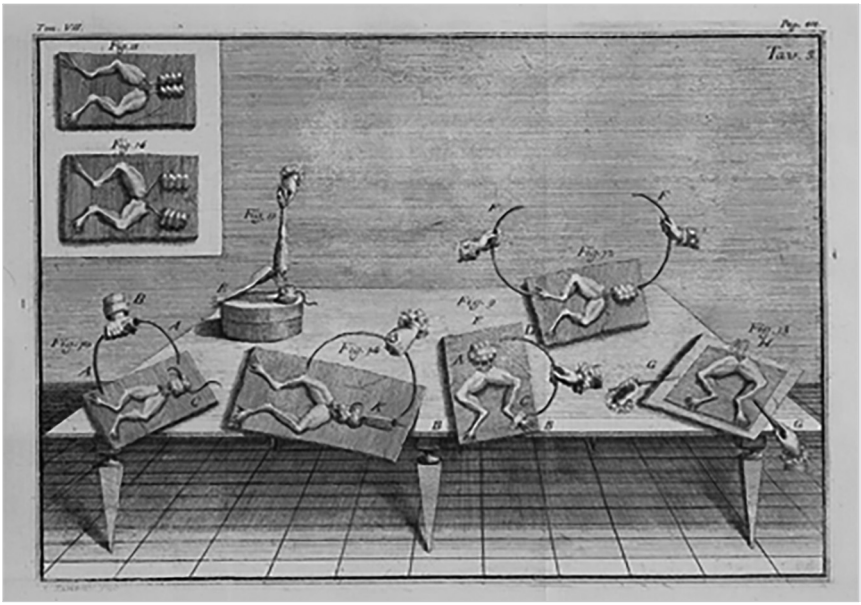


Fig. 1.2 Luigi Galvani (1737–1798). Illustration of 1780 experiments by Galvani showing activation of the muscles in a frog when touched with a metallic object that completed circuit powered by an electric source. He demonstrated the role of electricity in generating and conveying an impulse and concluded that "animal electricity" flowed within nerves. Galvani A., 1791. De Viribus Electricitatis in Motu Musculari commentarius. Bononiae, Ex Typographia Instituti Scientiarum. (Reprinted from Piccolino M. Animal electricity and the birth of electrophysiology: the legacy of Luigi Galvani. Brain Research Bulletin. 1998; 46(5):381–407 with permission from Elsevier.)

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Pivotal observations were made by *William Cruikshank* and *Augustus Waller* in support of nerve regeneration. In a dog model, when Cruikshank cut the vagus nerve on one side followed by the other side, the dog died 2 weeks later. When he examined the original site of vagotomy, he found a white substance bridging the nerve gap. When he repeated the experiment but waited a longer period of time before cutting the vagus nerve on the second side, the dog survived. This suggested enough time had passed to allow for nerve regeneration and the return of function on the first side.[13] *John Haighton* reproduced these results and further reported the dog regaining the ability to bark 6 months following bilateral vagotomies.[14] Waller's seminal observations of the histologic changes in the distal nerve following nerve transection have been eponymously named wallerian degeneration. He described the concept of a vital center (which we now know as the cell body), which maintained the viability of the attached axonal portion, unlike the entire distal portion, which underwent degeneration.[15] He postulated that function was restored from fibers regenerating proximally from this vital center. *Louis-Antoine Ranvier* supported this view and hypothesized that the axons were guided distally toward their targets by Schwann cells.

Camillo Golgi (1843–1926), professor of Histology and General Pathology, developed a staining method that beautifully visualized neuronal cell bodies and their entire network of axons and dendrites.[16] Originally termed the "black reaction," tissue sections are immersed in silver nitrate, which outlines the neuronal tissue with a black deposit (▶ Fig. 1.3). As a result of visualizing these complex interconnected networks, Cajal was a strong supporter of the "reticular theory," which posited that neuronal communication was made possible by cells fusing together to form a single large network (reticulum).

Santiago Ramón y Cajal further improved upon Golgi's staining method by utilizing thicker tissue sections and more intense staining protocols on neonatal specimens, before myelination occurred, so that axons could be exposed to the reagent. Using this technique, Cajal showed axonal sprouting from the proximal nerve stump of a cut nerve, proving that nerve regeneration was possible. However, Cajal could not find any supportive evidence of neurons physically touching each other. He, instead, supported the "neuron theory," that is, cells only had to be contiguous to be able to transmit signals. Golgi vehemently rejected this theory and the ongoing debate between the two continued even after they were jointly awarded the 1906 Nobel Prize in Physiology or Medicine.[17]

