INVITED REVIEW

HOW ELECTRODIAGNOSIS PREDICTS CLINICAL OUTCOME OF FOCAL PERIPHERAL NERVE LESIONS

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ABSTRACT: This article reviews the electrodiagnostic (EDX) prognostic factors for focal traumatic and nontraumatic peripheral nerve injuries. Referring physicians and patients often benefit from general and nerve-specific prognostic information from the EDX consultant. Knowing the probable outcome from a nerve injury allows the referring physician to choose the best treatment options for his/her patients. Nerve injuries are variable in their mechanism, location, and pathophysiology. The general effects of the injuries on nerve and muscle are well known, but more research is needed for nerve-specific information. Several factors currently known to influence prognosis include: nature of the nerve trauma, amount of axon loss, recruitment in muscles supplied by the nerve, the extent of demyelination, and the distance to reinnervate functional muscles. This article reviews these general concepts and also nerve-specific EDX measures that predict outcome after focal neuropathies.

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When evaluating patients with focal peripheral nerve lesions, the electrodiagnostic (EDX) medical consultant should provide several types of information in the EDX report and impression. Diagnosis and location are key elements of an EDX report.[1] The pathophysiology of the nerve lesion, such as whether there is axon loss and/or demyelination is also important. There is debate about the practice of describing the severity of the injury in some cases, particularly if there is no construct validity behind the categorization.[2] Prognosis, however, is an additional critical piece of information for both the patient and the referring physician. This review focuses on using EDX information to estimate prognosis for focal peripheral nerve lesions.

Prognosis is a valuable part of the EDX assessment for several reasons. Knowing the probable outcome from a nerve injury allows the treating physician to make a more informed recommendation regarding treatment options. For example, if a patient has a severe radial nerve injury unlikely to recover, tendon transfers may be a good early option. In some cases, early nerve transfers or grafting may be indicated. On the other hand, a patient with a good prognosis for recovery may be better treated conservatively with range of motion exercises (ROM) and splinting while awaiting spontaneous recovery.

For the patient, knowledge of prognosis may affect what one does at work or at home. A craftsman with an ulnar neuropathy with a low chance of recovery may seek to modify the workplace or elect to change occupation. A skier with a fibular neuropathy unlikely to recover may wish to change avocational interests or seek to modify his/her equipment. Others may be able to more readily accommodate to persistent deficits.

For these reasons, the EDX medical consultant should strive to provide as accurate prognostic information as possible from the EDX examination. The goal of this review is to discuss the types of nerve injuries that have better prognoses, to review which EDX measures are generally useful for predicting outcome, and to summarize the information available for focal injuries of the commonly affected and studied nerves so that nerve-specific information can be provided. This study also discusses the significant challenges in using EDX data to estimate prognosis, including: lack of information about the internal architecture of the nerve, anatomical variability, and significant impact of the timing of EDX studies.

FACTORS THAT INFLUENCE PROGNOSIS OF FOCAL PERIPHERAL NEUROPATHIES

Degree of Traumatic Nerve Injury. In the setting of trauma, peripheral nerve injury can be classified according to the degree of injury of axons and their supporting structures.[3] The classification of a particular nerve injury represents a significant determinant of outcome. Seddon defined 3 grades of classification: neurapraxia, axonotmesis, and neurotmesis.[4] Sunderland, on the other hand, used 5 grades numbered 1 through 5.[5] *Neurapraxia* is a primarily demyelinating injury which has a good prognosis, as most patients experience

Abbreviations: ADM, abductor digiti minimi; BR, brachioradialis; CB, conduction block; CFN, common fibular nerve; CMAP, compound muscle action potential; CSI, combined sensory index; CTS, carpal tunnel syndrome; EDB, extensor digitorum brevis; EDX, electodiagnostic; EI, extensor indicis; FDI, first dorsal interosseous; FPs, fibrillation potentials; MCP, metacarpophalangeal; MUAP, motor unit action potential; NCS, nerve conduction study; NCV, nerve conduction velocity; PACN, posterior antebrachial cutaneous nerve; ROM, range of motion; SNAP, sensory nerve action potential; TA, tibialis anterior; UNE, ulnar neuropathy at the elbow.

Key words: electrodiagnosis; focal neuropathy; outcome; prognosis; traumatic neuropathy

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recovery within 2–3 months when remyelination occurs and conduction block resolves[4]; this is synonymous with Sunderland grade 1 injury. In *axonotmesis*, there is axonal disruption with some preservation of supporting structures such as the perineurium or epineurium. Sunderland divides these injuries into grade 2 (disruption of axons), grade 3 (disruption of axons and endoneurium), and grade 4 (disruption of axons, endoneurium, and perineurium). Axonotmetic injuries have variable prognosis depending upon the ability of axons to regrow. Nerve injuries with more severe disruption of supporting elements (Sunderland grade 3 or 4) will have a lower chance of recovery than those with minimal disruption (grade 2).[5] In injuries with extensive disruption of the fascicular structure and local scarring and fibrosis, there is a lower chance of axons growing through the region of injury and reaching their end organs on the other side. Similarly, neuroma formation at the site of injury makes it unlikely that axons will make it through the neuroma and across the injury site to fulfill their destined function. Finally in *neurotmesis*, in which there is complete disruption of both the axons and the supporting structures of the nerve, there is little chance of recovery absent surgical intervention.

Degree of Demyelination. The extent of demyelination may also influence recovery, but this factor appears to have less impact than the degree of axon loss. Severe myelin disruption can produce conduction block and clinical deficits. After loss of myelin, Schwann cells have the capacity to remyelinate demyelinated areas of the nerve, and conduction improves. The morphology and function of the myelin will not be the same as before the injury.[6] and slowed conduction velocity may persist.[7] Because of the capacity for remyelination, in general neurapraxic traumatic nerve injury has a good prognosis, with the majority of patients experiencing substantial recovery within 2–3 months. In some chronic entrapment neuropathies, the degree of slowing is associated with the prognosis after treatment,[8,9] although even here it is unclear whether the demyelination itself is the key factor or whether greater slowing is a surrogate measure of other processes such as conduction block and axon loss.

