Review Article

Management of Iatrogenic Nerve Injuries

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Abstract

Iatrogenic peripheral nerve injuries from orthopaedic surgery can occur via many scenarios, including direct injury to the nerve during surgery, indirect injury via retraction or compartment syndrome, and injury from nonsurgical treatments such as injections and splinting. Successful management of iatrogenic nerve injuries requires an accurate diagnosis and timely, appropriate treatment. All orthopaedic surgeons must understand the preclinical study of nerve injury and the evaluation and treatment options for iatrogenic nerve injuries. Although a sharply transected nerve can be repaired immediately in the operating room under direct visualization, many injuries are not appreciated until the postoperative period. Advances in diagnostic studies and nerve repair techniques, nerve grafting, and nerve transfers have improved our ability to identify and treat such injuries.

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Orthopaedic surgery is the most common cause of iatrogenic peripheral nerve injury requiring treatment.[1] A retrospective study of 722 traumatic nerve lesions across multiple disciplines found that 17.4% were iatrogenic in nature.[1] In the upper extremity, the median nerve is most commonly injured, followed by the spinal accessory, superficial radial, common peroneal, and ulnar nerves.[2] Common iatrogenic nerve injury scenarios are listed in Table 1.

Mechanisms of Injury

A peripheral nerve is composed of single-cell axons surrounded by myelin sheaths grouped into fascicles with the interstitial endoneurium. Fascicles are surrounded by a thin perineurial layer, providing tensile strength. The epineurium is the connective tissue layer, which encircles and runs between the fascicles[3] (Figure 1). The internal topography of peripheral nerves is relatively consistent, allowing for fascicular repair and nerve transfer surgeries.

Table 2 lists common mechanisms of iatrogenic nerve injury. Intraoperatively, direct trauma to peripheral nerves may involve complete or partial transection during dissection. Surgeons must appreciate anatomic variations in the course of peripheral nerves to minimize the risk of iatrogenic nerve injuries during dissection.[2] Implants add an additional risk factor because nerves may be impinged by plates, penetrated or twisted with drill bits, screws, or K-wires, particularly when percutaneous or "minimally invasive" techniques are used.[4] Nerves can be indirectly injured and stretched during retraction or with the insertion and removal of orthopaedic implants. Thermal injury (secondary to exothermic reaction of cement or cautery devices) can result in irreversible damage to nearby neural structures. In some cases, determining the exact etiology of the nerve deficit may be confounded by the mechanism of the injury, such as in a supracondylar

Table 1

Common Iatrogenic Nerve Injuries

Procedure	Nerve Affected
Upper extremity procedures	
Cervical lymph node dissection	Spinal accessory nerve
Sternotomy	Ulnar nerve Medial cord
Clavicle fracture ORIF	Supraclavicular nerve
Submuscular biceps tenodesis	Musculocutaneous nerve
Proximal humerus fracture ORIF	Axillary nerve
Humeral shaft fracture ORIF	Radial nerve
Supracondylar humerus fracture CRPP	Median, radial, or ulnar nerve
Distal humerus fracture ORIF	Ulnar nerve
Distal biceps tendon repair	Lateral antebrachial cutaneous nerve
Radial shaft fracture ORIF	Posterior interosseous nerve
1st extensor compartment release	Radial sensory nerve
Distal radius fracture ORIF	Palmar cutaneous branch of the median nerve
Carpal tunnel release	Median nerve Recurrent motor branch of the median nerve
Lower extremity procedures	
Total hip arthroplasty	Sciatic nerve (posterior approach) Femoral nerve (anterior approach)
Total knee arthroplasty	Peroneal nerve Infrapatellar branch of the saphenous nerve
Hamstring tendon harvest	Saphenous nerve
Tibial shaft fracture ORIF with LISS	Deep peroneal nerve
Distal tibia fracture ORIF	Tibial nerve
Distal fibula fracture ORIF	Superficial peroneal nerve
Calcaneus fracture ORIF	Sural nerve
Plantaris tendon harvest	Sural nerve
Bunionectomy	Medial dorsal cutaneous nerve

CRPP = closed reduction percutaneous pinning, LISS = less invasive stabilization system, ORIF = open reduction internal fixation

humerus fracture. Other orthopaedic interventions, such as injections, splinting, and bracing treatments, may also cause a nerve deficit to develop. Lastly, a peripheral nerve may be resected when it is mistaken for a lymph node, vessel, or tendon.[5] Therefore, all patients with postoperative neurologic deficits should be approached with a high index of suspicion to prevent diagnosis and treatment delays.

