Ian A. Trail
Andrew N. M. Fleming
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Disorders of the Hand

Volume 2: Hand Reconstruction and Nerve Compression

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Preface

In recent years there have been significant advances in the understanding and treatment of disorders of the hand and wrist. This has resulted in a significant improvement in the quality of life for many patients. The authors who have produced this text were chosen as they are hand surgeons who have led many of these exciting developments in the management of both elective and trauma hand surgery. All are internationally respected.

The topics covered are well illustrated with images, radiographs and line drawings and provide practical guidance on surgical procedures. The references at the end of each chapter have been chosen as they are either classic papers or are the most relevant to modern surgical management.

Thus we hope that we have produced a book that will enable improved care for current patients with hand and wrist complaints and inspire surgeons to think in greater detail about treatment options that will provide even better care in the future.

Finally, we would like to thank all the contributors as well as Diane Allmark for her help, but also our families for their patience and support.

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We are also particularly grateful to Springer for allowing us to pursue this project and would like to especially thank Rachel Glassberg for all her helpful advice and prompting.

Finally we would like to thank our secretaries, particularly Diane Allmark, and respective families who, for longer than we dare think, have put up with us reading and re-reading manuscripts on what they think is only a small part of the body!

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Part I

Hand Reconstruction

Nerve Reconstruction

1

Julia K. Terzis and Petros Konofaos

Keywords

Peripheral Nerve Injuries • Nerve Reconstruction • Principles of nerve repair • End-to-end repair • End-to-side-neurrorhaphy • Nerve grafting • Vascularized nerve grafting • Brachial plexus injuries • Avulsion plexopathies • Nerve Transfers

Introduction

Until the late eighteenth century it was believed that peripheral nerves did not regenerate after injury. Introduction of microsurgical techniques [1] in peripheral nerve surgery and the establishment of the principle of tension free repair [2] allowed inspired surgeons such as Narakas, Millesi, Allieu, Brunelli, Terzis, Doi, Gu, and others to suggest several new approaches to nerve reconstruction.

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Many factors influence the success of nerve repair and reconstruction. The age of the patient, the timing of nerve repair, the level of injury, the extent of the zone of injury, the technical skill of the surgeon and the method of repair contribute to the functional outcome after nerve injury. The basic tenets of nerve repair continue to hold true, including an accurate preoperative assessment, properly timed and executed exploration, meticulous nerve repair and intensive postoperative re-education [3].

As soon as nerve injury occurs, its target muscles begin to undergo atrophy and lose their motor end plates. Expedient diagnosis and testing is the best means of maximizing functional return. An adequate and properly timed treatment of peripheral nerve injuries is crucial to achieve a reasonable satisfying clinical outcome, although a complete nerve injury always will lead to varying degrees of permanent dysfunction in adults.

The aim of this chapter is to review the principles and techniques of nerve reconstruction and to discuss the options of repair including direct repair, nerve grafts, end-to-side neurorrhaphy

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and nerve transfers following nerve injuries in the upper extremity.

Background – Aetiology

The hand has been called an extension of the brain, and the sensory and motor performance of the hand is based on adequate function of components in the peripheral as well as the central nervous system. From a hand surgery perspective, poor functional outcomes after peripheral nerve lesions represent a frustrating problem.

Injuries to peripheral nerves are common in all forms of upper extremity trauma but management of them remains a challenge. Common causes include lacerations, fractures, dislocations, ligamentous tears, crush and amputation injuries. Injuries are most often caused by domestic or industrial accidents or interpersonal violence. Nerve injuries range from nerve compression lesions, like carpal tunnel syndrome, up to severe rupture and avulsion of spinal nerve roots of the brachial plexus (BP). Males suffer traumatic nerve injuries at a ratio of 2.2:1 compared with females [4]. The typical patient who sustains a nerve laceration is a male in his late teens or early twenties.

As a protective instinct, the arm, forearm and hand are frequently outstretched during injury. The upper extremity therefore often absorbs the initial impact, with the dominant arm involved slightly more frequently. The most frequently injured nerves are the radial nerves of the index finger, the ulnar digital nerves of the small finger, and the median and ulnar nerves at the wrist level [5].

As far as BP injuries are concerned, high-velocity motor vehicle accidents account for the majority of the cases; most studies report that motorcycle accidents are responsible approximately twice as often as automobile accidents. Nerve injuries in these cases are from traction and compression, with traction accounting for 95 % of injuries. Other common causes include (in different percentages according to different studies) industrial accidents, pedestrian vehicle accidents, snowmobile accidents, gunshot wounds, and other penetrating injuries [6].

Only around 3 % of hand injuries include injury to peripheral nerve trunks. Even a minor injury to a finger causing a digital nerve injury (incidence 6.2/100,000 inhabitants/year) may induce dysfunction of the hand. The consequences of a median or ulnar nerve injury in the forearm are even more wide-ranging for the patient. The injury does not only cause problems in the patient's professional life but leisure activities are also severely impaired.

The overall incidence of BP injuries in multitrauma patients secondary to motor vehicle accidents ranges from 0.67 to 1.3 % [7]. This number increases to 4.2 % for victims of motorcycle accidents. This difference can easily be explained by the increased forces applied to the BP of the unprotected body during a high velocity motorcycle accident.

Presentation

Pathophysiology

Following peripheral nerve injury, morphologic and metabolic changes occur. Within the first few hours to days, morphologic changes occur in the corresponding neurons, including swelling of the cell body, displacement of the nucleus to the periphery, and disappearance of basophilic material from the cytoplasm, a phenomenon termed chromatolysis.

Within 2–3 days of injury, edema forms in the axonal stumps and the distal stump undergoes Wallerian degeneration. This degenerative process is called Wallerian degeneration after Augustus Waller, who first characterized morphological changes in the distal stump of sectioned frog glossopharyngeal and hypoglossal nerves 160 years ago [8]. During Wallerian degeneration, Schwann cells from the distal stump proliferate, help inflammatory infiltrating cells to eliminate debris, and upregulate the synthesis of trophic (factors which support neuronal survival and axonal growth) and tropic (factors which influence the growth direction of the regenerating axons) factors. The Schwann cells, close to the site of transection, go through the same type of changes as the Schwann cells in the distal nerve segment.

After 3–6 weeks, endoneurial tubes are left behind that consist of basement membranes lined with Schwann cells which proliferate and organize into columns, guiding the regenerating axonal sprouts within the basement membranes to their targets. In the gap between the proximal and distal nerve segment an inflammatory response occurs and a fibrin matrix, filled with macrophages, is formed. Schwann cells can migrate from both ends where the migration of such cells takes part in concert with the outgrowing axons. Metabolic changes within the neuronal cell body involve switching the machinery normally set up to transmit nerve impulses to manufacturing structural components needed for reconstruction and repair of the damaged nerve.

End organs also undergo changes after nerve injury. Complete atrophy occurs within 2–6 weeks of denervation. Fibrosis occurs in motor fibers at 1–2 years and fragmentation and disintegration occur by 2 years. It is generally agreed that functional recovery is diminished if the nerve does not reach the motor end-plate by 12 months. Sensory end-organs are less sensitive to denervation than motor end-organs. It has been shown that recovery of protective sensibility is possible even after many years from nerve injury [9] but that the degree of functional sensation decreases the longer the delay in nerve repair.

Classification of Nerve Injury

In 1941, Cohen introduced a classification to describe nerve injuries which was later popularized by Seddon [10]. According to this, there are three distinct clinical entities for a dysfunctional nerve: neurapraxia, axonotmesis or neurotmesis.

Neurapraxia, refers to a localized conduction block, is a comparatively mild injury, with motor and sensory loss but no evidence of Wallerian degeneration. The nerve distally conducts normally. Tinel's sign (a tingling sensation perceived distally when percussion is carried out over the injury site of a nerve which indicates involvement or in a partial lesion the commencement of regeneration as the nerve attempts to heal) [11]. The underlying mechanism is attributed to focal demyelination or ischemia. Recovery may occur within hours, days, weeks or up to a few months.

