2 Evaluation of the Patient with Postoperative Peripheral Nerve Issues

Maksim A. Shlykov, Katherine M. Velicki, and Christopher J. Dy

2.1 Intraoperative Nerve Injury Mechanisms

While it is difficult to obtain an accurate estimate of their incidence, iatrogenic peripheral nerve injuries are responsible for up to 20% of traumatic nerve lesions [1, 2]. Many of these injuries occur after orthopedic surgery due to the breadth and nature of procedures performed on the upper and lower extremities. A surgeon's best protective measures against intraoperative nerve injury are a detailed understanding of relevant anatomy (including potential variants) and understanding when these structures are at risk during each surgery (Table 2.1).

Iatrogenic nerve injuries can be broadly categorized into direct or indirect types. Direct injuries include nerve lacerations during dissection and injuries caused by insertion or removal of implants, such as the use of medial pins in unstable pediatric supracondylar humerus fractures. In revision or trauma settings, the relevant anatomy is more likely to be disrupted and may substantially increase the risk for direct nerve injury. Perioperative injection of local anesthetic, regional anesthetic, and steroids can cause neural injury if administered incorrectly or by way of neurotoxicity [3]. Indirect injuries are caused by stretch, compression, or thermal injury [3, 4]. Patient positioning and retractor placement deserve the same attention to detail as the critical portions of the case [5]. Meticulous soft tissue handling with avoidance of nerve stretch and direct nerve trauma is of utmost importance [5]. Direct visualization of nerves at risk can also minimize risk of injury. Thermal injury from electrocautery or during cementation may be irreversible but can be prevented with irrigation, adjustment of cautery settings, and protection of surrounding structures [1].

The mechanism and type of nerve injury are the major factors that shape prognosis. While 90% of "indirect" nerve injuries from stretch and compression due to improper patient positioning or aberrant retraction heal spontaneously, many lesions involving "direct" injury to a nerve require early repair or reconstruction to recover [1, 3]. Intraoperative or timely postoperative diagnosis of nerve injury is paramount to ensuring appropriate management, as delays in diagnosis can impact functional outcomes.

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Table 2.1 Common iatrogenic nerve injuries in orthopedic surgery

Procedure	Nerve at risk: mechanism
Upper Extremity Procedure	
Clavicle ORIF	Supraclavicular sensory nerves: Laceration
Submuscular biceps tenodesis	Musculocutaneous nerve: Laceration, traction
Humerus ORIF	Axillary (proximal): Laceration, traction; Radial (shaft): Laceration; Median/Radial/Ulnar (Distal): Laceration, traction
Distal biceps tendon repair	Lateral antebrachial cutaneous: Laceration; posterior interosseous nerve: laceration, traction, entrapment
Radial shaft ORIF	Posterior interosseous nerve: Traction, laceration, entrapment
First extensor compartment (De Quervain's) release	Radial sensory nerve: Laceration
Distal radius ORIF	Palmar cutaneous branch of the median nerve: Laceration; median nerve: traction, laceration
Carpal tunnel release	Median nerve, recurrent motor branch of median nerve: Laceration
Lower Extremity Procedure	
Total hip arthroplasty	Sciatic/CPN (posterior): Posterior retractor; Femoral (any approach): Anterior retractor; Lateral femoral cutaneous nerve (anterior): Laceration, anterior retractor
Hip arthroscopy	Pudendal: Post compression; Sciatic: Traction; Femoral: Traction
Total knee arthroplasty	CPN, tibial, infrapatellar branch of saphenous nerve: Retraction, laceration
Hamstring tendon harvest	Saphenous nerve: Laceration
Knee arthroscopy	Saphenous nerve: Inside-out repair
Tibia/fibula ORIF	Deep peroneal, superficial peroneal, tibial: Laceration
ACL reconstruction	Infrapatellar branch of saphenous nerve: Laceration
Calcaneus ORIF	Sural nerve: Laceration
Bunionectomy	Medial dorsal cutaneous nerve: Laceration

ORIF open reduction internal fixation, *ACL* anterior cruciate ligament, *CPN* Common peroneal nerve

2.2 Approach to History and Physical

The clinical assessment localizes the neurologic lesion and aids in predicting prognosis. The history should include details of the patient's preoperative symptoms, pain, weakness, and functional changes. Careful attention is paid to the timeline of events, recognizing that recollection of both the patient and referring surgeon may be hampered by bias. Although it is important to obtain an accurate surgical history, experts have noted that operative reports rarely divulge useful diagnostic information [1]. When possible, it is ideal to speak with the surgeon who performed the index procedure.

Serial physical examinations by the same physician are the best way to consistently assess whether the patient is improving after a nerve injury [3]. The affected extremity should always be compared to the contralateral side and the initial examination. The provider should observe muscle bulk, examine for atrophy, test passive and active range of motion, and document muscle strength on the Medical Research Council scale [6]. The scale relies on patient cooperation and grades effort from 0 to 5, with 0 being no contraction, 3 being movement against gravity, and 5 being full strength against resistance. While it is accepted as commonplace, this scale is subject to substantial inter- and intraobserver variability [7], particularly among patients with peripheral nerve injury [8]. These limitations in the grading of muscle strength emphasize the importance of systematic and serial examinations by the peripheral nerve surgeon.

The surgeon should be adept at isolating specific muscle groups in a manner that negates movement patterns that consciously or subconsciously compensate for subtle neuropathies. For example, it can be difficult to isolate the anterior and middle heads of the deltoid muscle. In patients with suspected axillary nerve palsy, the supraspinatus, long head of the biceps, coracobrachialis, and pectoralis major can provide com-

pensatory shoulder abduction. To isolate the anterior and middle deltoids, the shoulder is passively abducted and internally rotated. If the patient is unable to hold this position, there is high suspicion for an axillary nerve injury [9]. Strength against resistance is also checked in this position to assess for subtle axillary neuropathy. Another example is during assessment of extrinsic and intrinsic median nerve function. Patients with high median neuropathy or anterior interosseous neuropathy will have weakness in their flexor pollicis longus and index flexor digitorum profundus. When assessing for weakness, it is important to (1) rest the patient's forearm on their thigh or a flat surface (to minimize co-contraction of the elbow flexors and shoulder extensors to compensate for weakness in extrinsic thumb/finger flexion); (2) passively flex the wrist (to minimize the compensatory role of tenodesis); and (3) compare strength to the opposite side in the same position.