Perioperative nerve injury is one of the most common etiologies for anesthesia-associated medicolegal claims.[6] Ulnar and sciatic nerves are most commonly affected, but other terminal branches of the brachial or lumbosacral plexus may be involved. External nerve compression or traction due to patient malpositioning is often considered the mechanism of injury. In contradistinction, Warner reported that patients who sustained perioperative ulnar nerve palsy were more likely to have had contralateral ulnar nerve dysfunction, suggesting that patient factors may be important. Nevertheless, prolonged duration in one position increases the risk of neuropathy and is a common factor in both upper and lower extremity nerve palsies after surgery.[7]

Common Upper Extremity Lesions

Brachial Plexus Nerve Injuries

Rates of iatrogenic nerve injury after shoulder surgery range from 0.2% to 8.0%.[8] The entire brachial plexus and several peripheral nerves have the potential to be injured. Structures at risk include the musculocutaneous, median, and ulnar nerves during subpectoral biceps tenodesis, the axillary nerve during arthroscopic Bankart repair, and the entire

Figure 1

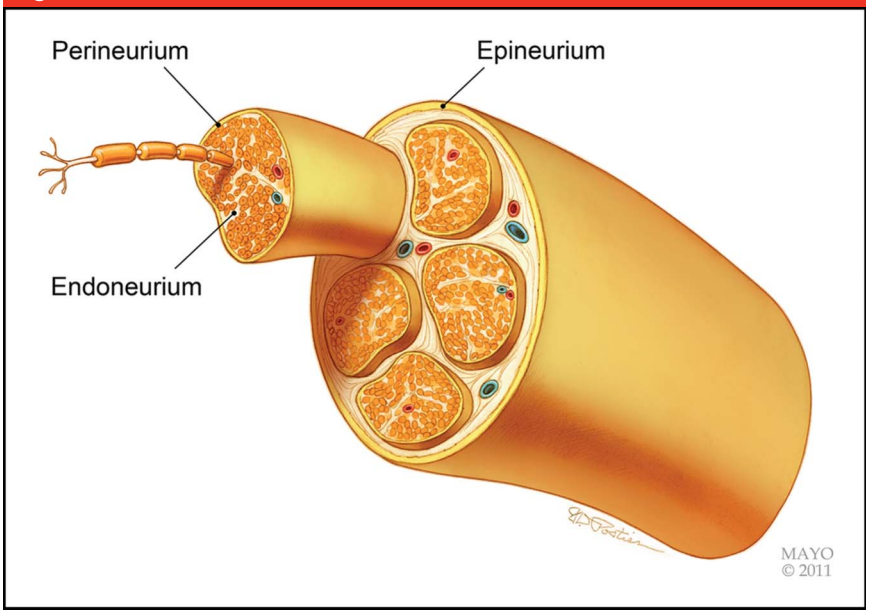


Diagram showing the anatomy of a peripheral nerve. (Reproduced with permission from the Mayo Foundation for Medical Education and Research, Rochester, MN. All rights reserved.)

brachial plexus during open surgery for instability.[8] Although it is possible that injury may be the result of regional anesthesia, a prospective registry found that the incidence of postoperative neurologic symptoms lasting more than 6 months was only 0.9 per 1,000 blocks.[9]

The spinal accessory nerve (CN XI) innervates the sternocleidomastoid and trapezius muscles and is most commonly injured in cervical lymph node dissection or radical neck dissection. Patients present with shoulder weakness, pain, and dysfunction. Although injury to the spinal accessory nerve may not be the result of an orthopaedic procedure, orthopaedic surgeons are likely to be the first to correctly identify the diagnosis and must be aware of this common injury and its sequela.[10]

Radial Nerve Injuries

Radial nerve dysfunction is a well-documented sequela of surgically treated diaphyseal humerus fractures, and iatrogenic injury is reported in approximately 7% of fracture fixation cases. Claessen et al[11] found that the surgical approach was the most important factor relating to iatrogenic nerve injury with the highest incidence in the lateral exposure. Notably, there was 18.5% incidence of iatrogenic radial nerve palsy after nonunion repair, where débridement of fracture callus may add an additional