Others, including zoologist *Theodor Schwann*, theorized that axons only regenerated in the distal segment. In 1838, together with botanist Matthias Jakob Schleiden, Theodor Schwann proposed the cell theory where "all plants and animals were made up of cells."[18] His research led to the discovery of the now named "Schwann cells," which produced the myelin sheath that enveloped axons. He theorized that the nucleus of each Schwann cell was responsible for regenerating an axonal segment, which then fused to reconstitute the distal axon. This also explained the rapid functional recovery seen within days of nerve repair. In hindsight, these findings were more likely due to redundant innervation or anatomic variations.[13] The pivotal role of Schwann cells in nerve regeneration would also be later refined. Proliferating "repair" Schwann cells form longitudinal Büngner's bands that guide regenerating axons via contact guidance. Waller's theories would remain debated until the 20th century, when embryologist *Ross G. Harrison*'s pioneering work showed that axonal sprouting occurred in neuronal cultures derived from embryonic neural crest cells but not from isolated cultured Schwann cells.[19]

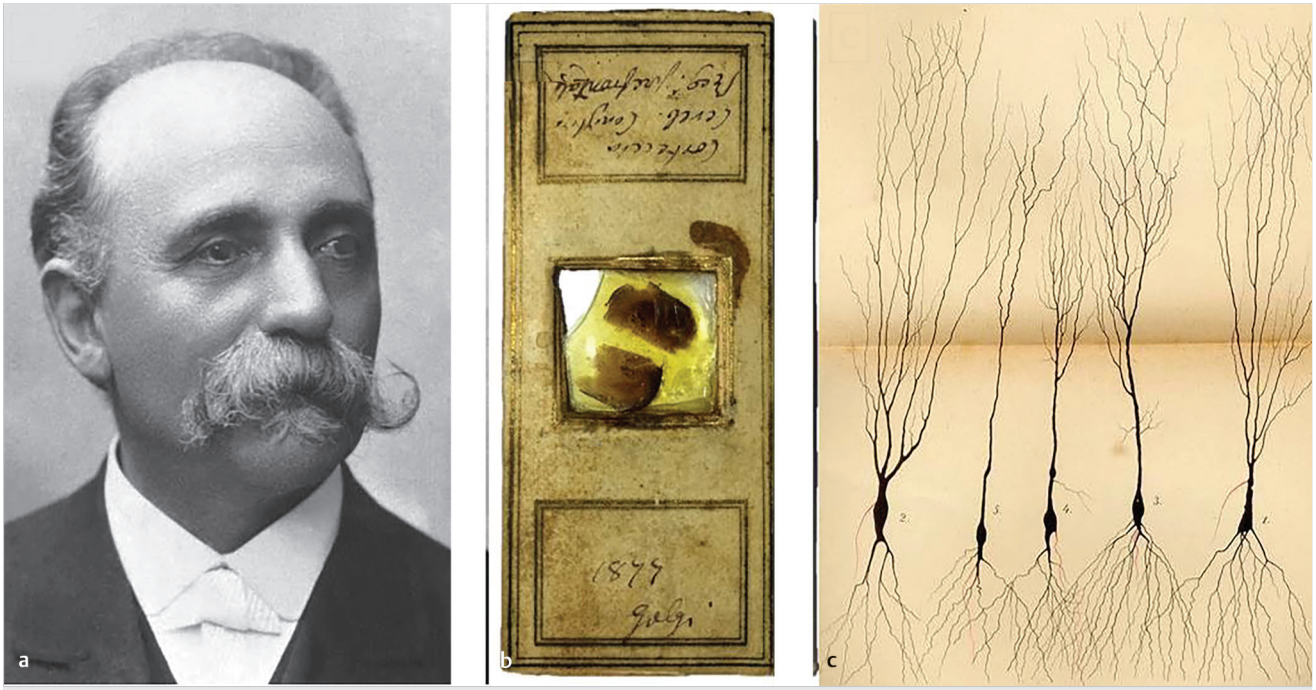


Fig. 1.3 Camillo Golgi (1843–1926). **(a)** Portrait of Golgi. **(b)** Photograph of tissue slides signed by Golgi in 1899 from the Golgi Museum in Pavia, Italy. **(c)** Representative drawing of Golgi stained rabbit hippocampus. (Reproduced from Bentivoglio et al. Front Neuroanat 2019;13;3. https://creativecommons.org/licenses/by/4.0/)

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In 1924, *Henri Delageniere* and *Jules Tinel* reported on the recovery of nerves after surgical repair. Based on Delageniere's observation of the slow 1- to 2-mm progression of regenerating axons, he advised that surgeons wait a sufficient amount of time to allow for spontaneous nerve regeneration before intervening.[20] This observation underpins a phrase commonly used today, "a millimeter a day, an inch a month, a foot a year" in regard to nerve regeneration. Hoffmann and Tinel reported on the ability to follow the course of a regenerating nerve. The paresthesias that are elicited with light percussion along the distal course of the nerve are now referred to as a positive "Hoffmann–Tinel sign."[21,22,23] These advances in understanding nerve physiology and regeneration would be mirrored by renewed attempts in nerve repair.