The sensory examination should assess peripheral nerve distributions, including static and moving two-point discrimination and responsiveness to light touch, pain, temperature, and vibratory stimuli [3]. Hypersensitivity, allodynia, trophic appearance of the skin, anhidrosis, and a lack of skin wrinkling during warm water immersion may provide information about disrupted sympathetic tone [3].

The surgeon should note the location and radiating features of a positive Tinel's sign, which may indicate potential axonal disruption. It is useful to use a measuring tape and reference from a reliable surface anatomy landmark to track progression of the Tinel's sign. Following nerve repair, a Tinel's sign that migrates distally over serial examinations is reassuring for axon regeneration, while failure to advance may signify neuroma formation [3]. For peripheral nerves with known areas of distal entrapment (such as the carpal tunnel for the median nerve, the cubital tunnel for the ulnar nerve, and the fibular neck for the peroneal nerve), assessment of a Tinel's sign at these locations can be helpful in determining the potential usefulness of distal decompression given the anticipated edema within the regenerating nerve [10].

It is important to rule out cervical and lumbosacral spinal causes of patient symptoms with nerve tension (i.e. straight leg raise) and upper motor neuron (i.e. Hoffman's sign) testing, as well as provocative tests such as the Spurling's maneuver. Careful attention is paid to whether the pattern of motor and/or sensory findings extends beyond a specific peripheral nerve distribution and better matches a nerve root distribution or dermatome. The presence of a peripheral nerve lesion does not exclude a spinal lesion and vice versa. In double crush syndrome, impaired axonal flow associated with a proximal nerve lesion may make more distal nerve segments more susceptible to compression that would have otherwise been tolerated [11].

2.3 Referral and Follow-Up

During the initial period of time following the presumed nerve injury, the potential for spontaneous recovery must be balanced with the chance of irreversible time-dependent end plate degeneration, after which nerve repair is futile. Following nerve injury, the motor end plate remains viable for approximately 1 year [3]. However, nerves regenerate at approximately 1 mm per day (one inch per month); thus, repair or reconstruction must be performed with enough time to allow the nerve to regenerate to target muscles before the motor end plate degenerates [12].

Timely referral for evaluation by a peripheral nerve expert is critically important to maximize the opportunity for restoration of function, whether it is from nonoperative or operative treatment [1–4]. If a partial or complete nerve transection is identified during surgery, a surgeon with capability of performing microsurgical assessment and possible repair should be consulted. If intraoperative consultation is not available, we prefer that the surgeon place an easily visible suture (such as dyed 6-0 polypropylene) at each end of the nerve to minimize retraction of the nerve ends. The location of the nerve injury relative to surrounding anatomic landmarks (such as osseous prominences or screw holes or markings of an associated implant) should be commu-

nicated. For direct injuries from a sharp/tidy mechanism, early repair should be performed by a surgeon with microsurgical capability [1, 3, 13]. For direct injuries with known partial or complete nerve discontinuity from a non-tidy mechanism (such as drills and reamers), nerve repair or reconstruction is performed after waiting an additional 2–3 weeks for the zone of injury to declare itself within the nerve. For suspected nerve injuries (when direct injury has not been visualized during the index surgery), nerve repair or reconstruction should be performed within 6 months after injury (and ideally within 3–4 months of injury) to maximize return of motor function [1–3, 14]. While the exact threshold upon which muscle fibrosis and atrophy are irreversible in humans is not clearly defined, a systematic review of the literature demonstrated improved outcomes with earlier intervention after known peripheral nerve injury [15]. Animal studies have demonstrated poorer motor reinnervation after prolonged denervation due to degeneration of the terminal ends of the distal nerve stump [16] and failure of the denervated muscle to recover from denervation atrophy [17]. While it is commonly believed that sensory nerve ends maintain the ability to regenerate for an indeterminate period of time [3], the degeneration of the distal nerve stump after prolonged denervation may compromise outcomes if reinnervation eventually occurs. Regardless of the decision whether to operate and the timing of operation, early referral to a peripheral nerve specialist allows the patient and surgeon to establish a trusting relationship and facilitates serial examination.

Unfortunately, delayed referrals to a peripheral nerve specialist are common following iatrogenic nerve injuries. Fewer than 40% of patients in two large retrospective studies underwent surgery within 6 months of their iatrogenic nerve injuries [2, 4]. There are many potential reasons for these delays in specialized care. First, peripheral nerve injuries may be difficult to diagnose due to either a lack of knowledge or failure to recognize the lesion [2]. Second, a prolonged observation period to see whether the clinical symptoms and EMG findings improve with conservative measures may delay appropriate referral [1, 18]. Out of hubris or hope, patients may be subjected to "therapeutic nihilism" and left unacceptably undertreated due to underlying skepticism that additional interventions would be helpful [19]. Third, patients with function-limiting nerve injuries are particularly vulnerable as they may be unable to return to work and continue to fund their treatment [1]. Lastly, shame, guilt, anxiety, possible professional repercussions, and fear of litigation that are experienced by the surgeon may discourage them from acknowledging errors and making timely referrals [3, 20–22].

Prompt referral to physical (PT) and/or occupational therapy (OT) after identification of a nerve injury can also improve prognosis until reinnervation of affected muscles is achieved [1]. Initial focus is placed on maintaining passive joint motion and incorporating strategies to alleviate neuropathic pain and maximize adjustment to altered or absent function. Once functional improvement begins either spontaneously or after surgical reconstruction, motor and sensory reeducation strategies are emphasized. In addition to the peripheral nerve specialist and PT/OT, pain management specialists experienced in the pharmacologic, procedural, and psychological treatments of neuropathic pain are critically important members of the treatment team. Social workers and vocational rehabilitation specialists are also incorporated into the treatment team, as the most severely affected patients may have difficulty with resuming their pre-injury employment.