Table 2

Common Causes of Iatrogenic Nerve Injuries

Mechanism of Injury	Example(s)	
Orthopaedic surgical procedure	Procedure	Nerve injured
Direct		
Nerve cut	ACL reconstruction	Infrapatellar branch of the saphenous nerve
Nerve injured by implant	Supracondylar humerus CRPP	Ulnar, median, or radial nerve
Indirect		
Nerve stretched or contused by dissection	Anterior hip surgery	Lateral femoral cutaneous nerve
Nerve stretched or contused during patient positioning	Prone positioning	Ulnar nerve
Compartment syndrome	Tibial shaft fracture	Peroneal nerve
Orthopaedic surgical procedure with unknown nerve status preoperatively	Gunshot wound to the humeral shaft	Radial nerve
Nonsurgical orthopaedic intervention		
Injection	De Quervain release	Radial sensory nerve
Splint application	Ankle fractures	Common peroneal nerve
Regional anesthesia associated	Total shoulder arthroplasty	Axillary nerve
Surgery by another specialty	Cervical lymph node biopsy	Spinal accessory nerve (CN XI)

risk factor.[12] In a retrospective study of 707 humeral shaft fractures treated surgically, Wang et al[13] concluded that there was no advantage to early exploration after iatrogenic nerve injury in the absence of obviously misplaced instrumentation or fracture displacement.

Median Nerve Injuries

Corticosteroid injections are common in the nonsurgical management of carpal tunnel syndrome. Injury to the median nerve is uncommon.[14-16] However, intraneural injection of a local anesthetic or corticosteroid can lead to permanent impairment because nearly all injectable anesthetic agents are neurotoxic.[15]

In a review of major nerve injuries associated with carpal tunnel release, Azari et al[16] reported that the most significant factor resulting in a referral was the operative surgeon's lack of specialized training in hand surgery. The true incidence of nerve injury associated with carpal tunnel could not be ascertained.

Sensory Nerve Injuries

Damage to sensory nerves can lead to dysesthesias and the formation of painful neuromas. Injuries to sensory nerve branches are frequently described but are likely under-reported. In a large series, only 25% of documented radial nerve injuries involved the superficial sensory branch.[17] The radial sensory nerve can be damaged with De Quervain release and other radial-sided hand and wrist procedures. The lateral antebrachial cutaneous nerve is similarly at risk with radial-sided wrist procedures distally and distal biceps repair proximally. The palmar cutaneous branch of the median nerve can be injured during exposure to the volar distal radius.

Percutaneous fixation of hand fractures has many advantages but can put several neurovascular structures at risk.[4] Knowledge of anatomy, small incisions with blunt dissection to bone, and soft-tissue protectors including large-gauge needles may be used to limit the risk of iatrogenic injury.

Table 3

Seddon[3] Classification of Peripheral Nerve Injuries

Nerve Injury	Pathophysiology	Management	Prognosis
Neurapraxia	Local myelin damage, intact axons	Serial examinations.	Usually recovers spontaneously and completely in a short period
Axonotmesis	Axonal loss, epineurium intact	Serial examinations. Therapy to maintain joint range of motion. May require surgery.	May recover spontaneously but over a longer time.
Neurotmesis	Complete division of the nerve	Early nerve repair or reconstruction.	No recovery without treatment leading to persistent severe deficits

Common Lower Extremity Lesions

Sciatic Nerve Injuries

The incidence of sciatic nerve injury after total hip arthroplasty ranges from 0.6% to 3%. Patients undergoing surgery for hip dysplasia or revision are most at risk.[18] Clinical assessment alone may underestimate the true incidence of these lesions.[19] After total hip arthroplasty, iatrogenic injury may be a result of patient positioning, retractor placement, penetrating implant, traction associated with leg lengthening, thermal injury (ie, electrocautery or methyl methacrylate cement), or compression secondary to hematoma. Because of its anatomic location, the common peroneal division is more commonly injured than the tibial division.[20] In treating more than 350 sciatic nerve lesions surgically, Kim et al[21] recommended surgical exploration in patients without clinical or electrodiagnostic evidence of improvement by 4 to 5 months after injury. Rarely was the nerve transected on surgical exploration. Injection injuries to the sciatic nerve have also been reported and are more commonly seen in children and elderly patients with less soft-tissue coverage.