1.4 Primary Nerve Repairs

In 1608, *Gabriele Ferrara* provided the first detailed technical description on repairing a transected nerve threading a split tortoise tendon soaked in red wine, rosemary, and roses through the eye of a needle.[24] Throughout the 18th and 19th centuries, numerous suture materials were trialed, including silk, silver, linen, hair, split animal tendons, and catgut. Various repair techniques were also attempted, including binding the cut nerves ends together in a side-by-side fashion (▶ Fig. 1.4),[14] using a tongue-in-groove or wedge technique for end-to-end coaptation,[24] configurations that resembled core sutures in tendon repairs,[25,26,27,28] and using the same suture through nerve and skin. *J. M. Philipeaux* and *Alfred Vulpian* used a single suture passed through the epineurium, out the center of one cut end, into the center of the other end, and out the epineurium.[29]

In 1919, *Charles Elsberg* described the deleterious effects of traction on nerve regeneration and recommended an end-to-end repair where the "funiculi" (fascicles) are just brought into opposition. He also emphasized the importance of trimming the proximal segment until normal, healthy fascicles were seen.[30] These would form the basic tenets of modern nerve repair, including segmental excision using the "bread-loafing" technique until healthy fascicles are seen proximally and distally and tension-free neurorrhaphy.

Surgeons have long debated over the best type of nerve repair. Since the 1800s, an epineurial repair had been the accepted standard.[31] In 1917, *Langley* and *Hashimoto* first described a perineurial repair technique allowing for better alignment of fascicles. However, this was largely disregarded due to the time-consuming and technically demanding nature of the repair.[32] The advent of the operating microscope brought renewed interest in perineurial repairs. Several small studies demonstrated favorable results from grouped fascicular repairs,[31,33,34,35] but these were counterbalanced by others demonstrating no benefit.[36,37] These efforts were swept away by the introduction of conduit-assisted repairs using tubes of various materials, which was faster and required less skill to perform. This was based on *Goran Lundborg*'s thoughts that neurotropism could guide axons across an unstructured gap toward a distal target.[38] While axons did make it across the gap, they were not guided to the correct Schwann cell tubes to allow for optimal functional recovery. Later investigations also found that many tube-assisted repairs with nonresorbable silastic, collagen, or polylactic/polyglycolic led to chronic compression.[39]

Surgeons faced another major challenge in the reconstruction of nerve gaps. Many approached this with creative, but now outdated, techniques focused on the restoration of physical

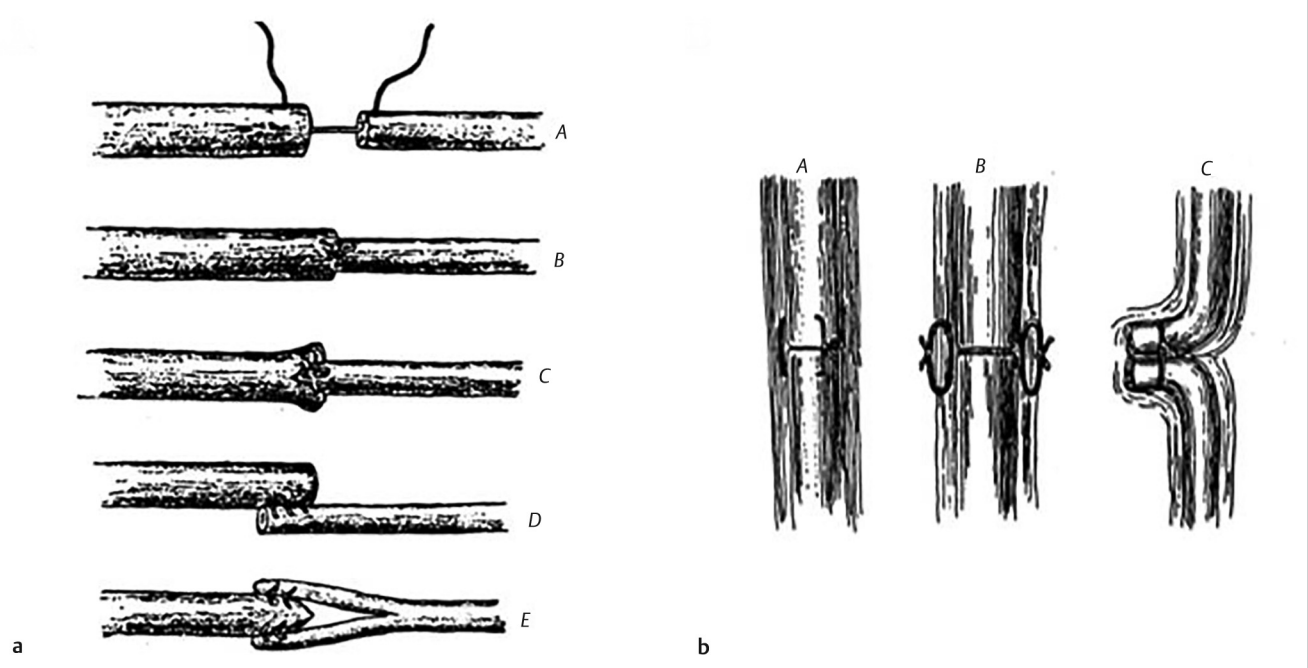


Fig. 1.4 Illustration of 19th century techniques for direct nerve repair. **(a)** Vulpian's center suture technique (A, B); wedge techniques (C, E); and Rawa's side by side technique (D). **(b)** Various types or epineurial sutures (A, B) and Rawa's coapted suture (C). (Reproduced from Walker AE. A History of Neurological Surgery. Martino Publishing; 1967 with permission from Wolters Kluwer Health, Inc.)