Extent of Axon Loss. The degree or amount of axon loss has a large impact on prognosis. Nerve injuries with minimal axon loss generally have a better prognosis than those with loss of most or all axons. Motor axons fortunately have the capacity to reinnervate some denervated muscle fibers by distal axon sprouting. Thus relatively mild degrees of axon loss can be accommodated by this process even if no axons regrow across the site of the injury. It is estimated that human motor axons can reinnervate approximately 5 times their original muscle fiber territory by this process[10]; hence, even substantial loss of axons can be handled without axon regrowth starting from the site of axon injury. However, when the great majority of axons are lost (e.g., >90%), the small number of surviving axons cannot expand their territory sufficiently to produce the strength needed for full function.

Distance to the Muscle. For axonal injury, the distance from the injury site to the muscle also has an important impact on prognosis. In axonotmetic injuries in which the endoneurial tubes are preserved, the axons can traverse the segment of injury in 8 to 15 days and regenerate along the distal nerve segment at a rate of 1–5 mm/day[5]; this is faster proximally and faster in younger individuals.[11] When the muscle is close, axons can reach muscle fibers while the axon tubes are still capable of accommodating regrowth and before axonal stenosis occurs and prevents reinnervation. The muscle will still be viable and not yet fibrotic, and the patient may still have adequate joint ROM to use regained strength. However, in severe injuries with complete or near-complete loss of motor axons, after 18–24 months the muscle can no longer be reinnervated even if the axons reach it.[12] The axon tubes will be stenotic, the muscle may be fibrotic, and contractures may have formed. No treatments, including electrical stimulation, are known to increase the time during which muscles can remain viable for reinnervation, although recent studies indicate early promise in using new electrical stimulation paradigms to promote axon growth after nerve surgery in animal models and after carpal tunnel release.[13,14] Thus in complete axonotmetic injuries with long distances to reinnervate, such as lower trunk brachial plexus injuries, the prognosis is poor even if fascicular disruption is minimal.[15] Moreover, when the injury is very proximal in the peripheral nervous system, reinnveration may not occur at all. For example, root avulsions have an especially poor prognosis, even with surgical repair, as motor neurons have little capacity to regrow in this setting.

WHICH EDX MEASURES ARE GENERALLY USEFUL FOR ESTIMATING PROGNOSIS?

Compound Muscle Action Potential (CMAP) Amplitude. CMAP amplitude can be a useful method for estimating the extent of axon loss or axon preservation. CMAP amplitude is roughly proportional to the number of muscle fibers depolarizing under the electrode; hence, it is also related to the number of excitable motor axons.

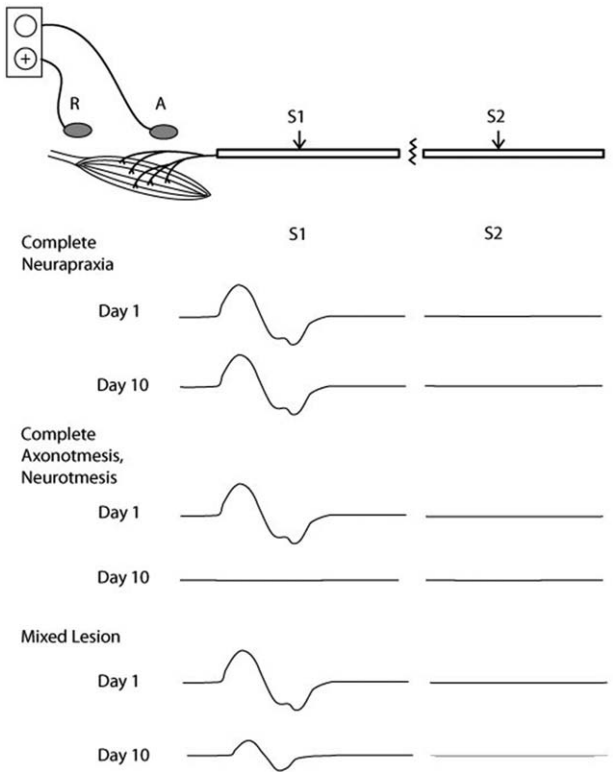


FIGURE 1. Motor nerve conduction findings expected in: neurapraxia, axonotmesis/neurotmesis, and mixed lesions at days 1 and 10 postinjury. Recording is from a muscle distal to the nerve injury site. Stimulation occurs both distal (S1) and proximal (S2) to the site of nerve injury either at Day 1 or Day 10. A, active electrode; R, reference electrode.

When stimulating distal to the injury site and recording from the supplied muscle, the CMAP amplitude gives an estimate of the degree of axonal preservation. If one stimulates proximal to the lesion, however, the CMAP is also influenced by the extent of demyelination and conduction block (Fig. 1). Therefore, by comparing the distal CMAP and proximal CMAP amplitudes, one can also estimate the degree of conduction block.

There are limitations to using the CMAP for estimating the number of axons. Early on after injury, it can take up to 7 days for the CMAP to diminish in size or disappear.[16,17] In contrast, it takes up to 10 days for the sensory nerve action potential (SNAP) amplitude to reach its nadir; the CMAP disappears first due to earlier failure of the neuromuscular junction.[16] Hence, using this measure too early can provide misleading results and overestimate the number of viable axons. Generally it is preferable to allow 7 or more days to pass before using the CMAP amplitude to distinguish between neurapraxia and axonotmesis or to estimate injury severity.

Beginning several months after injury, the CMAP amplitude can also overestimate the number of preserved axons. Once sufficient time has passed for distal axon sprouting to occur, typically 2–3 months, each axon will supply more than its usual number of motor axons. When excited, each axon will produce a larger motor unit action potential (MUAP), and when the whole nerve is stimulated a larger CMAP will be produced. One then can overestimate the number of excitable motor axons. Because the larger CMAP is recorded after axon sprouting has occurred, there is no longer the same unused capacity for reinnervation. Hence, a CMAP that is 30% of normal at 1 month after injury is a better sign than the same size CMAP 1 year after injury.

Another limitation of using the CMAP to estimate prognosis relates to which muscle is used for recording. For example, in upper brachial plexus injuries, standard upper limb motor conduction studies, such as median or ulnar, are of little use, because they reflect lower brachial plexus innervation. Instead, one should consider recording from the biceps (upper trunk) or flexor carpi radialis (middle trunk) that are more relevant. Despite these limitations, CMAP amplitude is an important measure of axon loss, injury severity, and ultimately prognosis.