Common Peroneal Nerve Injuries

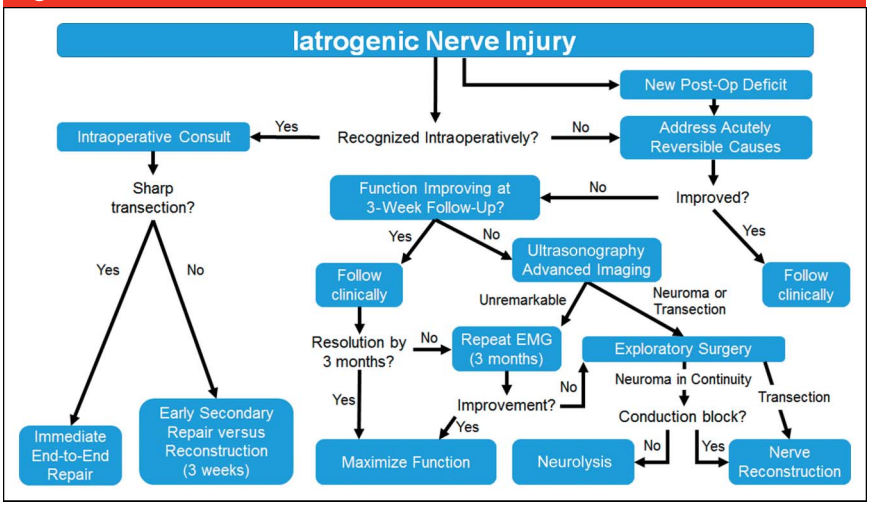
The superficial position of the common peroneal nerve, lateral to the fibular head, makes it susceptible to compression injury during casting and intraoperatively due to positioning. The nerve may be injured surgically during exposure and retraction in open cases and arthroscopically during lateral meniscal repair.

"Minimally invasive" techniques put the common peroneal nerve at risk, and injury has been reported with stab incisions for varicose vein procedures[22] and percutaneous fixation of distal tibia fractures. The deep peroneal nerve is most at risk with the less invasive stabilization system (LISS plating system).[23]

Sensory Nerve Injuries

In the lower extremity, damage to sensory nerve branches can range from numbness and paresthesias to the development of a painful neuroma and neuropathic pain on a weight-bearing surface. The infrapatellar

Figure 2



Flowchart showing management of iatrogenic nerve injuries.

branch of the saphenous nerve is frequently damaged with any parapatellar incision over the knee including patellar tendon harvest for ACL reconstruction, total knee arthroplasty, proximal tibia open reduction internal fixation (ORIF), and placement of an intramedullary nail for tibial shaft fracture. In addition, the saphenous nerve proper may be injured during hamstring tendon graft harvesting. In a meta-analysis, Pekala et al[24] found a rate of injury of 51.4% with vertical incisions and advocated for the use of the shortest possible oblique incision during harvesting over the pes anserinus.

Management of Iatrogenic Nerve Injuries

The Seddon[25] classification of peripheral nerve injuries helps to define the affected structures and to guide treatment (Table 3). The challenge in determining which treatment strategy to use lies in determining which type of nerve injury is present because all nerve lesions may present similarly in the immediate postoperative period.

In the setting of a known iatrogenic nerve transection with the patient still under anesthesia for the index procedure, the injury should be assessed and immediately treated. For small sensory nerve branches, the nerve ends may be left untreated or buried to minimize the risk of developing a symptomatic neuroma. For motor nerves and large sensory nerves, the decision to directly repair or graft acutely (if direct repair is not possible) is relatively uncontroversial.[3] Ideally, this should be done immediately by a surgeon familiar with the management of peripheral nerve injuries, one who is comfortable obtaining autologous nerve grafts and experienced with the use of an operating microscope to facilitate fascicular repair. An intraoperative consult by such a surgeon is warranted in this scenario if possible. When intraoperative consultation is not available, the nerve ends may be tagged and the wound closed with immediate postoperative consultation with a nerve surgeon for early re-exploration and repair.

When a patient is identified in the acute postoperative setting with a nerve deficit, urgent evaluation is necessary. Pain with passive stretch is concerning for compartment syndrome and may necessitate immediate fasciotomy. Constrictive dressings may put pressure on the peroneal nerve as it crosses the fibular neck, and knee braces may stretch the nerve beyond its physiologic tolerance. These should be loosened and the nerve function reassessed. Patients with sciatic nerve palsies after hip procedures should be positioned to relieve tension on the nerve (ie, hip extension and knee flexion). Poor positioning of the elbow, wrist, and ankle may similarly place nerves on stretch and tension and are potentially reversible causes of nerve palsy in the acute postoperative period.