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nerve continuity. These methods included physically stretching the nerve: two-stage "bulb stretching" whereby the proximal and distal end neuroma bulbs were forcefully reapproximated and then divided and repaired at the second stage; "nerve flaps" where half the fascicles in each end were cut proximally and transposed 180 degrees and sutured end-to-end or side-to-side; and double lateral implantation techniques, where the disconnected injured nerve was placed into slits made in a host nerve.[40,41,42,43] *Schuller* introduced the idea of flexing joints to help reduce the size of a nerve gap, followed by gradual stretching with serial casting. Others tried bridging the gap with various scaffolding materials for regenerating axons including multiple loops of catgut suture, central canals of decalcified bones, human arteries, veins, dura mater, and even gelatin.[44]

This was during the time of the First and Second World Wars where there were an unprecedented number of traumatic peripheral nerve injuries in wounded soldiers. Spurred by necessity, surgeons employed all of these techniques in an attempt to manage critical nerve gaps.[45] *Gotthelf Carl Huber* established multiple clinical and experimental peripheral nerve centers during the war and published *A Manual of Neurosurgery* in 1917 detailing the results of many of these procedures. One of his pupils, *Byron Stookey,* soundly refuted the ill-conceived nerve flap and showed histologic evidence that it did not function as a regenerating nerve conduit in animal models. Stookey's wartime experiences served as the foundation for his own landmark textbook, *Surgical and Mechanical Treatment of Peripheral Nerves*, which remained the definitive text on nerve repair for decades. Furthermore, *Highet* and *Holmes* performed secondary explorations on nerves that had been stretched. They found extensive intraneural hemorrhage and fibrosis both proximal and distal to the suture line.[39] Due to these findings and poor clinical outcomes, many of these techniques were abandoned shortly following the war.

1.5 Nerve Grafting

Robinson is generally credited with the first successful nerve autograft procedure in 1889. He bridged a 6-cm gap in the median nerve using popliteal nerve graft from an amputated limb. However, it would not be until the mid-1960s that the paradigm shifted when *Hanno Millesi* championed and defended the use of nerve autografts. Early results with nerve grafting had been mixed as they often reported the combined results from patients with nerve xenograft, allografts, and autografts. Many initially held great skepticism and considered nerve grafting in the same category as the nerve flap and other previously abandoned techniques.

Despite this early pessimism, successful results were reported by *Sterling Bunnell* in 1927 and 1937 from grafting digital nerves[46,47] and *Balance* and *Duel* in 1932 on grafting facial nerves.[48] In reviewing a large series of 375 cases, *Delangeniere* noted that the success of nerve grafting diminished with increasing graft length.[20] Similarly, in a series of 58 cases, *Sir Herbert Seddon* reported satisfactory results in only 52% of cases.[49] He wrote that "all grafts shrink; [and] the graft or collection of grafts should, therefore, be about 15% longer than the gap to be repaired." Furthermore, "the diameter of the graft or collection of grafts must be at least equal to that of the peripheral stump of the nerve that is being repaired." He also recognized that the nerve graft could derive nourishment from its surrounding tissue bed; thus, he opposed wrapping the nerve graft with metal foil, which was a practice recommended by some to reduce scar infiltration. Many of these early insights, reported almost 100 years ago, still hold true today.

In the mid-1960s and 1970s, *Millesi* emphasized the importance of having *no tension* across a nerve repair, utilizing meticulous microsurgical technique, including instrumentation, sutures, and magnification, and starting early range of motion starting on postoperative day 3. He presented persuasive animal and clinical data that demonstrated "axon[al] regeneration and neurotization in the distal stump occur to a much higher degree after nerve repair by nerve-grafting than after sutures under tension, despite the fact that after grafting, the axons must cross two suture lines."[50,51] He strongly advocated for the use of nerve grafts whenever there was any tension.[52] Millesi also made substantial effort to match and approximate corresponding fascicles between the proximal and distal nerve ends. His initial presentations were received with disbelief, hesitation, and criticism. *Erik Moberg* and *Donald Brooks*, both especially skeptical of his results, became supporters only after travelling to Vienna to personally evaluate his outcomes. Millesi presented his results in 1971 at a controversial and divided American Society for Surgery of the Hand meeting. Millesi would later thank Harry Buncke, who was moderating the session, for "coming to his rescue" during the heated discussion by those opposed to using nerve grafts (▶ Fig. 1.5). In 1978, at a symposium in Durham, North Carolina, the contentious debate between primary repair and nerve grafting continued. *Leonard Goldner*, a staunch advocate of primary nerve repair, presented