Sensory Nerve Action Potential (SNAP) Amplitude. The SNAP amplitude is a reflection of the number of viable, excitable sensory axons. There is some evidence that patients with preserved SNAP amplitude have less severe injury and better sensory outcomes.[18,19] However, SNAP amplitudes may be less useful than CMAP amplitudes for several reasons. First, SNAPs are smaller and can be more challenging to record than CMAPs. Moreover, motor strength may play a more obvious role in functional recovery than sensation. Finally, because sensory recovery is not dependent upon intact and viable muscle, it can recover over a longer period than motor function through a combination of axon regrowth and collateral sprouting from adjacent areas.[20]

Nerve Conduction Velocity (NCV). Slowing of NCV is primarily a reflection of the extent of demyelination of the nerve, although NCV is also influenced by loss of larger diameter axons. As noted above, demyelination has less impact on prognosis than axon loss. Hence, NCV is not a strong predictor of recovery. In some cases, slowing may actually be a good predictor of outcome,[21] as those with impairments resulting from demyelination are more likely to recover than those with deficits due to axon loss.

Needle EMG: Spontaneous Activity. With motor axon loss, fibrillation potentials (FPs) and positive sharp waves appear in muscle after 10–14 days in

Table 1. Generally useful prognostic measures for focal peripheral nerve lesions.*

Useful prognostic factors	Good prognosis	Poor prognosis
Recruitment of muscle distal to lesion	Normal or mildly ê	Discrete or absent
Distal CMAP	Normal or mildly ê	Absent
Conduction block or slowing	Present	Absent
Distal SNAP (for injuries distal to DRG)	Present	Absent
Distal SNAP (for root avulsions)		Present

*These factors are most useful after 7 days, but within the first 2–3 months after injury, before reinnervation by collateral sprouting.

CMAP, compound muscle action potential; SNAP, sensory nerve action potential; DRG, dorsal root ganglion.

muscles near the injury site and after 3–4 weeks in more distal muscles. These potentials are recorded in the presence of even small degrees of axon loss and are typically graded on an ordinal scale from 1+ (reproducible potentials in more than 1 area) to 4+ (obliterating the baseline). Higher grades represent more axon loss than lower grades. One might suspect that the presence and density of FPs and positive sharp waves would significantly impact prognosis, but it does not, either in focal or generalized neuropathies.[18,22,23]

There are several reasons for the lack of association between FP density and outcome. First, the grading scheme is an ordinal scale, not a ratio or interval scale. Thus a grading of 2+ positive sharp waves likely represents more axon loss than 1+, but not necessarily twice as much; we actually do not know how much more. Additionally, one does not require complete axon loss to observe 4+ FPs, and it may be that loss of a relatively small proportion of the total axons can result in 4+ FPs.[24]

Needle EMG Recruitment. Voluntary MUAP recruitment on needle EMG can be useful to establish the presence of intact motor axons under voluntary control and can provide a rough indication of the degree of axon preservation. This measure is especially useful for examining the muscles immediately distal to the injury site. The presence of MUAP recruitment in these proximal muscles indicates that the injury is not complete and that at least some axons are successfully traversing the injury site and exciting muscle fibers on the other side. When recruitment is normal or only mildly reduced in a muscle innervated just distal to the injury site, this is an important favorable prognostic factor that may be as important as, or more important than, CMAP amplitude.[22]

Recruitment has several limitations as a prognostic measure. First, it is difficult to quantify and is usually recorded on a subjective ordinal scale, such as full, reduced, discrete, or none. In addition, poor or absent recruitment does not indicate unequivocal axon loss; conduction block can produce similar findings. Finally, recruitment in 1 muscle does not necessarily reflect function of other muscles or of the nerve in general, and it is not always clear how much the findings in an individual muscle can be extrapolated to other muscles supplied by the same nerve.

Table 1 summarizes which EDx findings are most useful for estimating prognosis.

WHAT GENERAL CHALLENGES EXIST IN USING EDX MEASURES TO PREDICT OUTCOME?

EDX measures primarily reflect nerve and muscle function. These tests can detect the presence of functional axons and the ability of the nerve to conduct, even partially, across the injury site. However, a major limitation is the inability to obtain a structural picture of the nerve. For example, after trauma one cannot resolve grades of axonotmesis. One cannot resolve whether axon loss is in the setting of intact neural tubes, or if it is accompanied by severe disruption of intraneural architecture; the former has a relatively good prognosis, and the latter is poor.

There are also challenges to carrying out clinical research on the ability of EDX to predict outcome. Because nerve injuries are variable in their mechanism, location, pathophysiology, and severity, subjects in a study will be far from uniform, which limits generalizability. If some patients undergo surgery or other interventions and others with similar injuries do not, it can be unclear how much of the outcome is related to natural history and how much comes from the intervention. It is also often unclear what the primary outcome measure of these studies should be. Subjective improvement or complete subjective resolution is attractive, as it is from the patient's perspective and is perhaps what matters most. On the other hand, measures of strength, sensation, and function may offer more objective quantitative outcomes and greater statistical power. Thus the definition of a good outcome differs according to different authors. Some consider "symptom relief" as a good outcome,[7,15,25] while others have used "muscle strength,"[26] or "function of muscle."[12]

Another methodologic issue with studying the impact of electrophysiology on prognosis is that many investigators look for a linear relationship between EDX measures and outcomes. For instance one might assume that shorter latencies

suggest a better prognosis than longer latencies. This is not always the case. For instance in carpal tunnel syndrome (CTS), as discussed below, those with shorter or longer latencies do not do as well after carpal tunnel release as those with moderate delays in conduction. This inverted U-shaped relationship can be overlooked if one is only looking for linear relationships.

In some studies, there may also be an interaction between the EDX assessment and treatment options. For instance those with more severe nerve impairment may be more likely to undergo invasive treatment procedures[27]; in those cases, it can be challenging to sort out the impact of EDX measures versus treatment, because treatment may be dependent upon diagnostic studies.

Timing of the EDX study and the outcome measurement are also important factors to account for in studies of the prognostic value of EDX measures. When EDX studies are performed very early after onset, before sufficient time has passed for Wallerian degeneration and before the start of reinnervation, there may be more uncertainty than when studies are performed several months later. Studies performed much later will offer more confidence, but they may be less useful if one must wait until the nerve injury has already stabilized and recovery is self-evident to provide a reliable prognosis.

