1 The Basics of Electrodiagnostic Testing Carpal Tunnel and Cubital Tunnel Syndrome

George A. Small

Though carpal tunnel syndrome and cubital tunnel syndrome are the thrusts of this chapter, it is worth reviewing the basic anatomy of nerve to understand the types of pathology that give way to these syndromes, and determine prognosis for recovery. Endoneurium, perineurium and epineurium surround the axons of somatic peripheral motor and sensory nerves (Figs. 1.1 and 1.2). Endoneurium is the direct supporting structure around individual axons. Perineurium forms a looser tissue barrier over bundles of nerve fibers. Epineurium, the outermost surrounding nerve structure, envelops multiple nerve bundles itself. Diffusion of chemical constituents can occur across perineurium more readily than across endoneurium.

The spinal nerve roots can merge with the outermost supporting structure of peripheral nerves, but the lack of endoneurial collagen at the nerve root level can explain why some disease processes selectively involve the root. in some disease states, contributing to cubital tunnel syndrome and carpal tunnel syndrome. This is quite important, as chemical processes that affect the nerve roots via chemical diffusion from the cerebrospinal fluid through which the roots initially traverse will generally not occur directly across named peripheral nerves more caudally, where the 'blood nerve barrier' resists many extracellular tissue constituents.

Nerve trunks contain myelinated and unmyelinated fibers. In myelinated fibers, individual Schwann cells envelop their thick, fatty-rich cell membranes around axons to form the myelin sheath. The nodes of Ranvier, located between adjacent Schwann cells, represent gaps along the myelinated fibers on myelinated axons. This structure facilitates myelinated nerves conducting action potentials with high velocity. Many cases of cubital tunnel syndrome and carpal tunnel syndrome occur in patients with diffuse demyelinating processes that render the anatomical distinction between unmyelinated and myelinated fibers important. Both cubital tunnel syndrome and carpal tunnel syndrome are much more prevalent in patients with primary diffuse disorders of axons or myelin themselves, such as those who suffer from severe kidney failure, uncontrolled diabetes, or chronic alcoholism and inherited myelin disorders such as the family of Charcot-Marie-Tooth (CMT) diseases. Such individuals are much more likely to suffer from cubital or carpal tunnel syndrome, particularly with any extra traumatic provocation.

If myelin is damaged alone, it can readily regenerate (neuropraxia). If axon continuity is transgressed completely (axontmesis); however, reinnervation of muscle served by nerve is less reliably predicted- and therefore the focal

G. A. Small (✉)
Department of Neurology, Allegheny General Hospital, Pittsburgh, PA, USA
e-mail: George.Small@ahn.org; gsmall@wpahs.org

D. G. Sotereanos, L. K. Papatheodorou (eds.), *Compressive Neuropathies of the Upper Extremity*,
https://doi.org/10.1007/978-3-030-37289-7_1