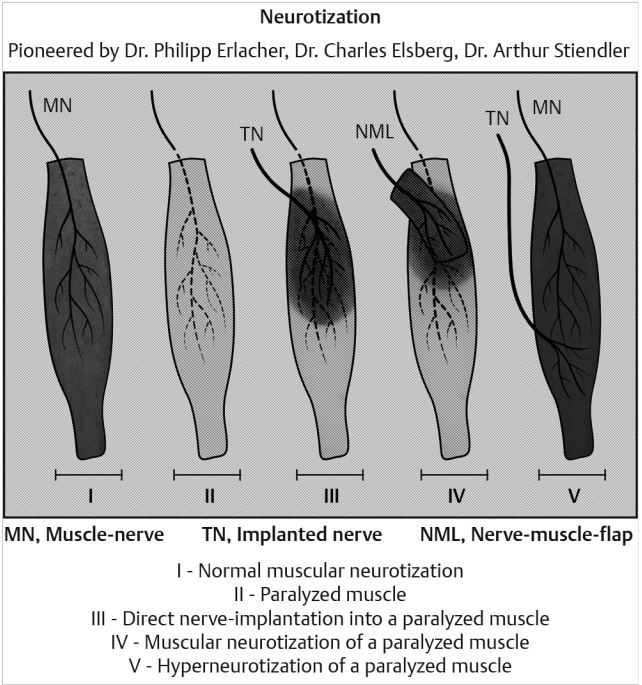


Fig. 1.5 Illustration of various techniques of nerve reconstruction.

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a young patient undergoing over 10 cm of humeral shortening to facilitate primary radial nerve repair with mediocre results. During this same session, *Raymond Curtis* presented 25 cases of long critical nerve gaps of the radial nerve managed with nerve grafting with superior results. Millesi credited Curtis as his first adopter and that meeting as the "breaking point" for the acceptance of nerve grafting.

The pivotal role of nerve grafting in the management of brachial plexus injuries would solidify its widespread acceptance. Up until this time, surgical outcomes of brachial plexus reconstruction were abysmal.[53,54,55] In 1925, Sever's series of 1,100 obstetric brachial plexus patients demonstrated no distinguishable benefit of surgery compared with conservative management.[56] Poor results from multiple groups led to the conclusion that attempted repair of brachial plexus injuries could not guarantee effective results.[57] The introduction of microscopy and use of intraoperative nerve monitoring would change the outlook and approach to the management of brachial plexus injuries.[58]

Algimantas Otanas Narakas, a Lithuanian-born surgeon practicing in Switzerland and pioneer in brachial plexus reconstruction, was an early adopter of Millesi's nerve grafting method.[59] In 1969, he reported a case of utilizing the ulnar nerve as a graft to repair the infraclavicular brachial plexus with good outcomes.[60,61] Narakas was also one of the first to use biologic glue in addition to two sutures at the nerve coaptation site, which dramatically decreased the operative time while increasing success rates.[62] He strongly advocated for early surgical intervention within 1 to 3 months postinjury[63,64] and the use of intraplexal neurotization for root avulsions.[61,65] His magnum opus would be a comprehensive review of over 1,000 brachial plexus injuries published in 1985. From his detailed analysis of clinical outcomes, he described the rule of 7 times 70%, which have largely withstood the test of time: 70% of cases were caused by traffic accidents, 70% of victims were motorcyclists, 70% presented with combined injuries, 70% were supraclavicular lesions, 70% involved the avulsion of at least one nerve root, 70% of patients with avulsions would develop chronic pain, and 70% of these avulsions involved a C7–C8–T1 level nerve root.[66] Narakas' contributions to brachial plexus and nerve surgery have been memorialized in the triennial meeting of international brachial plexus surgeons named the "Narakas Club."

1.6 Nerve Entrapment

Paralleling the debates in managing critical nerve gaps was the ongoing controversy on the necessity of internal neurolysis in managing compressive neuropathies. This arose from the combination of conflicting early studies, opposing hypotheses regarding the pathophysiology of nerve compression, lack of an experimental animal model, and anecdotal accounts on the deleterious effects of internal neurolysis. *Babcock* wrote of the value in opening the thickened nerve sheath of a compressed nerve to reduce internal pressure.[45,67] He advocated for splitting the nerve sheath in a procedure he termed "hersage," to illustrate the technique of "combing" the nerve fascicles. Internal neurolysis was advocated to address the intraneural fibrosis within the nerve that was thought to occur with chronic compression. Histologic reports based on case reports and postmortem analyses had supported these findings.[68,69,70,71]

In 1961, Raymond Curtis reported on the results of carpal tunnel release and internal neurolysis of the median nerve for patients with constant sensory loss, abnormal static two-point discrimination, thenar atrophy, or failed prior release.[71] His technique involved intraneural dissection and excision of intraneural fibrosis until individual fascicles were visualized. He kept the posterior epineurium intact to preserve vascular supply from the mesoneurium.[71] There was great reluctance from other surgeons to accept this technique due to concerns that this degree of intraneural dissection would create more scarring and result in worse outcomes.