In terms of timing, the optimal time frame for the EDX study will depend upon the question being asked. If one can stimulate above and below the potential injury site, one can differentiate between neurapraxia and axonotmesis/neurotmesis by day 7. However, the most diagnostic and localizing information can usually be obtained 3–4 weeks after injury, when needle EMG will show abnormal spontaneous activity in denervated muscles. In the setting of known neurotmesis and subsequent surgical repair, it may not be useful to perform EDX studies for several months until axons have had an opportunity to cross the surgical site and reinnervate muscle fibers.

Because strength can recover after peripheral nerve injuries for up to 2 years after onset, timing of outcomes measures can also impact studies of prognosis. When outcomes measures are obtained only a few months after onset, there is a greater likelihood that one will miss a good outcome that may become apparent only later. On the other hand, if subject inclusion is limited to only those with 2 years of follow-up, many patients will be excluded or lost to follow-up, and studies may be underpowered and ultimately not reflect the population at large.

Many of the studies we rely upon for information about prognosis are retrospective. While these studies provide much useful data, they are not as valuable as prospective studies. A much greater degree of standardization is possible in prospective studies with respect to inclusion/exclusion of subjects, which EDX measures are obtained, and which outcomes are assessed. Looking back at medical records for objective measures of strength, sensation, or function can be challenging due to absence of information, intraobserver variability, and limited follow-up times.

HOW MUCH VARIABILITY IS THERE BETWEEN NERVES?

In addition to the general principles of how one might use EDX measures to establish prognosis, there is considerable variability between different nerves in the limbs and face. This variability limits extrapolation and requires one to customize the assessment to each nerve.

First, there are *anatomical* variables that influence the prognosis of individual focal nerve lesions. Distance is a significant factor. Shorter nerves have a limited distance to grow to resupply muscles, such as the facial nerve, and they have different challenges than those with longer distances, such as proximal ulnar or sciatic nerve lesions. As discussed above, muscle may remain viable for only 18–24 months postdenervation; hence, muscles distant from the site of a severe nerve injury will have lower chances of recovery than those with shorter distances to cover. Intraneural architecture also plays a material role. Some nerves, such as the tibial, have many small fascicles separated by generous amounts of soft tissue, whereas others, such as the fibular nerve, have a few large fascicles with less intervening connective tissue; the latter may be more susceptible to injury.[28] Blood supply may also be different and can affect the capacity to recover.

For cranial nerves in particular, aberrant regeneration leading to synkinesis can be a limiting factor in recovery and can be problematic for injuries to the facial or laryngeal nerves.[29,30] After disruptive injuries to these nerves, axons can regrow from the site of injury but grow in the "wrong" neural tubes and supply different muscles than their original destination. This regrowth might not produce functional recovery and can even result in more dysfunction than if regrowth did not occur at all. In contrast, synkinesis is generally not a problem in limb nerves and muscles.

The function and functional requirements of each nerve are also variable. The facial nerve requires fine, precise control with relatively little force, as does the laryngeal nerve. In contrast, the femoral nerve does not require particularly fine control but needs to produce sufficient knee

Table 2. Best and worst prognostic groups for specific nerve injuries.*

Nerve	Best prognostic group	Poor prognosis
Median	SNAPs Present, CSI 2.5 – 4.6 Motor latency < 6.5 ms	Normal Study Absent CMAPs and SNAPs
Ulnar	Conduction Block or slowing across the elbow. Normal CMAP amplitudes.	Small or absent CMAP, no conduction block
Radial	BR recruitment reduced or normal	BR recruitment absent or discrete Absent CMAP to Extensor Indicis
Fibular	TA and EDB CMAP present	TA and EDB CMAP absent
Facial	CMAP >%30 of contralateral side Presence of Blink Reflex	CMAP < 10% of contralateral side

**CMAP, compound muscle action potential; SNAP, sensory nerve action potential; CSI, combined sensory index; BR, brachioradialis; TA, tibialis anterior; EDB, extensor digitorum brevis.*

extensor strength for weight bearing and functional ambulation. Different segments of each nerve may play more or less critical roles in function. For the ulnar nerve, innervation of proximal muscles, such as flexor carpi ulnaris or flexor digitorum profundus, are helpful, but innervation of distal hand muscles is more critical for functional recovery. However, for the fibular nerve, the proximally innervated tibialis anterior and fibularis longus are important for function, while distal muscles play a much smaller role; here just a few centimeters of reinnervation may be sufficient for a good outcome.

Although muscle strength is more straightforward to quantify, sensation plays a very important role in functional recovery for some nerves. The median nerve sensory supply covers the majority of the hand, and without adequate sensation, even with good motor strength, it would be difficult to use the hand to its full capacity. On the other hand, some nerves such as the anterior interosseous have no cutaneous sensation, and others, such as the deep fibular have limited sensory distributions. Lack of sensory function in the latter nerves would not be expected to produce a material functional deficit.

The mechanism and pathophysiology of injury also varies between nerves. The median nerve is most commonly affected by distal entrapment at the carpal tunnel, which is a predominantly demyelinating injury. In contrast, the ulnar nerve tends to have a substantial frequency of axon loss even in chronic compressive injuries.[31] Radial and sciatic nerves are rarely entrapped by chronic ongoing compression but are commonly affected by traumatic injury of the upper and lower limbs, respectively.[32] Each of these mechanisms of injury potentially carries a different likelihood of a good outcome, and prognosis may be different even with similar EDX measures.

Not only does natural history vary considerably between nerves, but so do treatment options. For some nerves there are good treatment options even if the nerve does not regenerate. In the setting of radial neuropathy, tendon transfers can produce a good functional outcome even in the absence of strength recovery in radial innervated muscles. Custom fabricated ankle foot orthoses can compensate for ankle dorsiflexor weakness in fibular neuropathy. There are fewer good options, however, for severe brachial plexopathies or complete median or ulnar nerve lesions.

SPECIFIC PROGNOSTIC INFORMATION FOR EACH NERVE

Some of the key prognostic variables for the more common focal peripheral nerve injuries are listed below. While it would be desirable to perform a literature synthesis and evidence-based review of this topic, the state of the literature and the research methodologic challenges noted above do not permit this type of approach at present. Thus we do not yet have the level of evidence needed to make definitive statements about prognosis in many cases. Table 2 summarizes useful methods for estimating prognosis for individual nerves.