In axonotmesis the axons are ruptured, but the epineurium and perineurium remain intact. It is commonly seen after crush injuries. Wallerian degeneration does occur distal to the injury, but regeneration from the proximal stump is still possible. Functional recovery depends on the severity of the lesion and the degree of internal disorganization in the injured nerve as well as its distance to the end organ.

Neurotmesis describes the situation in which the entire nerve trunk is completely ruptured and axonal continuity can not be restored. Sharp injuries, some traction injuries or injection of noxious drugs are the most common causes. Prognosis for spontaneous recovery is extremely poor without surgical intervention.

In 1951, Sunderland [12] expanded upon Seddon's classification system by defining five distinct degrees of nerve injury. Sunderland's 3rd and 4th degree injuries were included as extensions of axonotmesis and neurotmesis respectively.

First degree injury (neurapraxia) is a localized conduction block with preservation of the nerves'anatomical continuity. Although recovery is complete, the time required varies from days to 3 months.

In second degree injury (axonotmesis) the endoneurium and the perineurium remain intact. A Tinel's sign is present. Wallerian degeneration occurs distal to the site of injury. Nerve recovery may be complete.

Third degree injury involves endoneurial scarring and disorganization within the fascicles. The endoneurial tube is disrupted, resulting in erroneous alignment of the regenerating fibers. An advancing Tinel's sign indicates the level of regeneration, but the degree of recovery will not be complete.

In fourth degree injury the nerve is in continuity, but regeneration does not occur across scar block. A Tinel's sign is found at the level of the injury, but does not advance further. It is commonly caused by severe stretch, traction, crush, cautery injury or nerve injection. Surgical intervention is necessary.

In fifth degree injury there is severance of the nerve trunk. Recovery is not possible without surgical intervention. This lesion is associated with penetrating trauma.

Preoperative Investigation and Diagnosis

The formulation of a diagnosis, treatment plan, and prognosis can be largely accomplished by means of a careful and detailed history and physical examination. The timing of the injury will help guide treatment recommendations, which the mechanism gives clues about the severity of the lesion.

The examination of passive range of motion of all joints of the affected extremity should be done and recorded before examination of active range of motion. All the upper extremity muscles have to be tested and compared to corresponding ones on the contralateral normal side. The grip and pinch muscle strength are measured using a Preston dynamometer set on intermediate position. The sensory evaluation should include the supraclavicular area, the arm, the forearm, and the hand. Color and trophic changes of the arm should be observed. For evaluation of sensibility in the hand, static and moving two-point discrimination (needs to take place with the patient sitting across from the examiner and having the eyes closed), Semmes-Weinstein monofilament pressure testing or von Frey cutaneous pressure threshold testing, testing for perception of high- and low-frequency vibration, and ninhydrin testing should be performed.

A detailed history of pain, its onset, duration, quality, sharpness, and radiation is routinely recorded. The results are recorded on a BP chart (Fig. 1.1) which includes all muscle groups of the

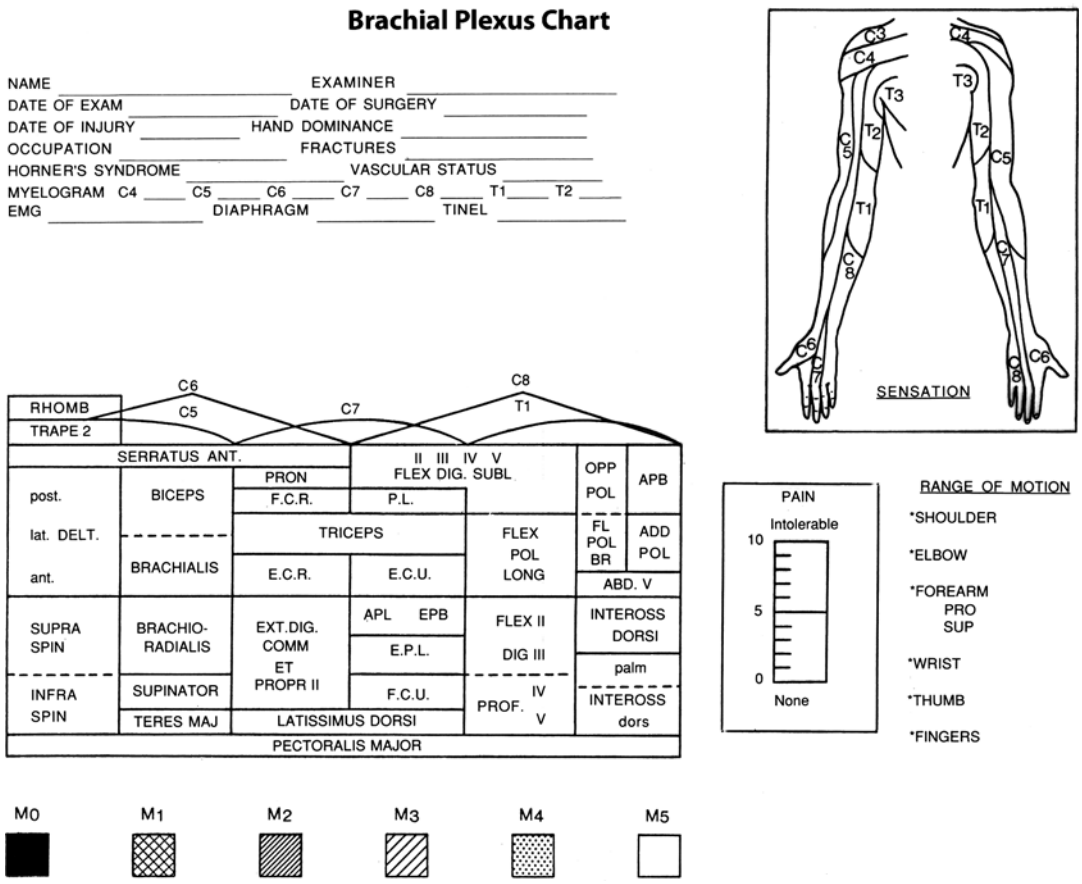


Fig. 1.1 Preoperative brachial plexus chart

upper extremity, sensory mapping, and pain level. This is important not only for the initial visit, but also to document and follow clinical recovery after repair.

The British Medical Research Council grading scale is used by most physicians. This system has been further modified by Terzis [13] with intermediate grades of (+) and (−).

In cases of BP injuries, the presence of Horner's sign is a strong indicator of avulsion of the C8 and T1 roots. Moreover, the absence of a Tinel's sign in the supraclavicular area is a strong indicator of root avulsion and is a bad prognostic sign because it indicates lack of intraplexus donors for reconstruction. On the other hand, a positive Tinel's sign is a strong indicator of roots connectivity with the spinal cord.

The initial electrodiagnostic evaluation of the upper extremity should include needle electromyography and nerve conduction studies. Axonal discontinuity results not only in predictable pathologic features but also in time-related electrical changes that parallel the pathophysiology of denervation. Wallerian degeneration results in the emergence of spontaneous electrical discharges for at least 3 weeks after the injury. Therefore, a needle electromyogram should be postponed for at least that long and preferably carried out at 6 weeks.

The lamina test is performed in cases of adult BP injuries. Tiny volleys of electrical stimulation are applied at the level of each foramen on each exiting root to determine whether the patient perceives the area of the dermatome innervated by this root. A positive response would be strong evidence against avulsion.

Depending on the mechanism of injury and the location of the nerve lesion, radiologic imaging may be necessary to confirm or support a diagnosis of a nerve injury. In cases of BP injuries, imaging studies (such as myelography, CT myelography, and magnetic resonance imaging) are used in order to detect abnormalities of the nerve roots (such as traumatic pseudomeningocele, deformity of nerve root sleeves, dural scar, and nerve root avulsion). A combination of myelography with computed tomography of the cervical spine is used to identify root avulsions (Fig. 1.2). In case of previous vascular injury and subsequent reconstruction, angiography should be employed to investigate the blood supply of the extremity and to identify any vascular compromise (Fig. 1.3).