2.4 Electrodiagnostic Studies

Electrodiagnostic studies (EDX) are useful to localize a peripheral nerve injury and predict prognosis. EDX should be considered an extension of the clinical assessment. EDX are composed of nerve conduction studies (NCS) and electromyography (EMG). NCS reflect function of the components of the nerve, specifically the axons and surrounding myelin. EMG indicates

the integrity of the arc between the peripheral nerve and its associated muscle. Following axonal injury, Wallerian degeneration occurs to prepare the proximal and distal stumps for regeneration. EDX obtained earlier than 10 days after injury may be falsely "normal," as the effects of Wallerian degeneration will have not manifested on a macroscopic level. One potential use of early EDX is to evaluate for baseline nerve pathology, such as underlying radiculopathy or small fiber neuropathy. In most circumstances, the initial EDX assessment is obtained between 3 and 6 weeks after nerve injury. At this time, fibrillations and positive sharp waves are detectable during the resting phase of the EMG. These changes reflect the instability of the muscle membranes following nerve injury. The presence of a motor unit action potential (MUAP) during the activation phase of the EMG is a helpful indicator as to whether spontaneous muscle recovery will occur. In incomplete (axonotmetic) nerve injuries, the intact axons may collaterally sprout in order to reinnervate portions of the muscle "vacated" by the injured axons. These collateral sprouting MUAPs will have a distinct pattern from those MUAPs associated with regeneration of axons across the injured nerve segment. An experienced electromyographer may be able to discern the differences between these two MUAP patterns, but the signals are technically difficult to detect and the ultimate clinical implications are unclear. If MUAP are not detectable by 3 months, we typically recommend consideration of surgical treatment given the high unlikelihood of spontaneous recovery [18, 23–25]. If any MUAP activity is detected at 3 months, we will usually recommend continued observation with an additional clinical assessment with or without a follow-up EMG in 6 weeks. Serial EDX are best performed by the same electrodiagnostician to minimize technical variability, to maximize patient comfort, and to coordinate treatment plans with the peripheral nerve surgeon (such as including interrogation of potential donor neuromuscular units for nerve transfer). The motor portion of NCS will typically corroborate those findings seen on EMG, with losses in compound motor action potential (CMAP) amplitudes reflecting the amount of axonal injury. For sensory and mixed nerves, the latency and nerve conduction velocity measures can provide an assessment of function and can aid in lesion localization. In purely demyelinating injuries (neurapraxia), nerve conduction velocities are typically normal if measured distal to the lesion but will be decreased if measured across the lesion. Concomitant slowing is usually seen until recovery. CMAP amplitudes will be normal given that neurapraxic injuries are not associated with axonal loss. Partial nerve (axonetmetic) injuries and complete nerve (neurotmetic) injuries will have partial and complete loss of CMAP amplitudes, respectively. Comparison of CMAPs to the contralateral uninjured side can estimate degree of axonal loss, although the potential exists for changes associated with underlying (and possibly subclinical) compressive neuropathy [26].

2.5 Imaging

Evaluation of preoperative imaging can provide indications of cases with a high likelihood of postoperative peripheral nerve issues. For example, correction of a valgus knee deformity with total knee arthroplasty and fixation of a distal third humeral shaft fracture both have a higher chance of postoperative nerve palsy. Inspection of intraoperative fluoroscopy or postoperative radiographs can also suggest the likelihood of nerve palsies, such as lateralization of the glenohumeral joint after shoulder arthroplasty or leg lengthening after total hip arthroplasty. The reduction quality and location of osteosynthesis constructs and the surgical exposures necessary to position the constructs are useful in determining the risk and nature of nerve injury. Advanced imaging can be helpful in certain situations, such as using a CT scan or MRI to evaluate for the presence of a hematoma or other fluid collection. Ultrasound has the added benefit of being able to visualize nerves longitudinally, allowing for easier identification of nerve discontinuity. Ultrasound may also demonstrate neuromas and

can be used to measure nerve cross-sectional area to assess for swelling [27–29].

2.6 Approach to Treatment

If a postoperative peripheral nerve issue is suspected, reversible causes of nerve injury, such as a tight-fitting cast or dressing, should be addressed [1]. If other potentially reversible causes, such as a hematoma or entrapment by implants, are suspected, early intervention is recommended. In most cases, the postoperative nerve injury is noted within the first 4–6 weeks after surgery. Motor and sensory loss are typically noticed after the initial pain from surgery subsides and the patient begins rehabilitation. Once suspicion arises for peripheral nerve injury, prompt referral to a clinician with experience caring for peripheral nerve injuries for the reasons stated above. While the duration of time to observe and await spontaneous recovery will vary based on the presumed nature and location of the injury, the absence of detectable MUAP on EMG at 3 months portends a relatively poor prognosis (Fig. 2.1). If operative intervention is considered, patient expectations should be set early, with repeated discussions between the patient and surgeon (as well as the patient and hand therapist) about the lengthy time duration for nerve recovery and the likely inability to restore "normal" or "perfect" function.

2.7 Early Nerve Repair

While the scenarios in which it arises are relatively uncommon, optimal results are obtained from immediate or early repair of a sharply transected nerve (Fig. 2.2). The primary goal of early nerve repair is to provide a supportive structure that guides sensory, motor, and autonomic axons distally toward their target organs. The proximal and distal ends of the nerve may need to be mobilized in order to facilitate a tension-free coaptation. This is technically much easier to accomplish, while the nerve ends are still "stuck" or scarred down. Following mobilization of the nerve ends, direct end-to-end epineural repair technique is typically used. In some cases where the nerve topography has been reliably established (such as the ulnar nerve in the distal forearm), a grouped fascicular repair technique is used. The nerve coaptation is performed using microsurgical technique. This attachment must be tension free, as excess stretch at the repair site can damage fragile endoneurial capillaries, devascularize the nerve, and lead to fibrosis at the repair site (Fig. 2.3) [23]. If a tension-free coaptation cannot be performed, nerve grafting is used (see below).