MEDIAN NERVE

The median nerve supplies several wrist and finger flexors of the upper limb, as well as several intrinsic hand muscles. Terminal branches supply the thenar muscles, which are responsible for fine movements of the thumb. The nerve also provides an important sensory supply to most of the palmar aspect of the hand and digits.

The most common site of median nerve impairment is at the wrist. Electrodiagnosis is very helpful in both the diagnosis and prognosis of CTS. While it is difficult to measure sensitivity in the absence of a superior gold standard, recent reviews indicate approximately 85–90% sensitivity.[33] Specificity depends upon how the testing is interpreted and the population being studied. If the problem of multiple testing and comparisons can be avoided, and appropriate tests are used, it should be >95%.[34] However, there are also some populations which may have a lower specificity, and care should be taken when interpreting results in individuals in these cohorts.[35] The natural history of CTS is not well studied, because such a great proportion of

individuals receive treatment early in the course of their disease. Limited data suggest that approximately half of patients develop progressive clinical deficits, and half are relatively stable.[36]

EDX studies, primarily nerve conduction studies, offer some ability to predict outcome after carpal tunnel release surgery.[9,26,27,37,38] Although many studies suffer from the inability to detect nonlinear associations, it appears from these studies that those with moderate NCS abnormalities have the greatest likelihood of achieving good relief of symptoms after surgery, while those with relatively normal studies, or those with marked abnormalities have a lower chance of complete relief.

One of the more comprehensive studies comes from Bland,[26] in which he measured NCS abnormalities in patients preoperatively and collected outcomes information postoperatively. Those with normal studies and those with more severe changes (motor latencies exceeding 6.5 ms) had only approximately a 40% chance of complete symptom resolution, whereas those with moderate changes between the 2 extremes did considerably better. A similar finding was reported by Malladi et al.[9] using the combined sensory index (CSI) as the NCS variable.[39] The CSI is calculated by adding 3 peak sensory latency differences: median minus ulnar to the ring finger; median minus radial to the thumb; and median minus ulnar across the palm; in healthy individuals this is < 1.0 ms. Patients with a CSI of 2.5–4.6 had the best prognosis for resolution of pain and paresthesiae following surgical intervention, while those with more or less severe changes had less likelihood of a successful surgical result. As a result of these studies, I insert Figure 2 in reporting for patients with CTS.

While there are other sites of nerve injury of the median nerve, such injuries are relatively rare. There is too little information specific to any single site to provide meaningful nerve-specific data.

ULNAR NERVE

The ulnar nerve supplies 2 flexors in the forearm and the majority of the intrinsic hand muscles. It provides sensory function for the ulnar side of the hand and usually the small finger and the ulnar side of the ring finger. While the forearm flexors are not critical for function, the intrinsic hand muscles and the sensory supply are important for hand function.

The most common site of ulnar nerve compression and traumatic injury and is at the elbow. Injuries are often nontraumatic and can commonly be attributed to prolonged or repetitive mechanical compression or stretch during elbow flexion.[40,41] Other potential causes include direct trauma, local fracture, burns, compression from fibrous bands, arthritic hypertrophy, or masses in the epicondylar groove or cubital tunnel.[25] Ulnar neuropathy at the elbow (UNE) is often attributed to the superficial location of the nerve as it approaches the epicondylar groove between the olecranon and medial epicondyle and its course through a narrow cubital tunnel created by the humeral-ulnar aponeurosis. The diagnostic sensitivity of EDX studies has been reported to range from 37% to 86%, and specificities of 95% or higher have been reported when using AANEM Practice Parameter criteria.[42] The sensitivity will also depend upon the specific techniques used. For example, a comparison between across-elbow and forearm velocities has a much lower sensitivity (51%) than comparing across-elbow velocities with reference values (80%),[43] likely because the forearm segment slows in focal neuropathy as well. The natural history of UNE is variable, with approximately half of patients noting spontaneous improvement in the absence of surgical intervention.[25]

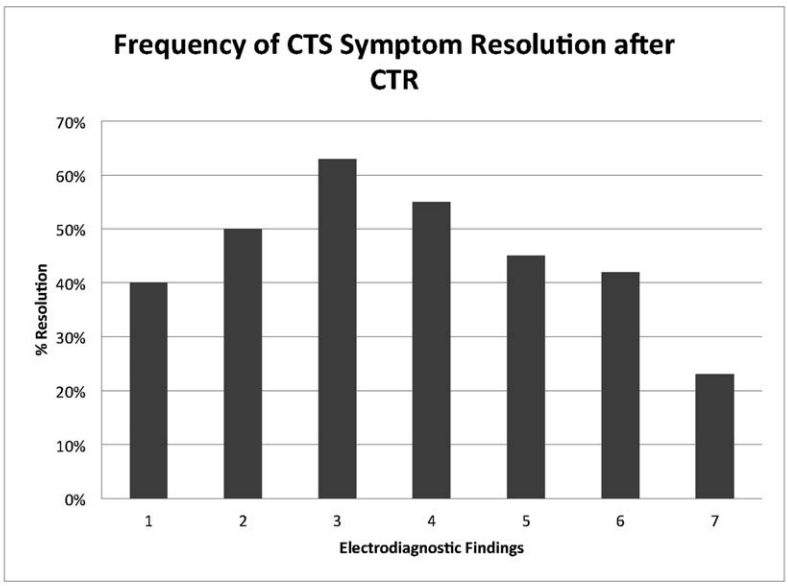
The 2 most important measures that predict a good outcome for patients with UNE are preserved CMAP amplitude in ulnar hand muscles and the presence of conduction block (CB) with slowing across the elbow.[18,21,31,44] In 1 study,[18] overall subjective recovery was best predicted by the combination of the presence of CB to the first dorsal interosseous muscle (FDI) and normal distal abductor digiti minimi (ADM) CMAP amplitude; 87% of patients recovered if the combination of CB and normal CMAP was present, while only 7% recovered who did not meet either criterion. The authors of this study reported that these 2 variables, taken together, produce a larger degree of separation of outcomes than any single variable alone (Fig. 3).[18] Other studies have also reported that presence of conduction block or slowing (indicating demyelination) is suggestive of a good outcome.[21,44] Two studies have suggested that the presence of an ulnar SNAP suggested a good prognosis[45,46]; an absent preoperative SNAP predicted less postoperative improvement in sensory symptoms, and a normal presurgical SNAP correlated with better postsurgical outcomes. There is little in the literature to suggest that needle EMG findings play a significant role in estimating prognosis.