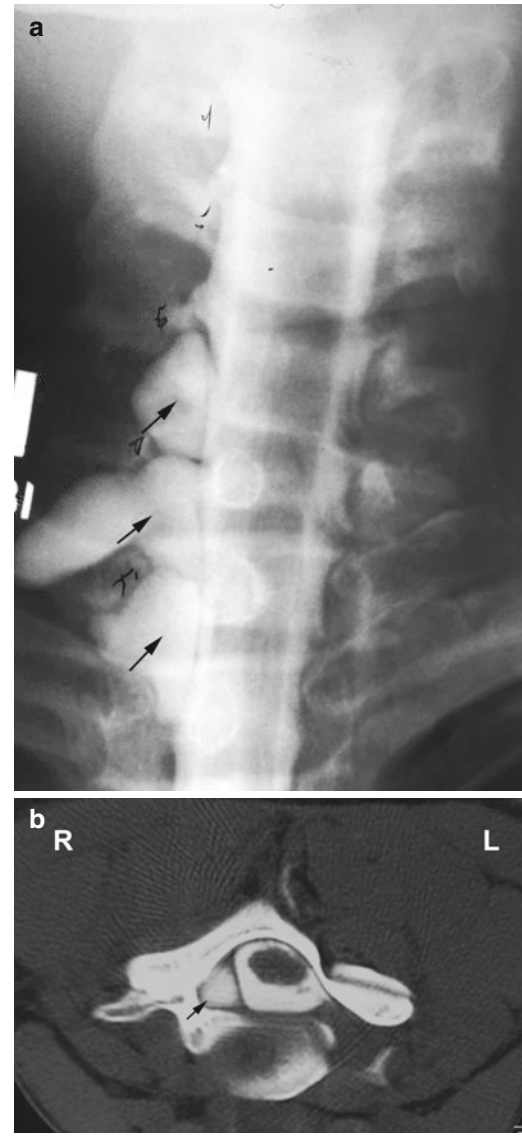


Fig. 1.2 CT Myelography showing root avulsion. (**a**) Myelography of the Cervical spine in a patient with multiple root avulsions (*arrows*). (**b**) Example of CT myelography in a patient with severe right brachial plexus injury. Note avulsed root on the right (*arrow*)

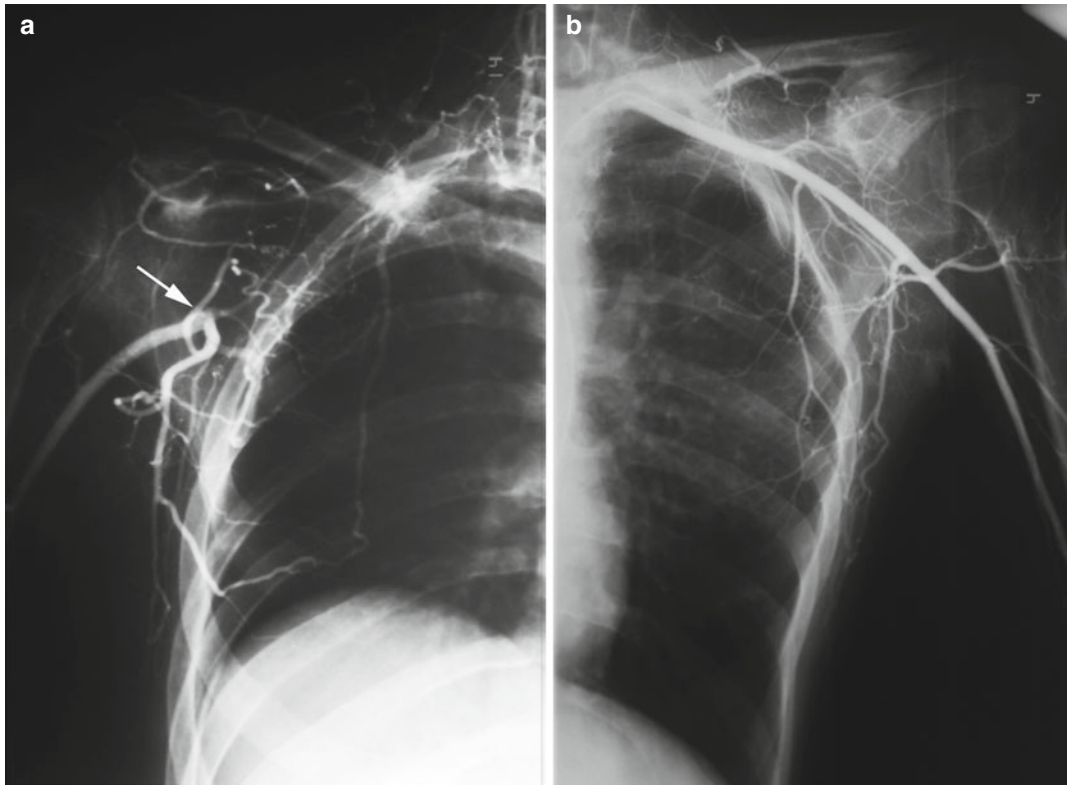


Fig. 1.3 Angiography of upper extremity in cases of vascular injury. (**a**) Angiography of right upper extremity. Note interruption of (*R*) subclavian artery (*arrow*). Axillary artery receives flow from collateral vessels. (**b**) Normal angiography of left upper extremity

Treatment Options

Principles of Nerve Repair

The basic principles of nerve repair include a sequence of eight basic principles that represent the basis of the microsurgical management of the nerve injured patient [14]:

1. Preoperative assessment of motor and sensory function
2. Adequate debridement of the proximal and distal nerve stumps in order to allow nerve regeneration to proceed across the repair site
3. Utilization of microsurgical techniques
4. Tension-free repair
5. When a tension-free repair is not possible, use of other techniques for nerve repair; nerve grafts, end-to-side nerve repair or nerve transfers
6. Primary repair; when this is not possible, delay repair for approximately 3 weeks when the 'zone of injury' is clarified
7. Utilization of a nerve repair technique that allows early protected range of motion to permit nerve gliding
8. Occupational and physical therapy in order to maximize the clinical outcome

Timing of Nerve Repair

A primary nerve repair is defined as reconstruction shortly after the injury. Secondary repair is defined as occurring at a later period after injury. Several investigators have reported that nerve repair is better when performed within 6 weeks of injury and several studies have shown primary repair to be superior to secondary repair as long as the tissue bed is adequate [15, 16].

In general, nerve injuries associated with open wounds require early exploration except from gunshot wounds, which are more appropriate to be treated as closed or blunt trauma. In crush nerve lesions or injuries associated with

significant soft tissue damage it can be difficult to estimate the extent of the zone of injury. In these cases, a delayed repair, after 3 weeks, is indicated, when the zone of injury becomes better demarcated and the extent of scar tissue can be easily defined.

In closed or blunt trauma, initial management is expectant with close observation. If complete recovery is not observed within 6 weeks, electrodiagnostic studies should be obtained for baseline evaluation. If at 12 weeks complete recovery has not occurred, repeat electrodiagnostic studies should take place. Presence of increase of motor units potentials in electromyography is an indicator that spontaneous reinnervation most likely will follow. Lack of signs of reinnervation (clinical or electrical) at 12 weeks post injury requires surgical exploration.

BP injuries are worth specific consideration regarding the timing of exploration and reconstruction. Such injuries require extra care since BP injuries usually come with other associated injuries including fractures, vascular injuries and associated soft-tissue injury. Although exploration of the BP injury may need to be performed with a slight delay, the modern management of BP injuries is early aggressive microsurgical reconstruction [17].

Techniques of Nerve Repair

In general, nerve exploration and repair should be performed under high magnification of the operating microscope. Exploration always takes place proximal and distal to the lesion site until normal nerve to inspection and palpation is encountered. If the history and physical examination is suspicious of double level injury then the entire length of the nerve needs to be explored. The ideal scenario for nerve repair is end-to-end coaptation of the nerve stumps.