For a patient whose nerve deficit is first recognized after the acute postoperative period, a history should be performed documenting the patient's symptoms, pain, and any changes over time. A thorough physical examination should be performed with a comparison to the contralateral side and to the documented preoperative examination. Every attempt should be made to obtain an accounting of the surgical procedure. Additional confirmation with electrodiagnostic and imaging studies may be warranted. Our approach to the nonsurgical and surgical management of iatrogenic nerve injuries is based on a large volume of referrals and an understanding of the preclinical study of nerve injury (Figure 2).

History and Physical Examination

A history of unremitting severe neuropathic pain immediately after surgery or after the anesthetic block has worn off is an ominous sign that needs to be taken seriously. This scenario typically occurs with entrapped or strangulated nerves. Hyperalgesia and allodynia are concerning symptoms for nerve transection and/or stump neuroma formation.

In contrast, a history of being normal postoperatively, then a few days later having an episode of sharp

debilitating pain, followed by paralysis, is more consistent with a brachial plexus neuritis (ie, Parsonage-Turner syndrome). Thus, a critical history is imperative regarding the characterization of pain, weakness, function, timing, and chronology.

Physical examination includes observing muscle bulk and atrophy, measuring active and passive range of motion, testing of sensory nerve distributions and muscle strength, and noting the presence or absence of a Tinel sign. When assessing sensation, static and moving two-point discrimination, light touch, pinprick, and vibratory sense should all be examined. In patients who are unable to cooperate with an examination, a skin wrinkle test may be valuable. Disruption of sympathetic tone results in the absence of skin wrinkling with the fingertips placed in warm water. Similarly, anhidrosis in a cutaneous nerve distribution indicates loss of autonomic function.

Muscle strength testing in the affected and contralateral extremities is graded according to a modified British Medical Research Council scale. Even if a muscle is not thought to be involved or related to the injured nerve, documentation of strength is important because it allows for future surgical planning for tendon or nerve transfers. Serial examinations over a period by the same evaluator allows for consistency in grading and determining whether any improvement has occurred. Our convention is that to be grade 3, the patient must have active range of motion against gravity equal to the passive range of motion. Even if the patient has some strength against resistance, the muscle group may not be graded at a 4 unless active and passive motion are equal.

A strongly positive Tinel sign after suspected nerve injury indicates axonal disruption. The location and radiating features should be well documented so that they may be compared on subsequent examinations or after nerve surgery. Regenerating axons will produce a Tinel sign that advances distally along the course of the nerve. A repaired nerve that continues to demonstrate a strong Tinel sign at the site of repair rather than progressing distally is concerning for neuroma formation.

Imaging Studies

Postoperative orthogonal radiographs should be scrutinized for any aberrant implant or fracture gapping, which suggest a cause of the postoperative nerve deficit. Advanced imaging is often complicated by the use of metallic implants, which create artifact at what is invariably the site most in need of scrutiny.

Ultrasonography provides at least two benefits over MRI. In the setting of metallic implants, interference may obscure imaging of the nerve even with metal subtraction sequences. Furthermore, ultrasonography is particularly well suited for visualizing nerves longitudinally. Ultrasound has been shown to identify both the topography of the nerve and pathologic changes in muscle as a result of denervation.[26] It is noninvasive and relatively low cost. Early ultrasonography may be helpful in identifying a transected nerve requiring surgical intervention or a nerve in continuity allowing continued observation. Ultrasonographic evaluation can also demonstrate later findings such as the presence of a neuroma.[27,28]

Electrodiagnostic Studies

All patients with continued neurologic deficits should undergo an initial electrodiagnostic examination (ie, nerve conduction velocity and electromyography [EMG] studies) 3 to 4 weeks after surgery. Nerve conduction velocity studies include the amplitude of compound muscle action potentials and sensory nerve action potentials (NAPs). EMG may show denervation changes (fibrillation potentials) as soon as 10 days after injury.[29] Earlier examinations, before the completion of Wallerian degeneration, will be misleading because they will falsely indicate that the muscles are normal.