2.8 Staged Exploration and Neurolysis

The vast majority of cases undergoing surgical treatment weeks to months after the initial procedure are likely to be neuromas-in-continuity (i.e., axonotmetic injuries attempting to recover). Because of the technological limitations in preoperative assessment of the injured nerve, currently there is no substitute to the peripheral nerve surgeon assessing the neuroma-in-continuity intraoperatively via surgical exploration. Relatively crude measures are still used, with the nerve surgeon relying on visual inspection and palpation of the neuroma-in-continuity. External neurolysis is used to dissect away the scarred mesoneurial and external epineural tissue. Depending on the look and feel of the nerve, handheld nerve stimulators and/or nerve-to-nerve action potentials may be useful to assess function of the nerve. Both of these modalities are subject to technical difficulty and reliance on them may preclude the prolonged use of a limb tourniquet. Preoperative EDX can provide information about conduction loss across the site of injury. If MUAPs on EMG and CMAP amplitudes are present, neurolysis with scar excision can be highly successful (Fig. 2.4) [30]. If a more severe intraneural injury is suspected, internal neurolysis of the neuroma is performed using microsur-

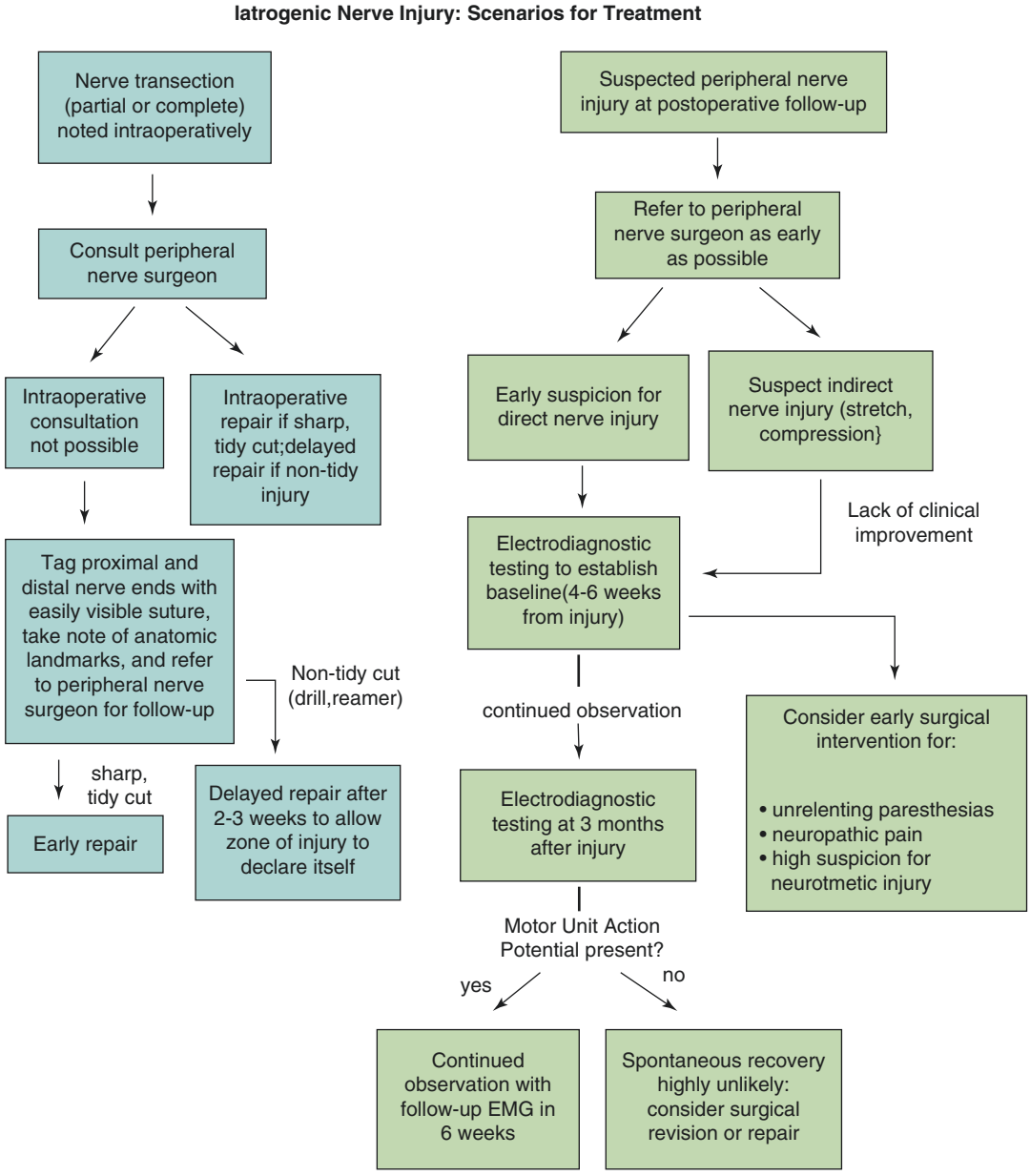

Fig. 2.1 Treatment algorithm to guide management of iatrogenic nerve injuries

gical technique to incise the perineurium and inspect individual fascicles. The individual fascicles are inspected, palpated, and can be interrogated with nerve-to-nerve action potentials. At this time, the surgeon makes a decision whether to excise scarred and unhealthy portions of nerve and how much to excise. If only portions of the nerve are thought to be diseased, then only these segments are removed and the healthy fascicles left intact. If the vast majority or entirety of the nerve is thought to be diseased, the neuroma is resected en bloc. In both situations, it is critically important to trim back to healthy, extruding fascicles. Performing neurorrhaphy within a diseased segment of the nerve is likely to lead to failure of nerve regeneration [31].