The procedure of repairing a nerve trunk can be divided into four steps. After the zone of injury is defined, the nerve endings are cut back to healthy fascicles. Then, the nerve ends are approximated keeping in mind the importance of considering the length of the gap and possible tension at the coaptation site. If additional nerve length is required, releasing constricting fascia, dividing adventitia attachments, dissecting any tethering bands, transposing nerves (e.g. ulnar at elbow) and flexing neighboring joints (e.g. wrist for median and ulnar lesions in Zone 5) will mobilize the nerve further. Tensionless repairs have demonstrated superior results. Exceeding 10 % of the resting length of the peripheral nerve has been shown to decrease blood flow to the nerve by 50 % [18]. Tension is assessed intraoperatively to determine the need for grafting. A good rule of thumb is that if nerve ends can be approximated with 8-0 sutures, then grafting is not required.

The next step is the correctly aligned coaptation of the nerve ends. Last step is the maintenance of nerve repair with microsutures (9-0 or 10-0 nylon) which are inserted into the epineurium. Placement of the sutures should avoid malrotation of the nerve ends.

Epineurial repair has been shown to have similar functional results to group fascicular repair in smaller, more distal nerves [19]. Group fascicular repair is preferred in larger nerves where motor and sensory fasicles can be accurately matched (most notably the ulnar nerve below the elbow). The cross-sectional appearance of the proximal and distal stumps should be carefully inspected under high magnification prior to proceeding with the nerve repair.

The accuracy of nerve apposition at the repair site influences the functional restoration. Presently, anatomic axon-to-axon reconnection and normal restoration of function after significant nerve injury remain an unobtainable goal. Electrophysiologically-aided motor- and sensory- fascicle differentiation has been an important tool that facilitates our ability to depict the intraneural composition of sensory and motor bundles prior to nerve coaptation [20]. In 1976, Williams and Terzis [21] introduced single fascicular recordings as an intraoperative diagnostic tool for the management of peripheral nerve lesions in continuity which was a new method of sophisticated intraoperative differentiation between motor and sensory components.

Several histochemical methods have been developed to permit differentiation of motor and sensory fibers. The enzyme carbonic anhydrase

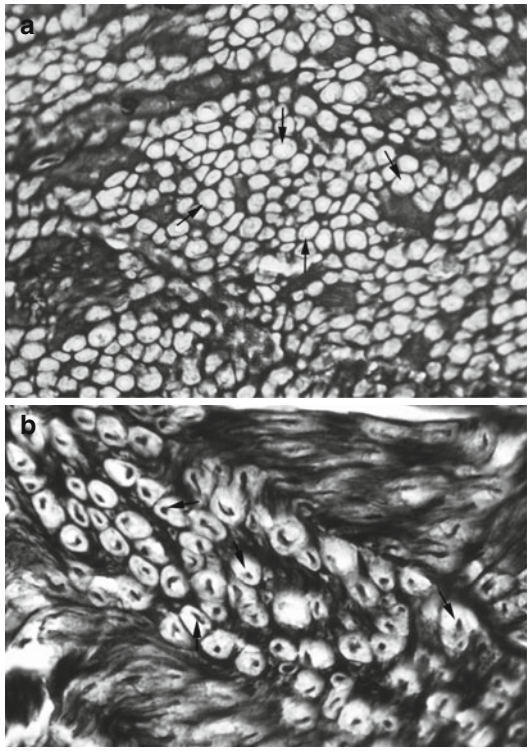


Fig. 1.4 Example of Carbonic Anhydrase staining. (**a**) Cross section of a motor fascicle. Note lack of axonal staining with the carbonic anhydrase (*arrows*). (**b**) Cross section of a sensory fascicle. Note dark staining of the axons (*arrows*)

can differentiate between motor and sensory fascicles of peripheral nerves [22] (Fig. 1.4). The application of this staining method to human peripheral nerve was first described by Riley and Lang in 1984 [22] and later modified for widespread clinical use by Carson and Terzis in 1985 [23]. Although it can provide a convenient method for identifying predominantly sensory versus motor fascicles in cut ends of peripheral nerves, its use depends on the surgeon's experience, available operating time and existence of an experienced laboratory in nerve histochemistry. Acetylcholinesterase histochemistry was also used in conjunction with peripheral nerve surgery, this enzyme in contrast to carbonic anhydrase, is present only in motor fibers [24].

End-to-End-Repair

The surgeon should be familiar with the various techniques available and tailor them to the situation, taking into account which nerve is injured and the level of the injury in the upper extremity. The basic choices include epineurial repair, group fascicular repair, fascicular repair or a combination of those techniques. The goal is to achieve tension free coaptation and proper alignment.

In the epineurial repair, coaptation is achieved by single epineurial stitches in the epineurium along the circumference of the nerve. A perfect superficial alignment can be achieved using epineurial vessels as landmarks, but the internal orientation of fascicular bundles and individual fascicles may not be correct. This method is indicated when one or only few fascicles are injured and is appropriate for distal nerve repairs (digital nerves).

In group fascicular repair, fascicular groups are coapted with single sutures in the perineurium or perifascicular connective tissue which surrounds groups of fascicles. Prior to coaptation, the fascicular groups need to be identified and matched together. In large nerves with multiple fascicles, nerve regeneration can be enhanced by use of this technique.

In fascicular repair, coaptation of individual fascicles is achieved by 10-0 or 11-0 microsutures in the internal epineurium surrounding individual fascicles. This type of repair is not feasible unless it can be performed with minimal tension.

End-to-Side Nerve Repair (Fig. 1.5)

The idea of end-to-side nerve repair was popularized by Viterbo et al. in 1992 [25] after its introduction a century ago [26]. This technique allows for additional muscle reinnervation with minimal detriment to donor-nerve function [25]. Using this technique a neurorrhaphy is created between the proximal end of an injured nerve and the side of an uninjured donor nerve by simple microsurgical attachment at the site of a window (epineurial and/or perineurial window).

The efficacy of end-to-side neurorrhaphy has been established in several rat models. Noah et al. [27] suggested that more axons went through the coaptation site when a perineurial window or partial neurectomy was created in the donor-nerve prior to coaptation vs leaving the perineurium or epineurium intact. Okajima et al.

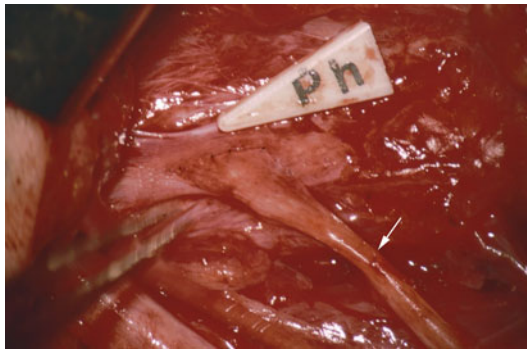


Fig. 1.5 Example of end-to-side nerve repair. Example of an end-to-side neurorrhaphy in an obstetrical brachial plexus case. An epineurial and perineurial window has been made on the phrenic nerve. An interposition nerve graft (*arrow*) is coapted by end-to-side repair at the site of the window. The nerve graft is targeted to neurotize the musculocutaneous nerve (not shown). Because an end-to-side coaptation was used there is no downgrading of the function of the ipsilateral diaphragm

[28] studied the early regenerative response after end-to-side neurorrhaphy and were able to identify increased nodal sprouting proximal to the perineurial window and/or partial neurectomy groups vs the intact epineurium group.

In clinical practice, Terzis [29] used end-to-side neurorrhaphy extensively in order to minimize morbidity from the various extraplexus donors. Thus, only the number of donor fibers needed are taken, such as in partial phrenic or partial hypoglossal transfers, which are used in combination with an end-to-side coaptation via an interposition nerve graft especially in cases of facial paralysis and obstetrical BP reconstruction.

Nerve Grafting (Fig. 1.6)

When tension-free repair is not possible, a suitable alternative must be pursued. The surgical technique employed in these alternatives is similar, whether it be a nerve graft or nerve transfer.