While the ulnar nerve may also be impaired at other sites, such as at the wrist, such injuries are less frequent than UNE. There are insufficient data to allow reliable prediction of outcome.

RADIAL NERVE

The radial nerve supplies essentially all the extensors of the elbow and wrist, plus finger

Below are the reported frequencies of complete symptom resolution after carpal tunnel release according to electrodiagnostic criteria. These outcomes are only reported for electrodiagnostic criteria and should not be used as the only predictive findings. Other factors may have strong influences on outcomes.



Electrodiagnostic Criteria		Frequency of Symptom Resolution
1.	Normal study	40%
2.	CSI 1.0 – 2.5	50%
3.	CSI 2.5 – 4.6	63%
4.	CSI > 4.6, Sensory responses present	55%
5.	Sensory responses absent, motor latency < 6.5 msec	45%
6.	Motor latency > 6.5msec	42%
7.	Absent motor and sensory responses	23%

FIGURE 2. Prognostic Information on likelihood of complete symptom resolution in patients with carpal tunnel syndrome if they undergo carpal tunnel release.[9,26]

extensors crossing the metacarpophalangeal (MCP) joint. It also supplies sensation to most of the dorsum of the hand. The radial nerve is predominantly a motor nerve, which reduces the chance of aberrant motor/sensory reinnervation. Radial nerve-innervated muscles perform similar extensor functions, thus there is a decreased chance of aberrant innervation of muscles with opposite functions compared to some other nerves.[47]

While the radial nerve is not commonly chronically compressed by surrounding structures, it is the most commonly injured nerve in peripheral nerve trauma.[32,48] Injuries most frequently result from motor vehicle accidents, followed by penetrating injury, falls, and industrial accidents.[32] It has been reported that 2–16% of humeral fractures produce radial nerve injury.[49] This is largely due to the proximity of the nerve to the humerus as it courses along the spiral groove. It can also become incarcerated between the ends of a fractured humerus during closed reduction.

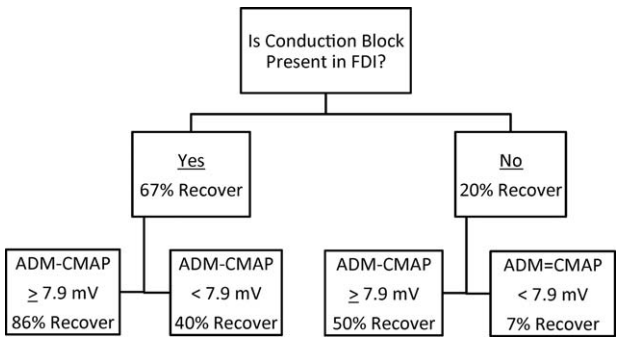


FIGURE 3. Recursive partitioning approach to establishing prognosis in patients with ulnar neuropathy.[18]

When injury occurs at this site, the triceps is generally spared, as it is supplied by branches departing from the nerve in the axilla, but wrist and finger extensors are severely affected. The brachioradialis (BR) muscle, an elbow flexor, is the first muscle supplied after the spiral groove; function of this muscle is an important contributor to prognosis. The sensitivity of electrodiagnosis in detecting radial neuropathy is not well studied. Natural history is generally favorable, with the majority (nearly 90%) having a good outcome in nontraumatic radial neuropathies.[50]

Electrodiagnosis can provide important information for determining outcome after radial nerve injury. In 1 study,[22] the most useful prognostic indicators were recruitment in the BR and CMAP amplitude recorded from the extensor indicis (EI). When recruitment in BR was full, 100% had a good outcome, even if distal muscles had poor recruitment. In addition, 80% of those with discrete recruitment (reduced numbers of rapidly firing MUAPs) in BR did well, whereas only 38% of those with absent recruitment in BR had a good outcome. CMAP amplitude from the EI contributed to estimation of prognosis, but less so than recruitment. If the CMAP was $\geq$0.5 mV, 100% had good recovery, whereas if the CMAP was <0.5 mV, 75% had a good outcome. It is surprising that of those with absent CMAPs (suggesting essentially complete motor axon loss), 65% still had good outcomes. The impact of BR recruitment on prognosis estimation suggests that recruitment of muscles innervated by the nerve just distal to the injury site may play a more instrumental role in prognosis than distal CMAP amplitude for some nerve lesions. The presence of MUAP recruitment demonstrates that some axons are traversing the injury site, and there is a pathway for others to establish reinnervation to distal muscles. As with other examples, the density of FPs has not been shown to be correlated with outcomes after radial neuropathy. Just as for the ulnar nerve, combining 2 variables (in this case recruitment and CMAP amplitude) appears to offer more separation into prognostic groups than using a single variable alone (Fig. 4). Another study looking at outcomes in nontraumatic radial neuropathies used multiple logistic regression analysis to find that conduction block on nerve conduction studies, younger age, and less severe initial weakness are indicators for a good prognosis.[50]

A normal posterior antebrachial cutaneous nerve (PACN) study has also been correlated with clinical improvement at 3 months. Conversely, PACN abnormality was associated with radial motor axon loss and a poorer prognosis. The PACN study is a simple adjunct which provides additional information relating to the diagnosis and prognosis of radial injuries.[19]