Two experimental studies on the effects of internal neurolysis by Lundborg in 1976 and Alan Hudson in 1981 had contradictory findings.[72,73] Lundborg examined the intraneural blood flow in normal rabbit tibial nerves following neurolysis and found that "intraneural dissection produce[d] considerable trauma to the nerve…[can] induce fibrosis in all layers of the nerve and may cause some nerve fiber damage. However, the barrier function of the perineurium and the endoneurial vessels seems to be generally well preserved."[74] Hudson's study on rat sciatic nerves, however, concluded that "it [was] possible to perform internal neurolysis without producing any significant long lasting morphological or physiological alterations" in a peripheral nerve. He wrote that "the crucial maneuver, however, is … preservation of the perineurium.[73] In a guest editorial in 1981, *George Phalen*, the "father of the carpal tunnel syndrome," stated "with the advent of microsurgery, some hand surgeons are advocating internal neurolysis of the median nerve as a routine part of every operation for carpal tunnel syndrome. Such advice courts disaster. Although the immediate results may be good, the later inevitable interfascicular scarring can produce irreparable damage to the median nerve."[75]

Development of the rat sciatic and primate median nerve models for chronic nerve compression in the 1980s allowed for more accurate evaluation of the associated intraneural changes with chronic compression.[76,77] These models demonstrated progressive inter- and extrafascicular epineural fibrosis and perineurial thickening with ongoing compression. Utilizing a primate model of compressive median neuropathy, *Susan Mackinnon* and *A. Lee Dellon* showed no significant neurophysiologic or morphometric differences between decompression alone and decompression with microsurgical internal neurolysis.[78] In particular, internal neurolysis did not produce scarring within the nerve. In subsequent studies, they demonstrated that breaching the perineurium resulted in only a temporary breakdown of the blood–nerve barrier, which recovered after 10 days. In 1991, a prospective, randomized, double-blind study showed no benefit of internal neurolysis in primary carpal tunnel surgery,[79] and this has since remained the preferred management strategy for primary carpal tunnel release by most surgeons. An unexpected outcome of this work was the realization that a surgeon could enter the nerve, open the internal epineurium to separate fascicles, and breach the perineurium without irreparable harm. This allowed surgeons "permission" to look for fascicular topography and ultimately perform modern nerve transfer surgery.

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1.7 Nerve Transfers

Despite the advancements in the understanding of compression neuropathy, the "distance problem" with brachial plexus and proximal nerve injuries still remained unsolved. As Delageniere astutely noted in 1924, the outcomes of nerve grafting diminished with increasing graft length. Given the proximal nature of brachial plexus injuries and the time required for recovery, outcomes of nerve grafting in many patients were still suboptimal. The earliest nerve transfers or "cross-unions" were described by Harris and Low (1903) and Tuttle (1913).[80,81] George Woolsey's descriptions of various "nerve anastomosis" in 1911[82] (▶ Fig. 1.6) and Pollock and Davis' depictions of "nerve crossings" in 1933[83] were early glimpses of nerve transfer variations (▶ Fig. 1.7). In 1963, at the Watson-Jones Lecture, Seddon described transferring the third and fourth intercostal nerves to the musculocutaneous nerve, using a nerve graft.[84] Despite these reports, it would require decades before the next paradigm shift in nerve surgery would occur.

Within this time, pivotal advancements in our understanding of nerve regeneration, functional recovery, and intraneural topography were key in setting the stage. In the 1970s, Kristensson and Olsson injected horseradish peroxidase (HRP) into distal targets including the hypoglossal nerve in the tongue and tibial nerve in the gastrocnemius muscle. Twenty-four hours later, there was positive HRP staining of neuronal cell bodies in the brain (for hypoglossal nerve) or spinal cord (for tibial nerve), demonstrating the successful staining of retrograde HRP transfer from its distal injection site.[85] Using this technique, *Thomas Brushart* then elegantly demonstrated the preferential regeneration of motor neurons down a motor pathway in a rat model.[86] He transected the femoral nerve proximally and interposed a Y-shaped silicone tube with the distal motor and sensory branches sutured into the two arms. Two months later, allowing adequate time for nerve regeneration, retrograde labeling was performed on the distal sensory and motor branches. These experiments demonstrated more motor neurons regenerating from the motor pathway. The subsequent use of fluorescent markers for retrograde labeling would be crucial in visualizing nerve regeneration through the first nerve transfers[87] and further our understanding of nerve regeneration.[88] They would beautifully highlight the regeneration of axons from the proximal stump of the donor nerve into the distal stump of the recipient nerve.

The next stepping stone was understanding the capacity for functional compensation of motor neurons in partially denervated muscles and, subsequently, the requirements for reinnervation and recovery. In the 1950s, Upton and McComas