Nerve grafting has long been considered the 'gold standard' for repair of irreducible nerve gaps. The choice of autogenous graft is dependent on several factors: the size of the nerve gap, location of proposed nerve repair, and associated donor-site morbidity.

Before grafting, the proximal and distal nerve stumps must be prepared to normal tissue outside of the zone of injury. In cases of polyfascicular nerve stumps, interfascicular dissection is preferred in order to prepare corresponding fascicular groups. The intraneural topography of both nerve stumps is obtained by means of intraoperative electrodiagnostic studies and carbonic anhydrase histochemistry.

Then, the defect size is measured and the nerve grafts are harvested. The nerve grafts are then tailored so that they bridge corresponding fascicular groups. The proximal end of each graft is coapted to the proximal fascicular group and its distal end to the corresponding distal bundles.

Selection of the graft donors is limited by the availability of donor nerves and the functional and aesthetic deficits created by their harvest. According to Sunderland and Roy [30] the ideal donor-nerve should possess the following characteristics:

1. the sensory deficit should occur in a non-critical area of the body
2. the donor-nerve should possess long, unbranched segments
3. the donor-nerve should easily be accessible and reliably located
4. the donor-nerve should be of overall diameter and possess large fascicles with little interfascicular connective tissue and few interfascicular connections

The commonly used donor-nerves available for grafting are typically the sural nerve, the saphenous nerve, the medial brachial cutaneous nerve and the lateral antebrachial cutaneous nerve.

Vascularized Nerve Grafts (Fig. 1.7)

The first vascularized nerve graft in the upper extremity was a pedicled nerve graft in 1945 by St. Clair Strange for reconstruction of large nerve defect: the ulnar nerve was transferred in two stages to reconstruct the median nerve [31]. Taylor et al. [32] used the superficial radial nerve as a vascularized nerve graft, to repair a large defect of a median nerve.

In 1984, Breidenbach and Terzis [33] defined the blood supply of peripheral nerves that could be used for microvascular transfer and introduced a classification of the blood supply of nerves

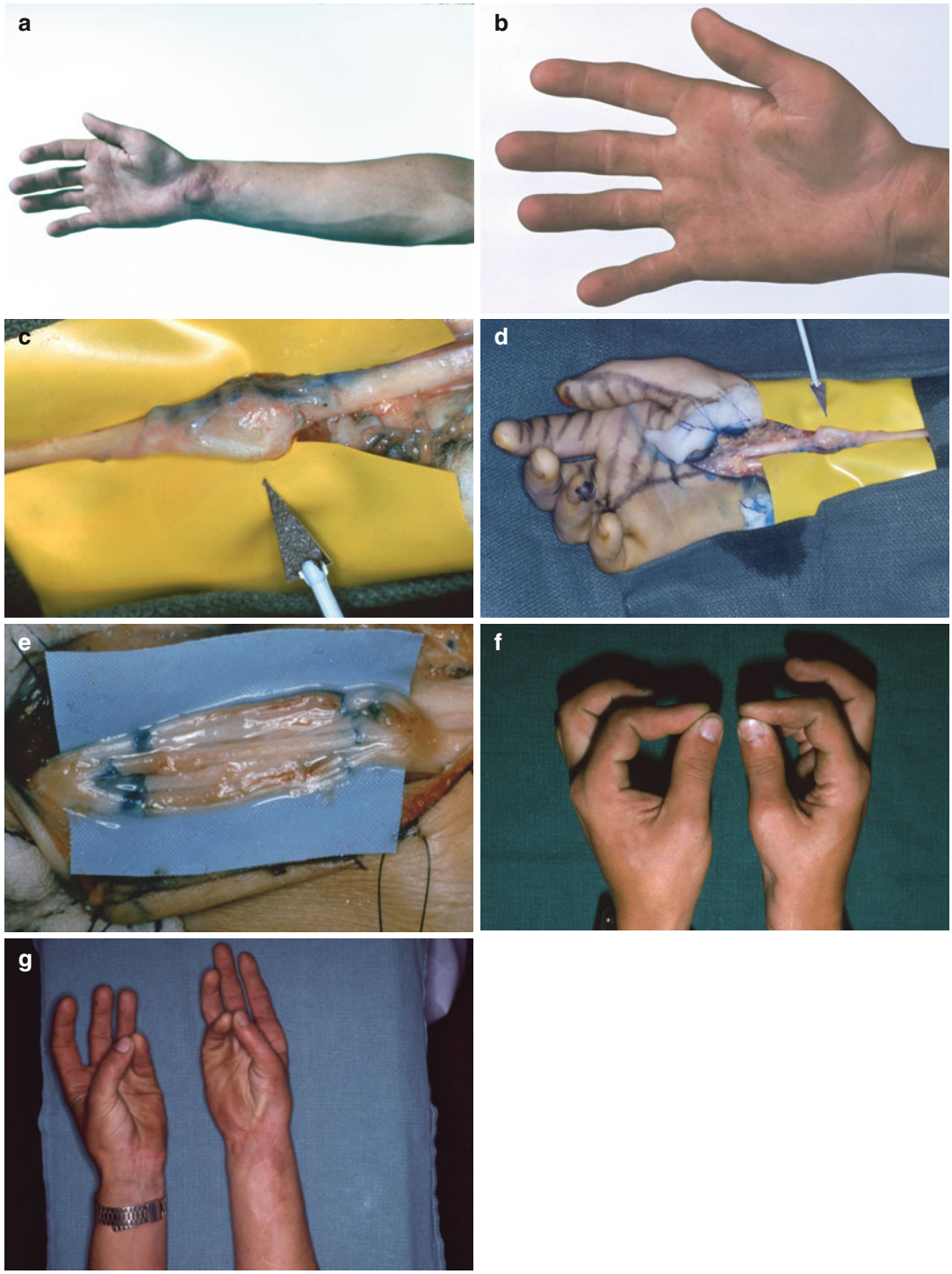


Fig. 1.6 Example of a case treated with interposition nerve grafting. A 19 year old boy was involved in an accident in which he sustained a glass laceration of the volar aspect of his right dominant wrist. He presented 18 months later to our Center with complete anesthesia of the thumb, index and radial side of the middle finger and had no thumb opposition (**a**, **b**). On exploration, a large median nerve neuroma was present (**c**, **d**). The neuroma was excised and the defect was reconstructed with five interposition sural nerve grafts (**e**). Eight months later, he also had opponensplasty which involved transfer of the sublimis tendon from the ring finger to the thumb to augment opposition. Upon follow-up the patient showed adequate pinch (**f**) and strong thumb opposition (**g**). Sensory return to the radial side of his hand has been satisfactory, enabling him to return to his previous work

based on the number of dominant vascular pedicles.

The clinical indication for a vascularized nerve graft is a scarred recipient bed that will not support a nonvascularized nerve graft. In cases of long gaps, vascularized nerve grafts can be placed in association with nonvascularized nerve grafts to cover the cross-sectional area of the injured nerve. The obvious advantage of this technique is the ability to provide immediate intraneural perfusion in a poorly vascularized bed and to reconstruct large nerve defects.

The use of vascularized nerve grafts is particularly important in BP surgery. In cases of avulsion of the C8 and T1 roots, the ulnar nerve should be used as a vascularized nerve graft for ipsilateral plexus reconstruction or as a cross-chest nerve graft from the contralateral C7 root for neurotization of the denervated upper extremity [34] (Fig. 1.8).

Breidenbach and Terzis [35] first reported that the ulnar nerve can be transferred in its total length on the superior ulnar collateral vascular pedicle (Fig. 1.9). Terzis subsequently reported a series of 151 vascularized ulnar nerve grafts for posttraumatic BP palsy patients [34]. According to this study, pedicled or free vascularized ulnar nerve grafts achieved superior results compared to those obtained with conventional nerve grafts.