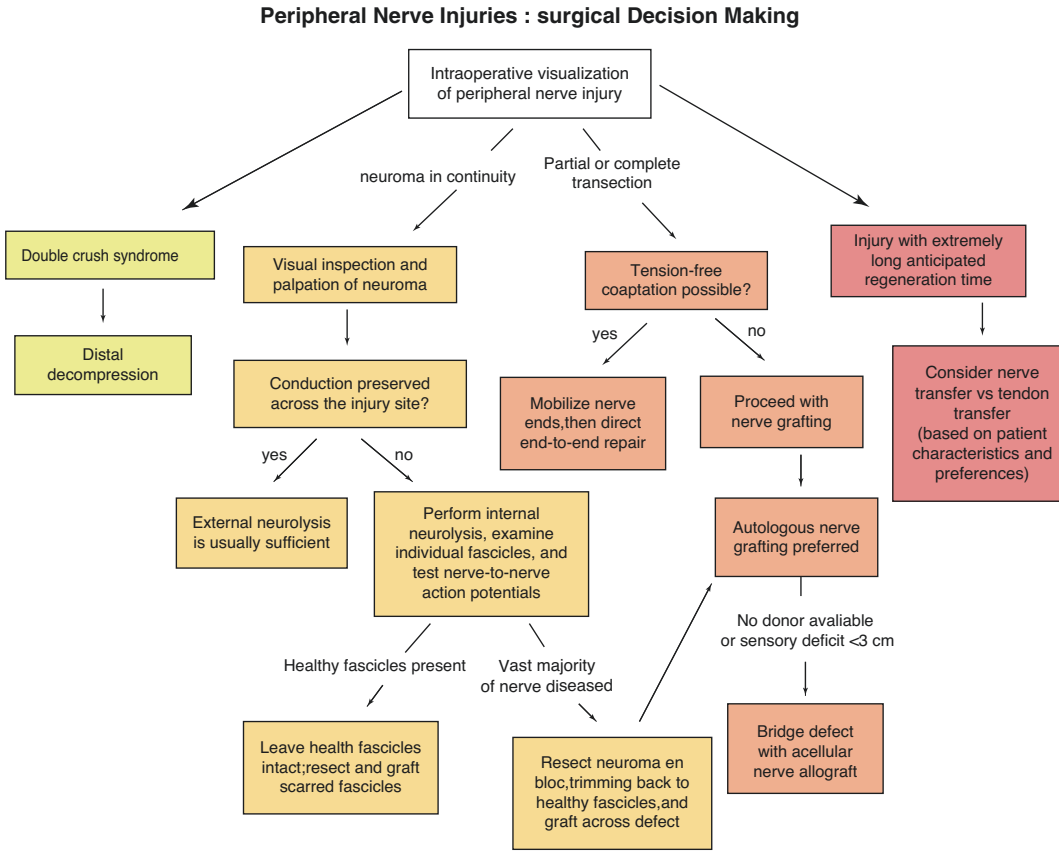

Fig. 2.2 Algorithm to guide surgical decision making for peripheral nerve injuries

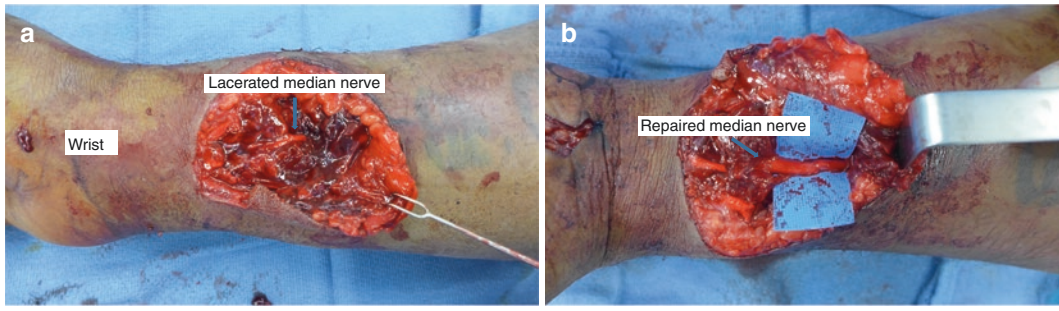

Fig. 2.3 Patient with a sharp median nerve laceration after being stabbed. (**a**) Distal aspect of median nerve is visualized. (**a**–**b**) Proximal aspect of median nerve is mobilized and tension-free end-to-end epineural coaptation is performed using microsurgical technique. (Photographs copyright Christopher J. Dy, MD MPH – used with permission)