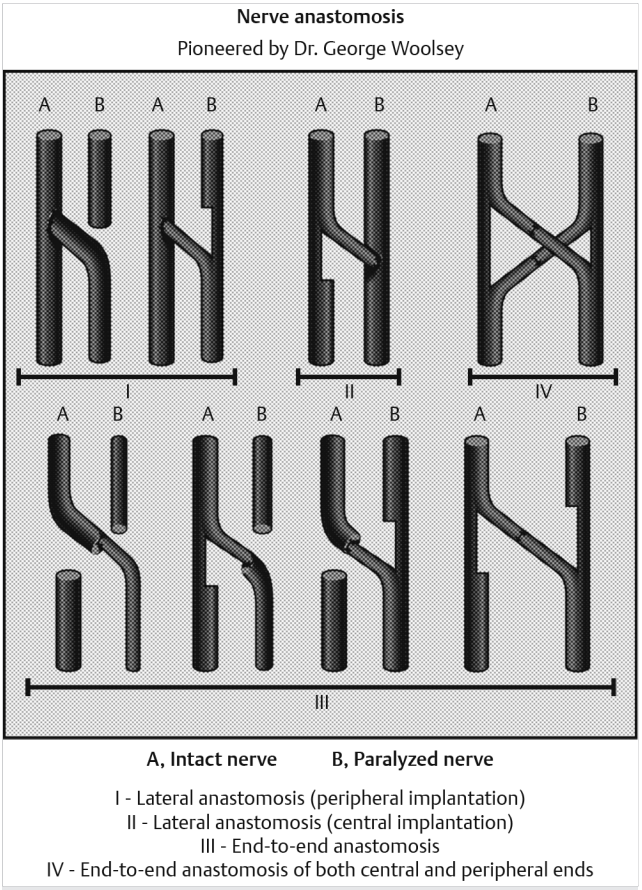


Fig. 1.6 Nerve anastomosis. George Woolsey's depictions of various methods of "Nerve Anastomosis" from the 1911 textbook, *Surgery: It's Principles and Practice*. In each panel, "A" represents the intact nerve and "B" represents the paralyzed or injured nerve. Various configurations are depicted including lateral anastomosis with peripheral implantation (I); lateral anastomosis with central implantation (II), end-to-end anastomosis (III), and end-to-end anastomosis of both central and peripheral ends (IV).

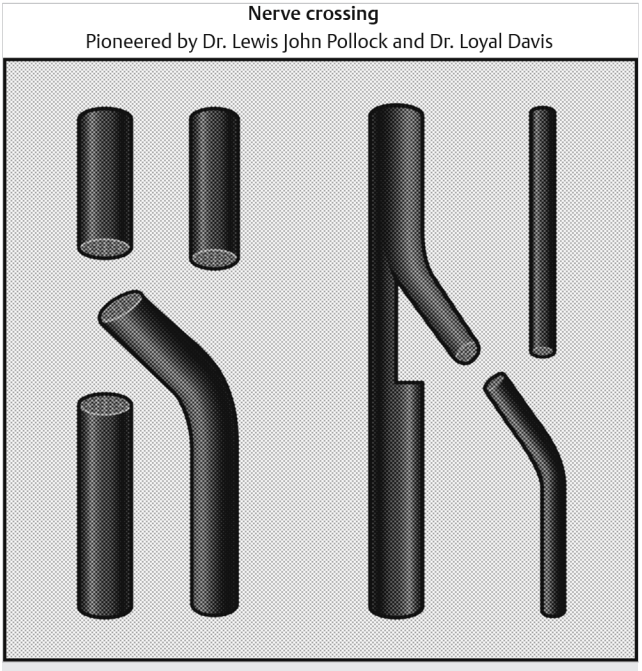


Fig. 1.7 Nerve crossings. In their 1933 textbook *Peripheral Nerve Injuries*, Lewis J Pollock and Loyal Davis described "nerve crossings," which are served as early glimpses of the idea of nerve transfers taking shape. The second panel bears remarkable similarity to fascicular nerve transfers.

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described the incredible intrinsic reserve and ability for functional compensation in patients with chronic motor neuron diseases. These patients had muscle twitch tensions within normal ranges until there was over 90% motor axonal loss.[89] In 1993, *Tessa Gordon* quantified these findings in human and animal models showing that a single motor unit can enlarge up to five times its original size, allowing for compensation of up to 80% motor neuron loss.[90] This also indicated that only reinnervation of a relatively small number of regenerating axons would be necessary to restore function. However, based on *Sir Sydney Sunderland*'s descriptions in 1945, the intraneural topography of the nerves was thought to be too complex and intricate to allow for safe interfascicular dissection.[91,92] He described extensive and unpredictable interfascicular plexal connections with "repeated divisions, anastomosis, and migration of the bundles." Given that the longest segment without topographical changes was only 15 mm, surgeons were reluctant to perform interfascicular dissection and also attributed the poor outcomes of nerve repair to the inability to properly align fascicles.

Michael Jabaley performed detailed anatomic studies of the median, radial, and ulnar nerves in the forearm and changed these views.[93] While he confirmed the findings of interfascicular interconnections, these were not as extensive as previously described, and Jabaley concluded that portions of nerves "proceed for considerable distances with no major change…and thus may be isolated surgically." He also noted the "purposeful" alignment of fascicles that maintained relatively similar positions within the nerve from proximal to distal, despite these intraplexal connections.