Technique

Using this technique, the ulnar nerve with its supplying vascular pedicle is transferred as a pedicle or free vascularized nerve to bridge several nerve defects. The vascular pedicle is anastomosed to an artery and a vein of the recipient site and subsequently the nerve coaptations take place. The vascularized ulnar nerve graft is folded into segments maintaining their vascular connections according to the technique proposed

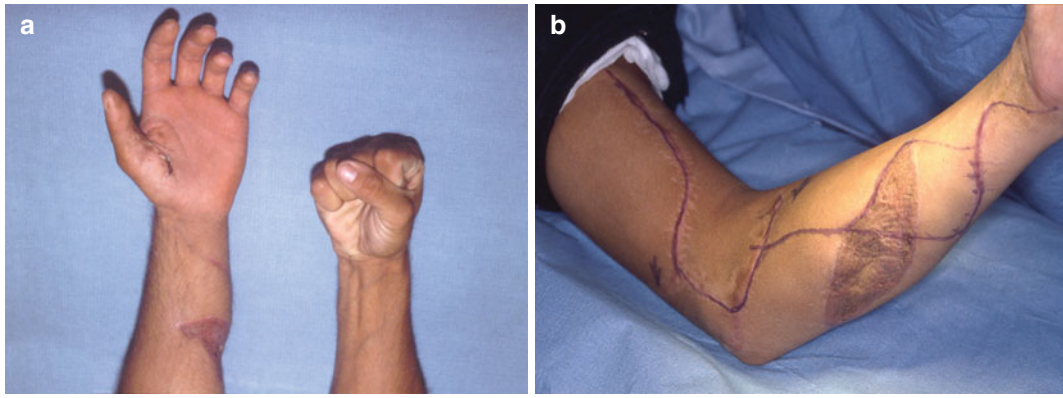
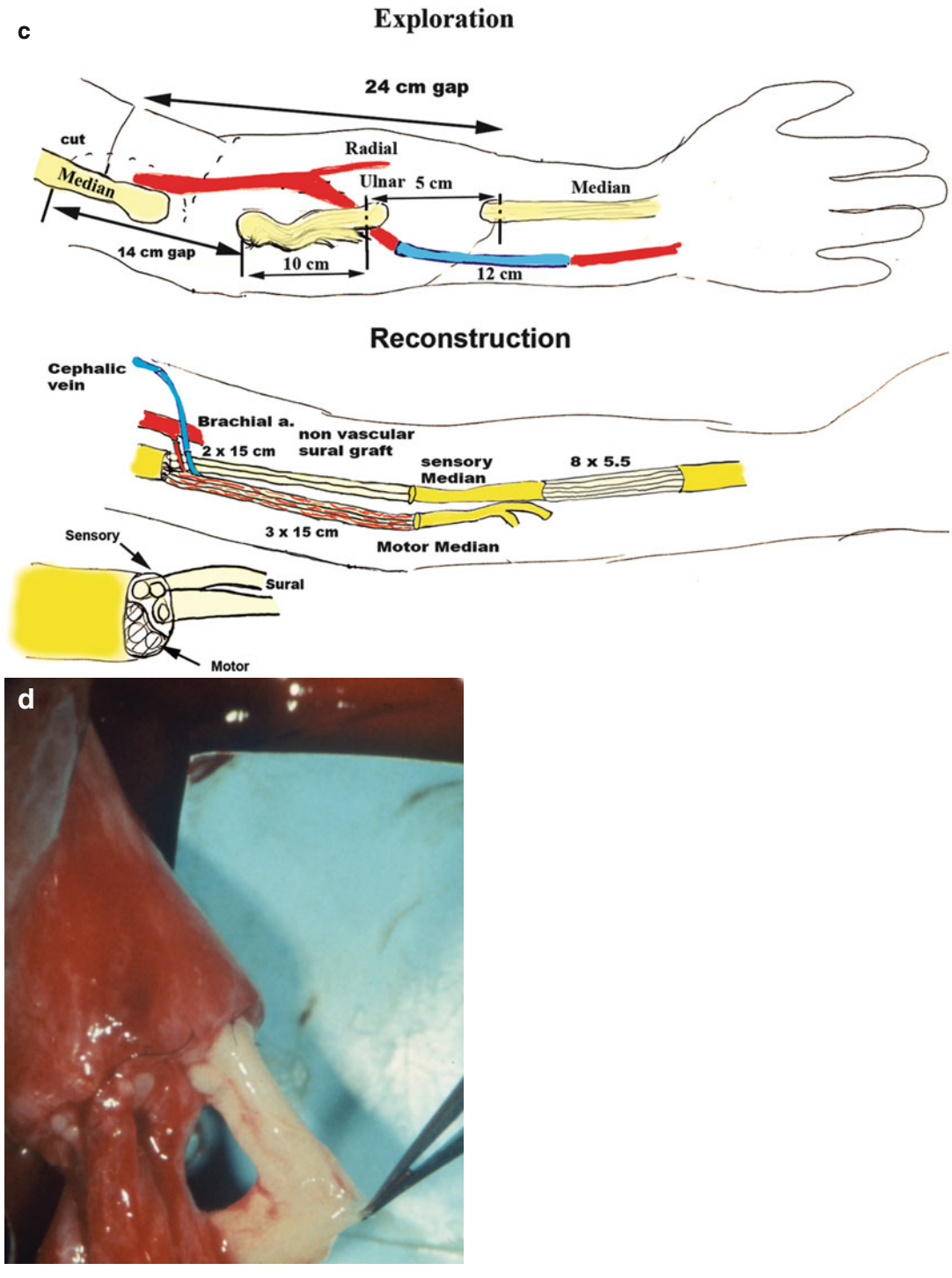


Fig. 1.7 Example of a case with Vascularized Nerve Grafts. This is a 23 year old male who was involved in a boating accident in which the propeller of a motor boat ran over his left arm. He was taken emergently to a local hospital where he was noted to have severe neurovascular injuries as well as tissue loss of the left forearm. He received elsewhere emergency revascularization of his left extremity with the use of saphenous vein grafts. He also had multiple levels of nerve injuries of the left ulnar and median nerve. Preoperative view of the patient (**a**, **b**). Three months later, he underwent reconstruction of his left median nerve which was transected at four levels (**c** above). The sensory part of the superficial and deep peroneal nerves based on their common vascular supply was harvested and used to reconstruct the motor portion of the median nerve (3 × 15 cm, one deep and two superficial peroneal nerve grafts). Nonvascularized sural nerve grafts were used to reconstruct the sensory portion of the median nerve (2 cables × 15 cm proximally and 8 cables × 5.5 cm distally: **c** below). Close-up of the proximal coaptation: vascularized nerve grafts on the left, nonvascularized sural nerve grafts on the right (**d**). Seven months after the injury he underwent reconstruction of the left ulnar nerve utilizing vascularized saphenous nerve graft (1 cable × 30 cm) for the motor portion of the ulnar nerve and sural nerve graft for the sensory component of the ulnar nerve (**e**). Four years postoperatively, we can see very good results. Powerful finger flexion, thumb opposition, and intrinsic function (**f–i**). He can easily pick up a can of soda (**j**) and has never had any morbidity in the donor extremity (**k**) (Requested permission from: Terzis and Kostopoulos [67])