When monitoring for recovery of nerve function, serial electrodiagnostic examinations performed every few months by the same practitioner in conjunction with repeat physical examination are ideal. Reduced recruitment of motor unit potentials (MUPs) can be seen after weakness on physical exam. Low amplitude, short-duration MUPs are indicative of early reinnervation due to regrowth. In contrast, reinnervation via collateral sprouting from undamaged, neighboring axons is demonstrated by high-amplitude, short-duration MUPs. It is critical to understand that electrodiagnostic studies correlate with muscle grade and objective sensory testing and do not correlate with functional outcome scores.[30]

Surgical Treatment

Appropriate timing of surgical intervention is one of the most important factors in the prognosis of these injuries. Primary nerve repair may be feasible at the time of injury, and acute transection of any major nerve identified in the operating room should be repaired in that setting. If a direct repair can be performed without excessive tension, it is done under an operating microscope to facilitate fascicular repair. The repair technique is surgeon dependent with no one method clearly outperforming any other. Typically, the fascicles are aligned with several epineural 8-0 or 9-0 nylon sutures. Augmentation of the repair has been described with fibrin glue and with a variety of bioabsorbable nerve wraps/conduits.[31]

When a direct neural repair cannot be performed because of segmental loss, blunt or stretch injury, interposition

Figure 3

A B C

Photographs showing nerve grafting with cabled sural nerve autograft. **A**, Median nerve defect after harvest for tendon transfer. **B**, Resection of the proximal nerve stump to healthy fascicles. **C**, Proximal reconstruction using the cabled sural nerve graft to approximate the caliber of the median nerve.

cable grafting should be performed. Autologous cable grafting remains the benchmark for large mixed and motor nerve lesions (Figure 3). The sural nerve can be easily harvested and provides nearly 30 to 35 cm of graft per leg, leaving only an area of numbness on the lateral side of the foot. Other potential sites of autograft include the superficial peroneal, saphenous, and medial antebrachial cutaneous nerves. Cable grafting to match the diameter of the nerve to be repaired increases the number of axons while maximizing the viability of a nonvascularized graft. Vascularized grafts have been described in the management of brachial plexus injuries but are less often used. Sensory nerve grafts have been placed retrograde so that regenerating nerve fibers are not lost to branching points. However, this dogma has not been shown to improve functional outcomes.[32]

Decellularized nerve allografts have been advocated by some authors because of donor site morbidity and the limited options for autologous nerve grafts and concerns regarding size mismatch. Motor regeneration has been inferior to autogenous grafts. However, sensory nerve function of small nerve defects has been successful after reconstruction. In a multicenter study of upper extremity lesions treated with allograft reconstruction, Cho et al[33] reported meaningful recovery in 89% of digital nerve repairs and 75% of median nerve repairs. When reconstructing a noncritical function or the potential for recovery is limited, the ease of use and limited donor site morbidity may favor the use of allografts.[34] For critical motor or mixed senory motor nerves, cabled autograft reconstruction remains the benchmark.[35]

Made up of a variety of absorbable and nonabsorbable materials, nerve conduits maintain a physical barrier containing a milieu that serves as a substrate for regenerating axons. The best indication for nerve tubes is in the reconstruction of digital sensory nerves. Their use in treating large mixed or motor nerve lesions is not currently supported by the literature.[36]

Delayed management of iatrogenic nerve injuries considers the patient's age, comorbidities, neurologic deficit, time from injury, and intraoperative findings. If there is potential for spontaneous recovery (and possibility for complete transection is low), the surgeon must balance observation for spontaneous recovery with time-dependent irreversible end plate degeneration. Interposition nerve grafting with autologous nerve or nerve transfers are performed ideally by 6 months from injury. Nerves regenerate at approximately 1 mm per day (or inch per month), and a motor end plate remains viable for approximately 1 year. Delay in reestablishing continuity of a motor nerve may doom the chance of success if the time for the nerve to regenerate to the target muscles is greater than the survival time of the motor end plate.[37] Although sensory innervation is important, sensory nerve function may return as late as three years after repair, thus timing for sensory reconstruction can be delayed if necessary.

When surgical exploration and reconstruction is chosen, all injured neural structures are identified. Previous surgery may make distinguishing these nerves difficult. Using the axiom of working from "known to unknown," a wider surgical exposure is usually necessary, confirming the identity of normal proximal and distal nerve, followed by dissection to the zone of injury.

If a nerve has been completely transected, it will likely have formed a neuroma. The neuroma is serially excised (ie, breadloafed) until healthy fascicles are identified. The same principles of tension-free surgical repair apply. When direct opposition

is not possible, interposition grafting discussed earlier should be used.