In 1993, *Keith Brandt* and *Susan Mackinnon* described the transfer of the medial pectoral nerves to the biceps motor branch for reconstruction of elbow flexion in patients with upper trunk brachial plexus injuries.[94] Four of five patients had ≥ M3 recovery and three achieved ≥ M4 elbow flexion strength, outperforming the results of prior reconstructive strategies. This success solidified the key principle of selecting donor nerves close to the recipient motor end plates to hasten the time to reinnervation. A year later, *Christophe Oberlin* described transferring 10% of the ulnar nerve to the biceps motor branch in patients with C5–C6 root avulsions.[95] This fascicular nerve transfer approach expanded the possibilities of utilizing proximal donor fascicles from within major nerves. While 94% of patients in a follow-up series achieved ≥ M3 elbow flexion strength, a third of these patients still required a secondary Steindler flexorplasty.[96] Given that the brachialis is the strongest elbow flexor, Mackinnon et al described the double fascicular transfer using donor fascicles from the median and ulnar nerves to reinnervate both the biceps and brachialis motor branches.[97]

There is debate whether the Oberlin single fascicular transfer or Mackinnon double fascicular transfer provides better functional recovery.[98,99,100,101] A recent comparative study of 237 patients spanning two decades attributes the addition of the double fascicular transfer as one of the crucial factors leading to more consistent outcomes in recovery of elbow flexion.[101] However, the knowledge that reinnervation of the biceps alone could allow 79% to 88% of patients to regain M3 elbow flexion strength sparked Mackinnon's idea of being able to safely utilize the brachialis as a donor without losing elbow flexion. This should especially be the case in patients with normal elbow flexion, such as patients with lower trunk brachial plexal injuries. Wilson and Mackinnon first described the transfer of brachialis to the anterior interosseous nerve for pinch in such a patient and subsequently also for recovery of pronation.[102] This would also expand the applications of nerve transfers from beyond the brachial plexus to patients with spinal cord injuries. Mackinnon et al described the use of this nerve transfer bilaterally in a patient with ASIA A C7 level injury that same year.[76]

This paradigm shift toward nerve transfers has provided renewed enthusiasm for a surgeon's ability to reconstruct complex nerve injuries and for patients to achieve meaningful functional recovery. Since the mid-2000s, there has been a dramatic increase in the number of publications on nerve transfers, which now surpass those on nerve repair and nerve grafting.[77] This is also paralleled by a similar increase in the number of orthopaedic and plastic surgeons performing nerve transfers,[103,104] the number of novel nerve transfer combinations, and its ever-expanding applications, including spasticity and spinal cord injuries,[105,106] acute flaccid myelitis,[107,108] lower extremity injuries, neuroma and pain mangement,[109,110] neuroprosthetic control,[111] and bionic limb reconstruction.[112]

1.8 Conclusion

The growing trend of interdisciplinary translational research across surgical subspecialties, basic science, and biotechnology will continue to evolve our understanding of the management of complex nerve injuries. Revisiting the history of nerve regeneration through notable textbooks such as those described in ▶ Table 1.1 not only sheds light onto how our understanding of nerve repair and regeneration evolved, but also provides key insights into the fundamentals of nerve surgery.

As Thomas Kuhn writes, scientific truths are not solely established based on objective evidence but are also defined through consensus within a scientific community. From Ranvier's notion of axonal regrowth starting proximally at the cell body, to Millesi's defense of using nerve grafts to bridge nerve gaps, to Mackinnon's use of distal nerve transfers in complex nerve injuries, each major paradigm shift has gone through rigorous scrutiny, criticism, and debate before reaching the tipping point of acceptance. The perseverance of the scientists and innovators outlined in this chapter has led to improved outcomes, affected countless patients, and revolutionized nerve surgery.

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Table 1.1 Notable textbooks prior to the 21st century

Guy de Chauliac	1363	Chirurgia Magna. Republished in 2007 by Rosenman, The Major Surgery of Guy De Chauliac
Leonard of Bertapaglia	1498	Chirurgia sive recollecta super quartum canonis Avicennae. Republished in 1988 by Ladenheim Leonard of Bertapaglia, On Nerve Injuries and Skull Fractures
Gabriele Ferrara	1596	Nova Selva di Cirugia Divisa in Due Parti
Weir Mitchell	1872	Injuries of Nerves and Their Consequences
Etienne Letievant	1873	Traite Des Sections Nerveuses
William Williams Keen	1911	Surgery: Its Principles and Practice
Byron Stookey	1922	Surgical and Mechanical Treatment of Peripheral Nerves
Lewis J Pollock, Loyal Davis	1933	Peripheral Nerve Injuries
Lehmann	1936	Allgemeine Therapie
Learmonth	1943	Aids to the Examination of the Peripheral Nervous System
Walker Earl	1951	A History of Neurological Surgery
Sidney Ochs	1977	The Early History of Nerve Regeneration Beginning with Cruikshank's Observations in 1776. Printed in Medical History, 21:261–274
Sunderland	1978	Nerves and Nerve Injuries
Spinner	1978	Injuries to the Major Branches of the Peripheral Nerves of the Forearm
Eversmann	1988	Entrapment and Compression Neuropathies
Mackinnon and Dellon	1988	Surgery of the Peripheral Nerve
Omer, Spinner, Van Beek	1998	Management of Peripheral Nerve Problems

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