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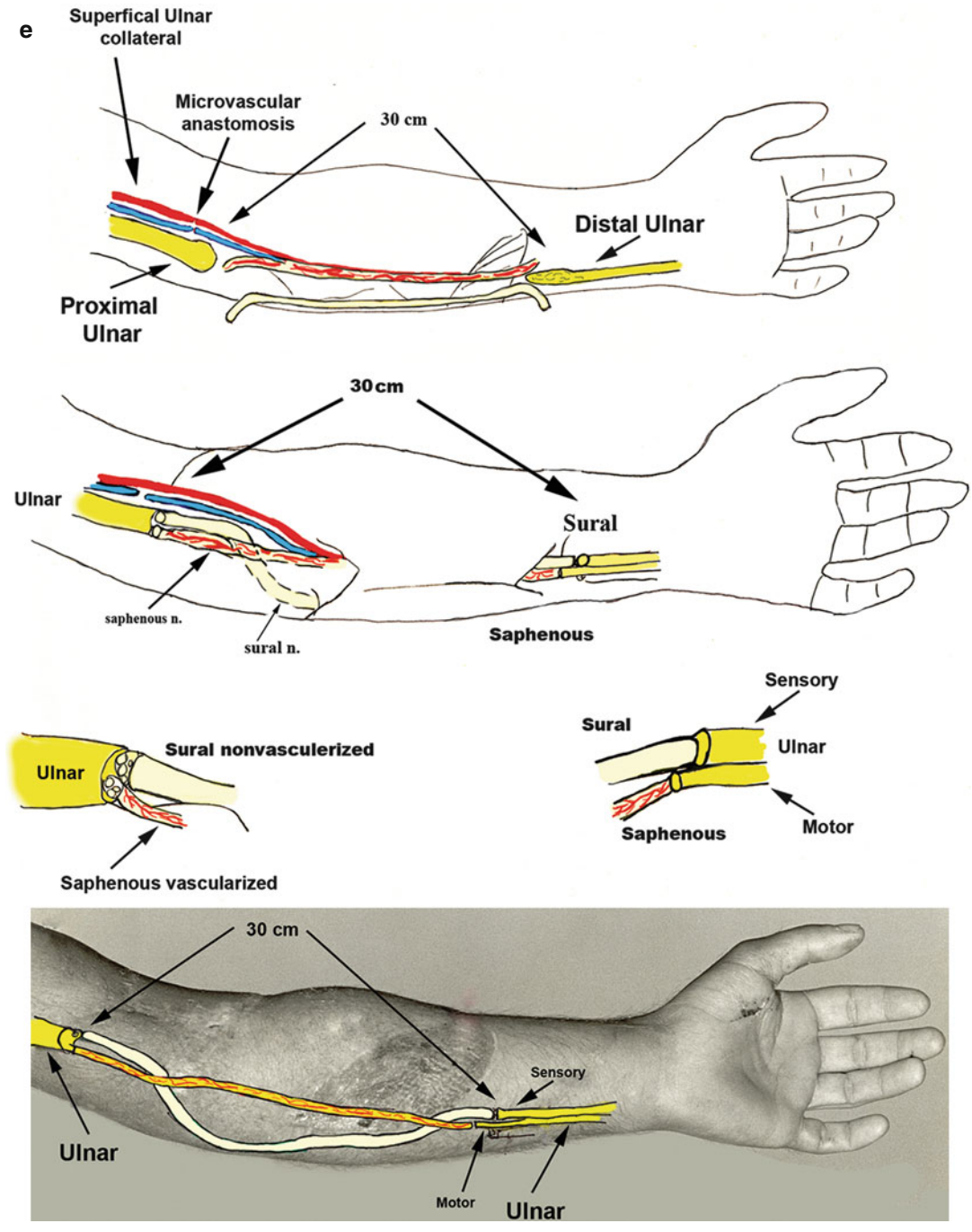

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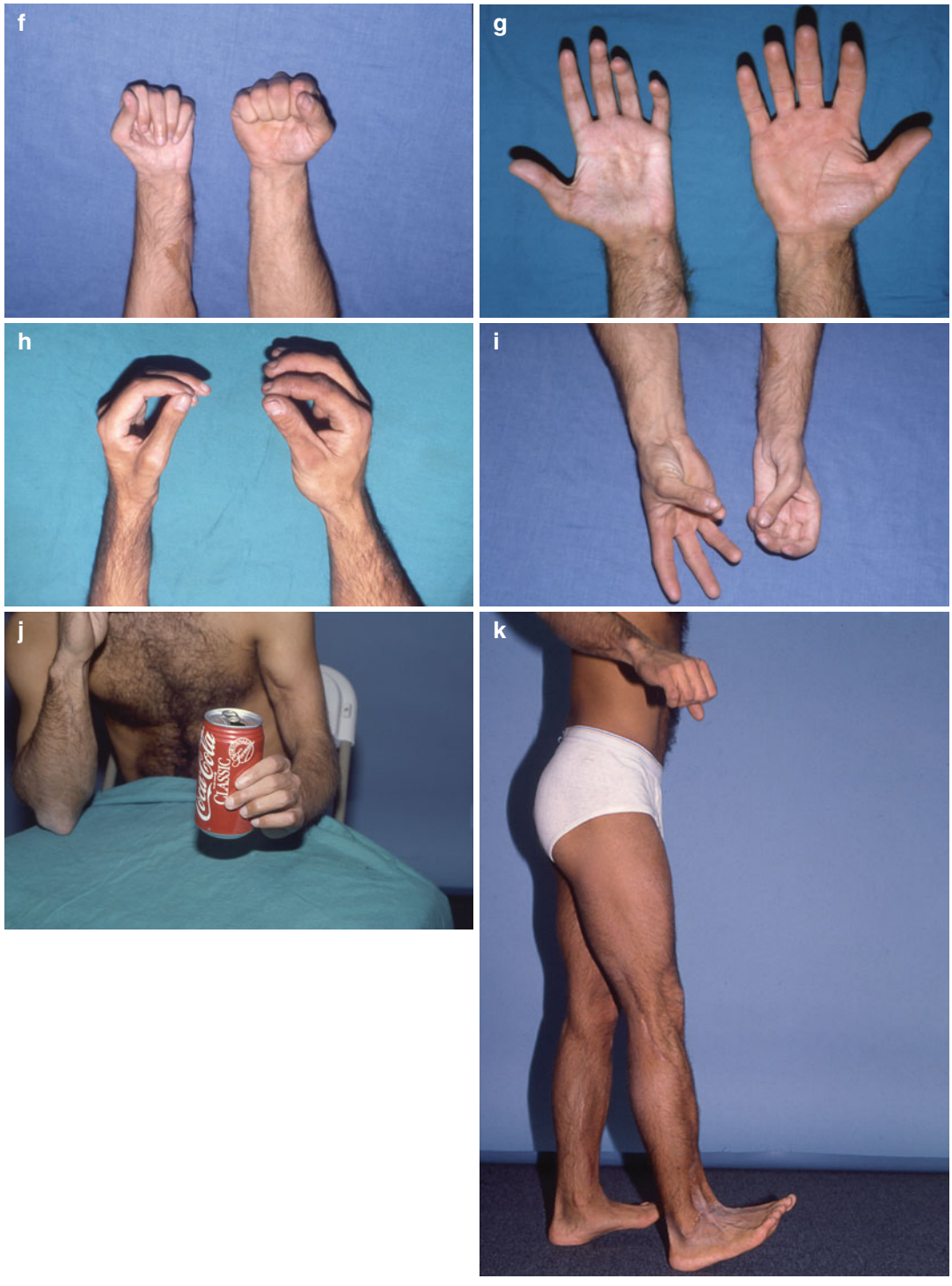

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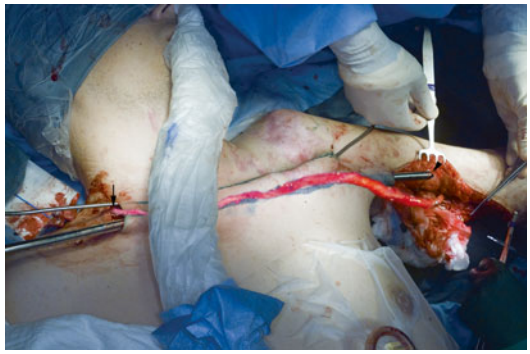

Fig. 1.8 Example of a cross chest vascularized ulnar nerve graft. Cross chest vascularized ulnar nerve graft prior to tunneling. The proximal ulnar will be coapted to the anterior division of the right C7 root. The distal ulnar nerve will be coapted to the median nerve of the left paralyzed extremity. *Arrow* points to the metal "passer" that will be used to transfer the nerve across the chest