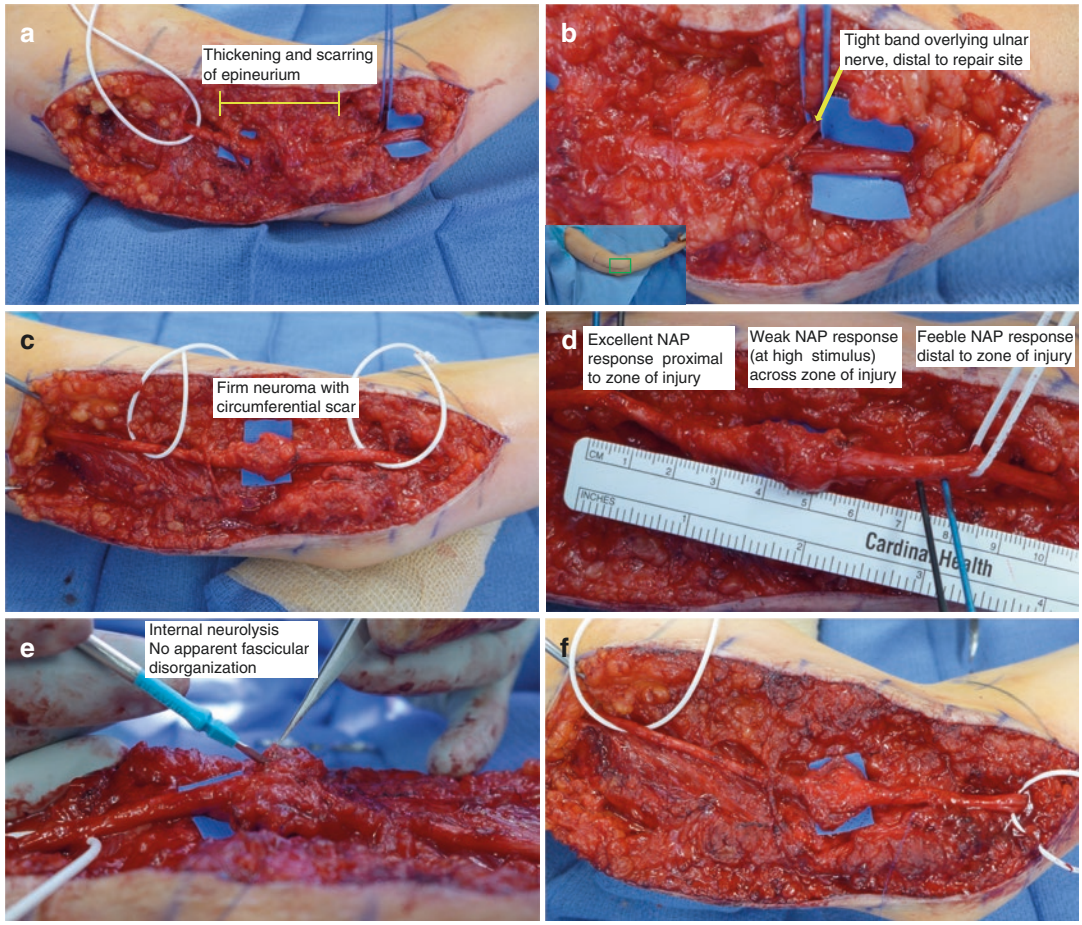

Fig. 2.4 Patient's 20 months status post primary repair of high ulnar nerve injury (sharp/tidy mechanism) at the elbow and primary supercharge end-to-side anterior interosseous to ulnar motor nerve transfer who presented with persistent pain and intrinsic weakness. (**a**) Thickening and scarring of ulnar nerve epineurium. (**b**) Tight band overlying ulnar nerve distal to repair site released. (**c**) Firm neuroma with circumferential scar encountered after performing external neurolysis. (**d**) Testing of nerve action potentials (NAP) demonstrated excellent NAP response proximal to zone of injury and weak responses across and distal to the zone of injury. (**e**) Internal neurolysis performed as no apparent fascicular disorganization was observed. (**f**) Ulnar nerve after external and internal neurolysis. (Photographs copyright Christopher J. Dy, MD MPH – used with permission)

2.9 Nerve Grafting

Autologous nerve grafting involves harvesting an expendable donor nerve segment from a patient to bridge a gap between proximal and distal ends of a nerve lesion [32]. The sural nerve is an easily accessible donor that can provide 30–35 cm of graft per leg with minimal donor site morbidity (Fig. 2.5) [3, 33]. Other potential donor sites include superficial peroneal, saphenous, and medial antebrachial cutaneous nerves [3]. The graft should extend approximately 15% longer than the lesion to minimize any potential tension from movement of the surrounding tissues and to account for contracture of the graft itself [33].

Prior to placing the graft, healthy fascicular architecture at the proximal and distal recipient nerve ends is confirmed. Single nerve grafts are used to span lesions when the donor and recipient nerves are similar in diameter. Cable grafts, or bundles of multiple small diameter nerves, are preferred for large-diameter nerves; this tech-