If the nerve is not transected, it may have formed a neuroma in continuity. Neuromonitoring can be informative at this later stage. NAPs can be performed as early as 6 weeks from injury and can provide information about conduction across the site of injury, dictating whether to perform neurolysis. When NAPs are present, the probability of reinnervation of the target muscle has been reported to be 88%. In these cases, careful neurolysis of scar is performed.[38] When the injury is more severe in one part of the nerve than another, internal neurolysis may be indicated. If NAPs are absent, there is a block to conduction. Excision of the damaged nerve as described earlier and interposition cable nerve grafting with autograft nerve or nerve transfers are recommended.

Nerve transfers have been developed to provide restoration of a more nuanced motion that direct innervation provides. A nerve transfer is the transfer of a less important and expendable motor nerve to a more important and denervated motor nerve. Nerve transfers diminish the time to reinnervation by coapting the viable nerve close to the motor end plate and eliminate the need for autologous nerve graft. They do, however, require one donor for each function and do not address sensory recovery. Sensory nerve transfers have been described as well to promote sensory nerve recovery. Although popularized in the upper extremity, nerve transfers in the setting of foot drop have also been demonstrated to be successful in select patients.[40]

End-to-side transfers have been reported to potentially preserve the motor end plates. They are most commonly used with ulnar nerve lesions, where an anterior interosseous nerve is transferred end to side distal to a mixed ulnar nerve repair.[41] A full discussion of both end-to-end and end-to-side nerve transfers is beyond the scope of this review, but suffice it to say that early referral to a peripheral nerve surgeon permits many more surgical options than may be available once the motor end plates become nonviable.

If the time to motor nerve reconstruction exceeds 12 months, traditional tendon transfer options, selected joint arthrodesis, or tenodesis should be considered to improve function. Specific examples of common clinical scenarios and subsequent application of these treatment principles are outlined in Table 4.

Table 4

Common Scenarios and Application of Treatment Principles

Scenario	Treatment Algorithm
Complete radial nerve palsy after humeral shaft fracture plating, no clinical recovery at 3 months, and electrodiagnositic studies suggest neurotmesis	Exploration of the radial nerve, with probable cable grafting if the neurotmesis is confirmed. PT to ECRB transfer is useful as a temporary dynamic wrist extensor splint during 6 months until anticipated motor recovery
Ulnar nerve palsy is found after medial pin for a pediatric supracondylar humerus fracture.	Most of these injuries resolve spontaneously over several months. Pin revision and nerve exploration are intuitive, but some suggest that the medial pin does not have to be removed[39]
Lateral femoral cutaneous nerve is cut during pelvic osteotomy.	Intraoperatively bury the distal end of the proximal nerve in muscle.
PIN deficit in the recovery room after radial plating from the anterior approach	Check for compartment syndrome, releasing tight bandages. Monitor for recovery and follow-up at 3 weeks and consider ultrasonography if no recovery at that time.
Dense sciatic palsy in the recovery room after THR with lengthening	Extend hip and flex knee and monitor for recovery.
Peroneal palsy in the recovery room after medial closing wedge osteotomy of the proximal tibia	Release tight or compressive dressings around the knee and monitor for recovery.
Thumb opposition weakness after carpal tunnel release	Early ultrasonography if no recovery. Consider nerve repair/grafting if identified early. Opposition tendon transfer if identified late.
Axillary palsy after arthroscopic Bankart repair	Monitor for recovery and consider ultrasonography if no recovery at 3 months. Consider nerve repair/grafting or nerve transfer if identified early.
Phantom limb pain after a transtibial amputation	Consider pain management and PM&R consultation for multimodal regimen including mirror therapy.

PIN = posterior interosseous nerve

Outcomes of Surgery

As in any peripheral nerve lesion, outcomes are dependent on the location and type of lesion, patient factors, and time to intervention. Lesions in continuity treated with neurolysis have a better prognosis

than discontinuous lesions. Within discontinuous lesions, shorter segment reconstructions are associated with better outcomes.