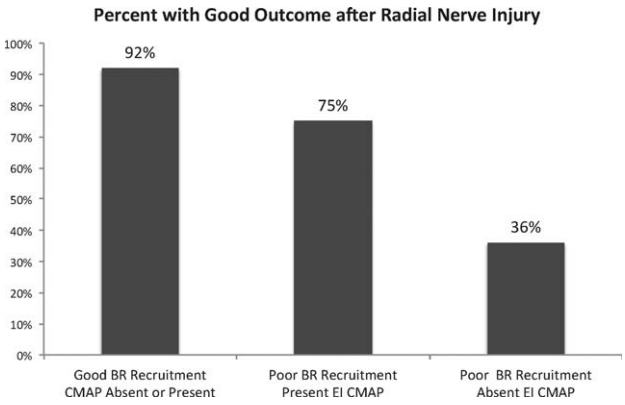


FIGURE 4. Percent of patients who achieve good recovery after radial nerve injury. Of those who have good recruitment in brachioradialis 92% have a good outcome regardless of CMAP presence or size. When recruitment is poor, the presence of a response from extensor indicis suggests that 75% will do well, whereas only 36% of those without a response will have a good outcome. CMAP, compound muscle action potential; BR, brachioradialis; EI, extensor indicis.[22]

FIBULAR NERVE

The fibular nerve (formerly known as the peroneal nerve)[51] supplies ankle dorsiflexors and evertors and sensation over the dorsum of the foot. It arises as a branch of the sciatic nerve, but in contrast to other nerves, fibular nerve fascicles remain separate in the sciatic nerve and do not generally intermix with those destined to form the tibial nerve. The fibular nerve is the most commonly injured peripheral nerve in the lower extremity when injuries to the fibular division of the sciatic nerve are included.[52] These injuries frequently result from blunt and penetrating trauma, acute and chronic compression, or idiopathic etiologies. Predisposing factors include recent anesthesia/surgery, weight loss, recent prolonged hospitalization, diabetes, peripheral neuropathy, prolonged squatting, and braces/casts.[53]

When assessing prognosis for fibular nerve lesions, it is important to carefully consider which muscles to evaluate. For reasons stated above, I typically record from 2 channels simultaneously (TA and EDB). The diagnostic sensitivity of EDX assessment in fibular neuropathy depends upon the techniques used, the threshold used for diagnosis, and the study population, but it is reported to be between 45% and 100%.[54] Specificity is not well studied because of the lack of large control groups, but it is generally reported to be very high, often near 100%.[54] The natural history in the absence of trauma and underlying polyneuropathy is generally quite favorable, with nearly 90% reportedly having a good outcome.[50]

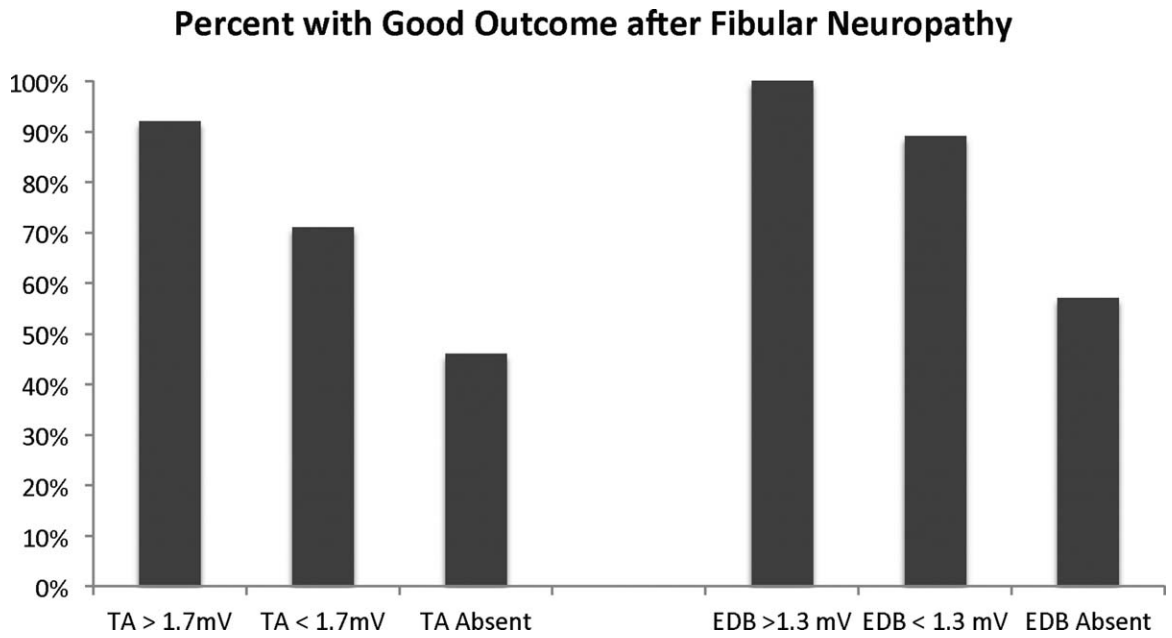


FIGURE 5. Percent of patients with fibular neuropathy who achieve a good outcome, based on CMAP amplitude in tibialis anterior (left 3 bars) and extensor digitorum brevis (right 3 bars).[55]

There have been several studies in which EDX measures are most useful for assessment of prognosis. One study,[55] combined NCS to TA and EDB with recruitment data and showed a linear relationship between CMAP amplitude and outcome (Fig. 5). Of those with a normal CMAP to the TA, 92% had a good outcome, whereas only 46% of those with an absent CMAP did well. This study also demonstrated that additional useful information was obtained by recording from both EDB and TA, as opposed to recording from only a single muscle. Although the numbers were too small to make meaningful conclusions from the data, good recruitment in the TA muscle also predicted a good outcome. The presence or density of FPs did not impact prognostic outcome.

Similarly, in a series of patients with common fibular mononeuropathy, Katirji and Wilbourn showed that axon loss was substantial in 80% of cases and that TA CMAP amplitude may be best suited to assess the severity of the injury.[56] Selective involvement of motor fibers to EDB versus TA has also been shown, with motor fibers to EDB more likely to undergo axonal degeneration and fibers to TA more likely to undergo conduction block in common fibular neuropathy.[57,58] This again speaks in favor of TA CMAP recording when assessing prognosis in these patients.