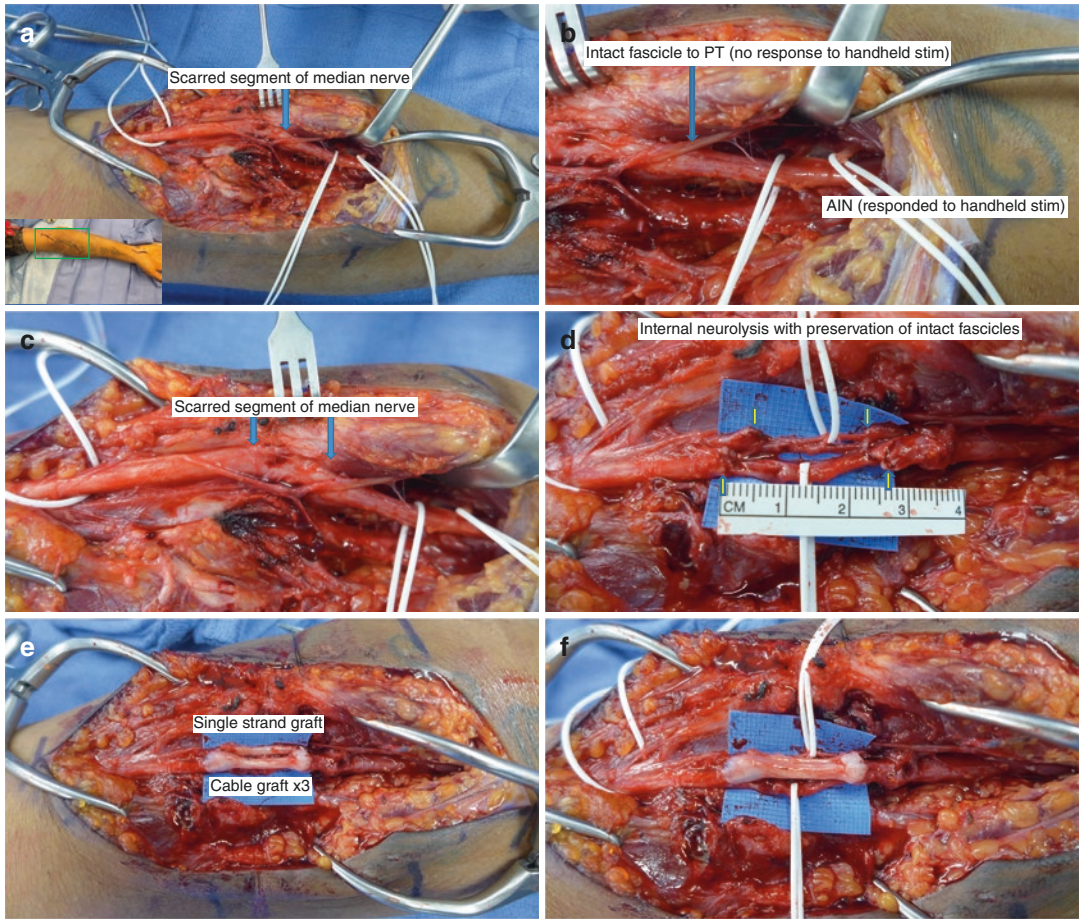

Fig. 2.5 Patient's status post gunshot wound to antecubital fossa with reported 85% median nerve transection on exploration and bascilic vein ligation by vascular surgery. (**a**) Zone of injury with significant scarring of the median nerve identified by working from known to unknown. (**b**) Intact fascicle to the pronator teres (PT) without response to handheld stimulation and anterior interosseous nerve (AIN) with response to handheld stimulation. (**c**) Close-up view of scarred portion of median nerve. (**d**) Internal neurolysis performed with preservation of intact fascicles with 3 cm gap in median nerve. (**e**) A 3 cm cabled sural nerve autograft using three fascicles and a 3 cm single fascicle graft were coapted. (**f**) Median nerve status post sural nerve autograft with vessel loops demonstrating preserved fascicles. (Photographs copyright Christopher J. Dy, MD MPH – used with permission)

nique maximizes the number of axons and enhances the viability of nonvascularized grafts [3, 23]. Although autologous nerve grafts undergo Wallerian degeneration after harvesting and interposition, the remaining Schwann cell basal laminae, neurotrophic factors, and adhesion molecules serve as a stimulating scaffold for distal axon migration [23].

One emerging alternative to autologous nerve grafting is the use of acellular nerve allografts. Acellular nerve allografts have been processed to minimize immunogenicity, but this process also removes the Schwann cells from the nerves. The endoneurial tubes, basal lamina, and laminin that remain in acellular nerve allografts provide an organized conduit for axon growth [23]. Allografts have the theoretical advantage of abundant supply, although they are not widely available outside of the United States. Advocates for nerve allograft contend that the additional cost of the tissue implant is minimal compared to the additional operative time and potential donor

site morbidity associated with autograft harvest. While the current evidence has established it as a reliable option for treatment of small (<3 cm) sensory deficits, the role of acellular nerve allografts for mixed and motor nerves is in evolution.

2.10 Nerve Transfer

Nerve transfers have become a useful strategy in cases when the anticipated time to regeneration is extraordinarily long, either due to the distance between the nerve injury and the target muscle or due to a lengthy gap between nerve ends. In a nerve transfer, a healthy donor nerve is cut and sutured to the injured nerve's distal end (Fig. 2.6) [23]. Benefits of nerve transfers include only one neurorrhaphy site, short distance for nerve regen-

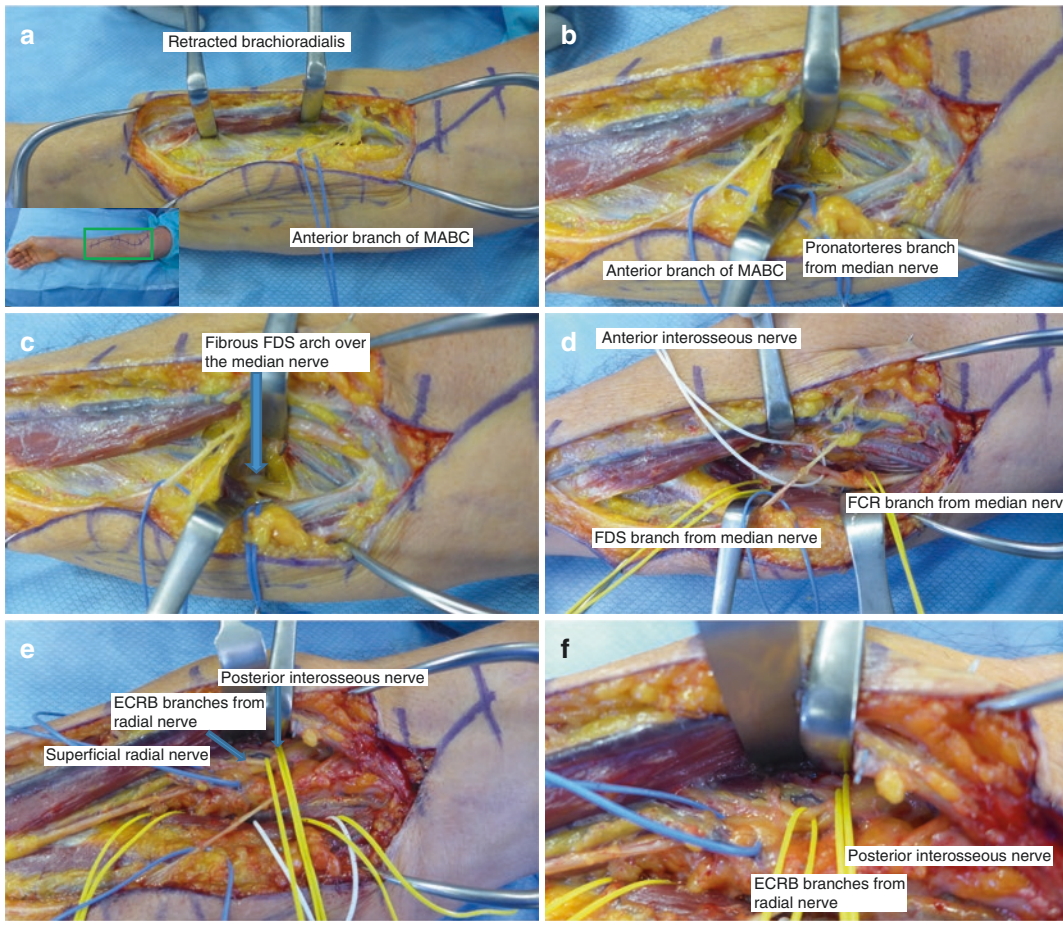

Fig. 2.6 Patient's 5 months status post radial shaft fracture open reduction internal fixation without resolution of preoperative radial nerve palsy. (**a**) The anterior branch of the medial antebrachial cutaneous nerve (MABC) is protected and brachioradialis is retracted. (**b**) Pronator teres branch from the median nerve is identified. (**c**) Flexor digitorum superficialis (FDS) aponeurotic arch is released. (**d**) Anterior interosseous nerve as well as the flexor carpi radialis (FCR) and FDS branches from the median nerve are identified. (**e**) Posterior interosseous nerve (PIN), superficial radial nerve, and extensor carpi radialis brevis (ECRB) branches from the radial nerve are identified. (**f**) Close-up view of PIN and ECRB branches. (**g**) Pronator teres (PT) and ECRB tendon transfer performed to help restore wrist extension. (**h**) Final coaptation of FDS to ECRB and FCR to PIN nerve transfers. (Photographs copyright Christopher J. Dy, MD MPH – used with permission)