Although iatrogenic nerve injuries are usually isolated lesions, results can still be devastating. A retrospective study of 126 surgically treated iatrogenic nerve injuries demonstrated some improvement in approximately 70% of patients. However, at a mean follow-up of 18 months, only 24% achieved a "very good" outcome, defined as the patient experiencing considerably less pain and/or considerable improvement in motor or sensory nerve function. Notably, nearly two thirds of the injuries were treated greater than 6 months from injury, which may have led to poorer results.[1]

Early identification and management of iatrogenic nerve injuries is crucial to maximize outcomes. Experimental evidence shows that immediate direct nerve repair can halt motor neuron loss, maximizing the potential for return of function.[42] The results of a recent 2017 study with 122 patients show a statistically significant difference in satisfactory recovery rates for patients receiving intervention before 6 months compared with after 6 months, which are consistent with the previous literature.[43]

Therefore, patients should have the appropriate referrals to a surgeon who specializes in nerve repair and reconstruction as soon as feasible. Rarely does a review of the previous surgical report provide any information regarding the iatrogenic nerve injuries.[2] The most efficient method of referral comes directly from the operating surgeon. Even then, there is often a delay in presentation. In one large series at a single center specializing in traumatic nerve repair, most patients presented after 6 months, and most of the referrals were not from the operating surgeon who caused the injury.[1]

Medicolegal Aspects

Medical malpractice litigation is common, and most orthopaedic surgeons are predicted to have a medical malpractice claim during their careers.[44] A recent analysis of 464 malpractice claims in California for orthopaedic surgery demonstrates that the highest impact allegations were failure to protect structures in the surgical field. Nerve injury was the most common allegation, with 41 cases resulting in more than $5.8 million in payment to plaintiffs.[6]

Surgeons must appreciate that no matter how skilled or intelligent they think themselves to be, they are human, and the possibility of iatrogenic nerve injury exists in every case. Surgeons should communicate with patients that despite all the ways nerve injuries are avoided, the risk of iatrogenic injury is real and does occur. This phenomenon is especially important in the consent process and critical to forming a good therapeutic relationship with patients based in honesty. Good therapeutic relationships are helpful in preventing and limiting litigation. One analysis shows that having an adverse outcome not well explained in the surgical consent process was a risk factor for malpractice suits.[45] Finally, the documentation process should be thorough, from preoperative examinations (perhaps the injury was not truly iatrogenic), to intraoperative notations that nerves were protected, to the identification and description of the injury (was the nerve cut, crushed by a plate, or caused by retraction) to help maximize patient outcomes.

If a nerve is damaged intraoperatively, notify the patient as soon as feasible. Inevitably, the patient and his or her family will have questions about the injury. Maintaining the physician-patient relationship is critical, and communication must be open and honest. Being a patient advocate means putting the needs of the patient first. When appropriate, immediate referrals to a peripheral nerve surgeon should be made. If the patient pursues litigation, it is important to seek legal counsel. During the entire process, thorough documentation, prompt recognition and diagnosis, referral to specialists, and being a patient advocate can support the surgeon's defense.

Second Victim Phenomenon

A recent brave reflection of an ulnar nerve transection in an orthopaedic surgeon highlights, among other topics of operating room safety, the second victim effects that have previously gone under-reported.[46] The second victim phenomenon occurs when health care providers involved in an adverse event feel traumatized themselves.[47] It is important to appreciate the societal and personal factors associated with the surgeon's response to iatrogenic injury, where it is possible that shame and guilt unintentionally impede the process of prompt referral. A compassionate handling of not only the patient but also the surgeon facilitates appropriate surgical intervention in a timely manner, maximizing outcomes.

Summary

Orthopaedic and hand procedures are the most common cause of iatrogenic nerve injury, with rates approaching 20% in revision upper extremity surgery. The Seddon classification of nerve injury is useful in identifying the prognosis and management of iatrogenic nerve injuries outlined in Table 3. When iatrogenic nerve injuries are discovered during a surgery, an intraoperative consult is warranted and appropriately addressed. Nerve injuries suspected in the acute postoperative period should be evaluated for any reversible causes and addressed. Early clinical follow-up is warranted

to ensure improvement and appropriate therapy. When iatrogenic nerve injuries are identified late or have no recovery, additional studies, such as ultrasonography and/or EMG, are helpful in correctly diagnosing the injury. Treatment options include exploration and neurolysis, nerve repair, nerve grafting, or nerve transfers. In cases with no potential for recovery, tendon transfers may be warranted. Early recognition in the operating room and a high index of suspicion in the postoperative period can positively affect outcomes. The injury can be devastating to the patient and the surgeon and has the potential for significant medicolegal consequences, but neither should get in the way of prompt referral to a surgeon familiar with the management of iatrogenic nerve injuries.

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