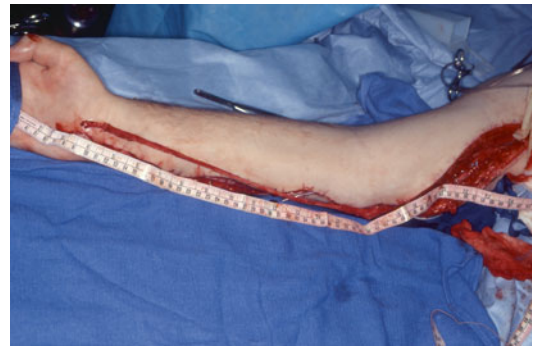

Fig. 1.9 Example of ulnar nerve harvested as a VNG next to the arm. Exploration of the right vascularized ulnar nerve graft prior to microvascular transfer. The entire length of the nerve receives its blood supply from the superior ulnar collateral vascular pedicle. Terzis' method for the use of the free vascularized ulnar nerve for ipsilateral intraplexus reconstruction. The epineurium is transected longitudinally without compromising the longitudinal epineurial blood supply and the fascicles are transected transversely. The blood supply is maintained through the folded epineurium

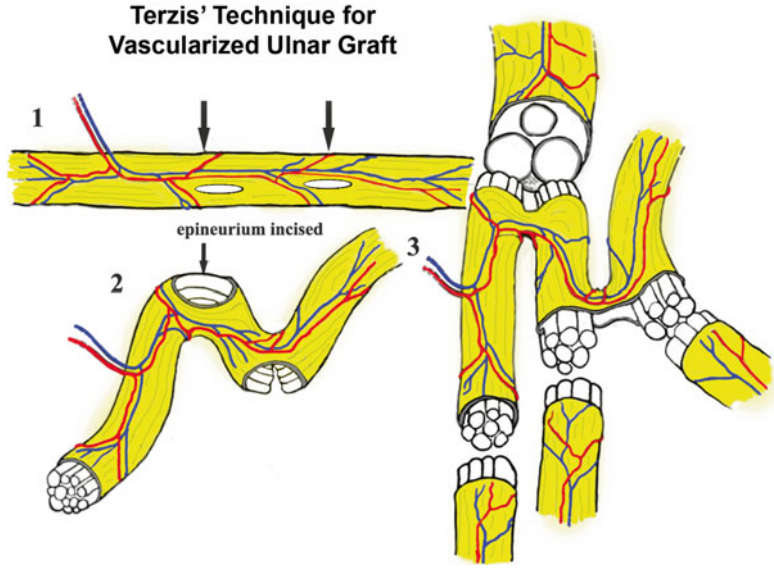

Fig. 1.10 Terzis' technique of using the vascularized ulnar nerve for ipsilateral BP reconstruction. Terzis' technique of folding the vascularized ulnar nerve graft into segments while maintaining its blood supply by preserving the integrity of epineurial vessels

by Terzis and Kostopoulos [34] (Fig. 1.10). In this situation, the longitudinal blood supply of the epineurium of the ulnar nerve is preserved while the intraneural contents are transected to address the bridging nerve defects, thus maintaining excellent blood supply throughout the vascularized ulnar nerve graft. In more distal lesions, vascularized fascia can be used to improve the blood supply of the underlying bed by enveloping the nerve reconstruction (Fig. 1.11).

Ulnar Nerve

Cases of global plexopathy with avulsion of the lower roots and rupture of the upper roots provide the best indication for using the ipsilateral ulnar as a vascularized graft for BP reconstruction. The ulnar nerve can be harvested on the superior ulnar

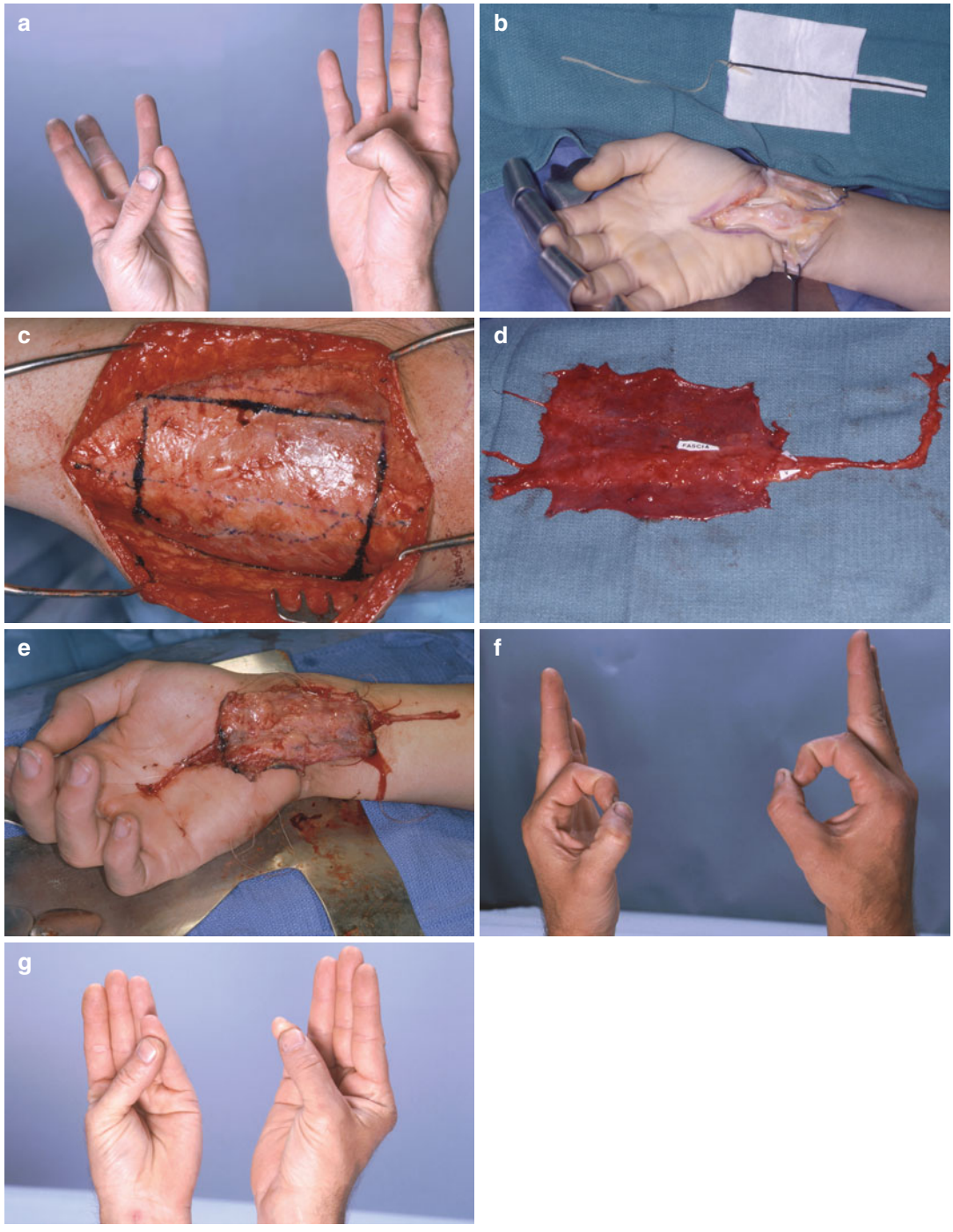

Fig. 1.11 Example of a vascularized fascia to improve the blood supply of nerve grafting in an unfavorable recipient bed. (**a**, **b**) Patient with right carpal tunnel syndrome and pain secondary to severe crush injury of the right distal forearm and hand. Note lack of opposition of the right thumb (**a**). Upon exploration a large neuroma in continuity of the median nerve was apparent (**b**). Extensive microneurolysis under high magnification of the operating microscope took place along with the transfer of a vascularized posterior calf fascia to envelop the nerve at the wrist. (**c**) The vascularized posterior calf fascia has been outlined in the non-dominant lower extremity. (**d**) The vascularized fascia flap after harvesting. (**e**) The vascularized fascia on the right wrist prior to microvascular anastomoses. (**f**, **g**) On the last follow-up, note excellent pinch and opposition. In addition, the patient is pain free and has returned full time to his job as a jeweler