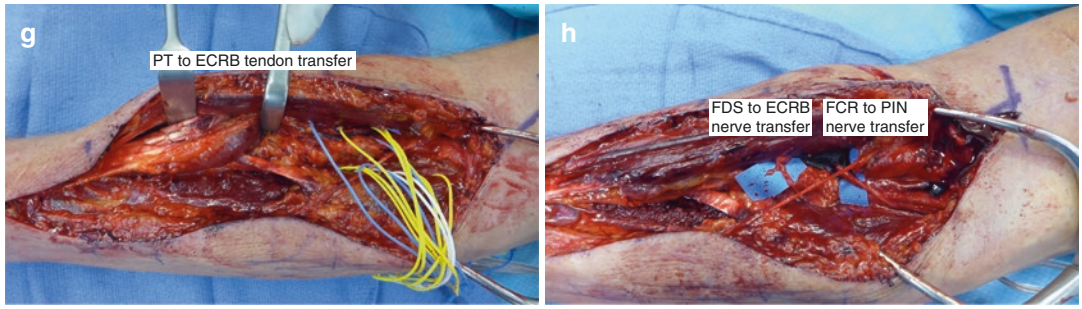

Fig. 2.6 (continued)

eration, rapid reinnervation and motor reeducation, and unaltered muscle biomechanics [23, 33]. Limitations of nerve transfers include their requirement for an expendable donor motor nerve in close proximity to the target and loss of the donor nerve's original function [23, 32, 33]. The latter may have implications in compromising options for subsequent tendon transfers.

2.11 Distal Decompression

Distal nerve decompression is based on the concept of double crush syndrome [11]. After surgery, swelling in the affected extremity increases due to the insult of surgery, but also immobility. Disruption of axoplasmic flow as a result of a proximal nerve injury may also create nerve swelling. Nerves particularly at risk in this setting include the median nerve at the carpal tunnel, ulnar nerve at the cubital tunnel, and the common peroneal nerve at the fibular neck (Fig. 2.7). Monitoring of Tinel's sign as well as ultrasound examination may be useful in this setting to evaluate nerve cross-sectional area. In a retrospective review of 142 patients undergoing nerve repair, Schoeller et al. demonstrated functional recovery in two cases where distal decompression was performed after clinical evaluation and EMG recordings were suggestive of nerve compression distal to the nerve repair [10]. A retrospective study evaluating common peroneal nerve decompression after proximal sciatic nerve injury sustained during total hip arthroplasty demonstrated recovery of dorsiflexion strength of ≥3 in 65% of patients compared to 50% of patients who were treated nonoperatively in other studies [34].

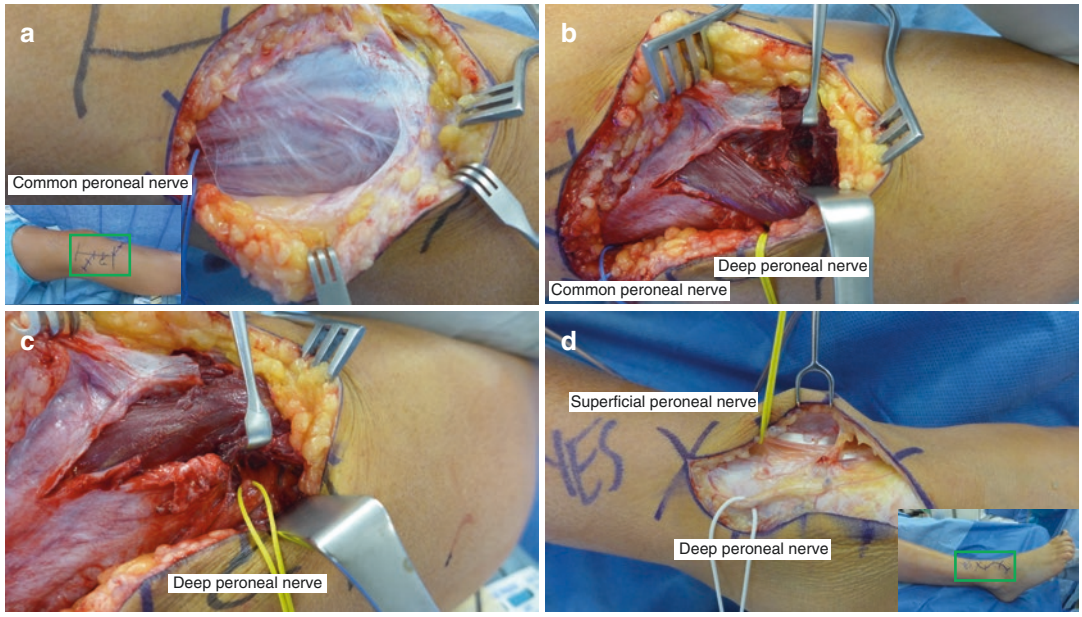

Fig. 2.7 Patient's 1 year and 3 months status post basketball injury and 8 months status post short head of biceps release, peroneal nerve release, and diagnostic knee arthroscopy with persistent foot drop. (**a**) An S-shaped incision (vs original longitudinal incision) was used demonstrating scarring overlying the common peroneal nerve. (**b**) The anterior crural, posterior crural, and deep intermuscular septa are released. (**c**) The deep peroneal nerve is sufficiently released. (**d**) Distal decompression of the deep peroneal nerve was performed due to concern for double crush syndrome. The patient had return of extensor hallucis longus function in the post-anesthesia care unit immediately after surgery. (Photographs copyright Christopher J. Dy, MD MPH – used with permission)

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