Normal sensory nerve conduction studies distal to the fibular head and conduction block isolated to the fibular head may predict a good outcome.[59] Another report[60] has suggested that slowing may play a role in prognosis, as those who have NCV < 30 m/s having a worse outcome; however, it is not clear if slowing has a significant unique contribution once MUAP recruitment and conduction block are considered.[55]

As with radial neuropathy, Bsteh and colleagues reported that a good outcome is predicted by younger age, greater strength on initial evaluation, and the presence of conduction block on nerve conduction studies.[50]

After traumatic injury to the sciatic nerve, which commonly affects the fibular branch more than the tibial branch,[61] it appears that the prognosis for fibular branch recovery is not as good as for the tibial branch. Several factors may be at play. The fibular nerve is fixed at both the sciatic notch and the fibularis longus muscle, as the common fibular nerve (CFN) passes over and around the fibular surgical neck, while the tibial nerve is fixed only at the sciatic notch. Moreover, the tibial nerve has a better blood supply and more fascicles with intervening connective tissue than the CFN, allowing more force to be absorbed by these structures.[62]

FACIAL NERVE

The facial nerve supplies the majority of muscles in the face and head, with the exception of the muscles of mastication (supplied by the trigeminal nerve) and the extraocular muscles (supplied by cranial nerves III, IV, and VI). Facial nerve lesions, result from a variety of etiologies. Idiopathic peripheral facial palsy, often known as Bell palsy, is thought to result from inflammation and swelling of the nerve in the bony canal after viral infection.[63] Facial nerve injuries can be

associated with temporal bone fractures or other forms of trauma[64] or tumors either in the facial nerve[65] or in nearby structures.[66] In most patients, particularly with Bell palsy, the injury is predominantly due to demyelination, and good recovery ensues. Overall, in approximately 70% of patients normal function is obtained; sequelae in a study of 2,500 subjects were reported to be slight in 12% of patients, mild in 13%, and severe in 4%[67]; contracture and associated movements were found in 17% and 16% of patients, respectively. It is estimated that in approximately 20% of patients with Bell palsy, a significant percentage of axons undergo Wallerian degeneration.[30] Some patients with axon loss will still have a good recovery, but others will have a long-lasting flaccid paralysis. There will also be others who have abnormal branching of regenerating axons leading to clinically evident hemifacial synkinesis.[68] This synkinesis is often more debilitating than the weakness and represents a significant cause of morbidity. Synkinesis is believed to result from disorganized regeneration and is analogous to "miswiring," whereby the injured axons will supply different muscles than their original targets. These misdirected axons do not produce functional recovery and can even result in more problems.

With respect to EDX estimation of prognosis, it has been demonstrated that patients with CMAP amplitudes $\geq$30% the opposite side have an excellent outcome, those with 10 to 30% amplitudes have good but not always complete recovery, and those with <10% amplitude have a poor outcome.[69] This finding has often been used by otolaryngologists to determine the need for surgical intervention. More recent studies have found different cut-offs to be useful. In a study of 120 subjects, Mancini and colleagues reported that amplitudes <23% of the unaffected sided were the strongest predictor of nonrecovery of normal function.[70] Using receiver operating curve analysis, the strongest predictive values of amplitude compared with the contralateral side were 33% (67% sensitivity, 81% specificity) and 45% (100% sensitivity, 68% specificity) at baseline and second evaluations, respectively; amplitudes below these values were associated with poor prognosis. In addition, younger age and needle EMG evidence of denervation also contributed to a stronger prediction of synkinesis.[3]

Timing of EDX assessment plays an important role in evaluation of facial nerve lesions. Especially in the setting of temporal bone fracture, the timing of changes seen on facial motor nerve conduction studies impacts the prognosis of facial nerve injuries. Studying the nerve too early, before occurrence of Wallerian degeneration (i.e., before day 7), will lead to a false conclusion that more axons are viable than there really are. On the other hand, waiting too long could result in missed opportunities for early surgical intervention. Generally, studies between days 7 and 14 after onset are preferable.[71]

In terms of outcome after temporal bone fracture, those with less severe injuries have good spontaneous return of function, including those with <95% decline in the CMAP in the first 14 days.[72–74] Poorer outcomes have been noted for patients with >95% degeneration in CMAP within 14 days of injury, and these patients are routinely offered surgical decompression. There is also some recent evidence that blink reflex studies can help predict prognosis in both the acute and subacute phases.[75]

BRACHIAL PLEXUS

Brachial plexus injuries occur in roughly 2–3% of all admissions to level I trauma hospitals.[32] Most commonly, they result from car or motorcycle crashes, or motor vehicle versus pedestrian accidents.[76] A key prognostic element in evaluation of brachial plexus injuries is determination of whether there has been avulsion of 1 or more roots from the spinal cord. This is critical because nerve root avulsions do not recover, and performing plexus surgery distal to a root avulsion is generally pointless.[15,77] The 2 primary ways to evaluate for root avulsion are by studying SNAPs and paraspinal muscles. When SNAPs are present in the presence of severe limb denervation, this suggests that the injury may be proximal to the dorsal root ganglion, i.e., root avulsion. When SNAPs are absent or markedly reduced, this indicates a more distal lesion, but it does not necessarily indicate that the root is intact, because root avulsion and plexus injuries can co-exist as a result of severe trauma. The presence of abnormalities in the paraspinal muscles also suggests root avulsion, or at least that the injury has occurred proximal to the division of the posterior primary ramus from the spinal nerve root. Somatosensory evoked potentials may also be helpful in distinguishing proximal plexus injuries from root avulsions; in the former, the Erb point potentials are absent, but in the latter they are present, while more rostral potentials are absent in both settings.[78]

The prognosis of brachial plexus injuries is also affected by the site of injury. Kim et al.[79] studied the outcomes of 1019 surgically managed brachial plexus lesions. Outcomes were best when injuries involved the upper and middle trunks, primarily supplied by the C5, C6, and C7 roots, or the lateral or posterior cords, but they were poor for C8 or T1 root injuries, lower trunk lesions, or medial cord involvement. Complete injuries of the plexus have a very poor prognosis.[80] It is possible that a reason for

a relatively good prognosis in upper and middle trunk injuries is that these segments of the plexus innervate proximal muscles with relatively short distances for axonal regrowth. Moreover, these muscles supply robust proximal arm motions only, in contrast to the fine hand movements that are generally related to lower and middle trunk innervation.

SUMMARY

Providing a prognosis for patients with focal peripheral nerve injuries is an important component of the EDX medical consultation. There are a variety of EDX measures that may provide valuable prognostic information. While there are some generally useful principles to apply to most nerve lesions, the interpretation should be individualized for each nerve and clinical context. Future research should focus on prospective studies to provide additional insight into the factors that enhance the accuracy of outcome prediction.

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