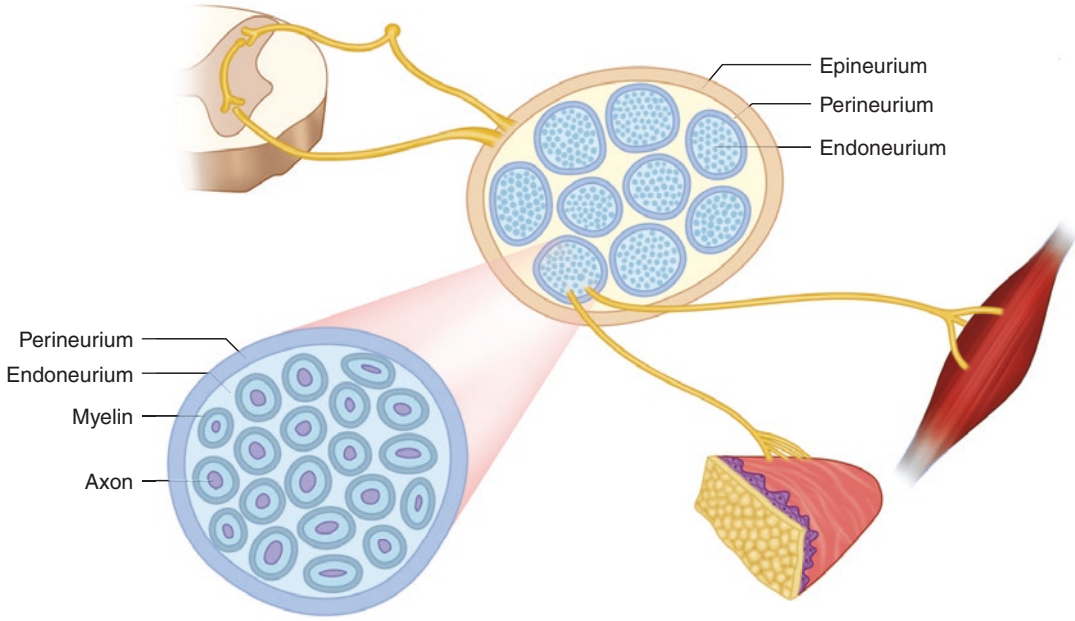

Fig. 1.1 Schematic of basic anatomy of the nerve

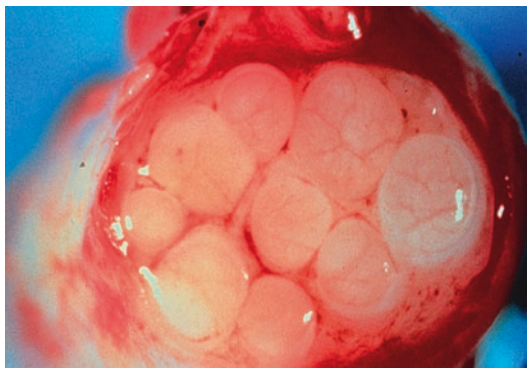

Fig. 1.2 View of basic anatomy of the nerve

demyelination which occurs with traumatic entrapment of nerve which does not involve the axon but does compress myelin has a much better prognosis. This reversible type of pathology causes 'conduction block' of nerve action potentials. This is true of any entrapment syndrome in any nerve throughout the body, and is especially true of cubital tunnel syndrome and carpal tunnel syndrome.

The carpal tunnel, through which the median nerve passes at its distal extent, is surrounded by tendons, bones and connective tissue. The dorsal surface is bounded by the tendons of the muscles to the extensor digitorum communis, extensor indicis proprius, extensor pollicis longus and extensor carpi radialis brevis muscles. On the ventral, or palmar boundary is the abductor pollicis longus muscle (APL) laterally, and the transverse carpal ligament, and muscles to the little finger. Within the carpal tunnel, which is bounded itself by a sheath of connective tissue is the median nerve itself and the tendons of the flexor pollicis longus, flexor digitorum profundus, as well as flexor digitorum sublimis muscles. The bones on the dorsum of the hand surrounding the carpal tunnel include the hamate, capitate, trapezium and trapezoid. This entire area, subject to a lifetime of chronic flexion and extension as well as crowding of tendons and connective tissue is the site of median nerve compression resulting in the carpal tunnel syndrome.

The median nerve arises from the lateral and medial cords of the brachial plexus. It is a mixed nerve (meaning it contains motor and sensory fibers). These fibers are derived from the C6, C7, C8, and T1 roots. It supplies muscles of the thenar eminence of the hand, but also cutaneous sensory fibers of the skin over the lateral aspect

of the thenar eminence, and the index and middle fingers, as well as the tips of the terminal phalanges. It also serves sensation on the medial half of the ring finger. The sensory fibers of the middle finger are derived from the C7 nerve root, the lateral cord of the brachial plexus, as well as the middle trunk. The surface of the index finger receives sensory fibers from the C6 and C7 nerve roots through the lateral cord and upper and middle trunk of the brachial plexus. There is much variance to the sensory innervation of the fingers mentioned. There are no muscles in the proximal arm innervated by the median nerve proper. It traverses the arm alongside the humeral bone and enters the forearm between the heads of the pronator teres muscle- which it supplies with motor fibers along with the flexor carpi radialis, palmaris longus and flexor digitorum superficialis muscles. It gives rise to a pure motor branch, the anterior interosseous nerve, which innervates the flexor pollicis longus, pronator quadratus, and flexor digitorum profundus muscles to digits one and two. The remainder of the median nerve descends in the forearm and goes through the carpal tunnel along with the tendons previously mentioned. It first branches off a recurrent thenar nerve, and then branches to the lumbrical 1 and 2 muscles of the hand. The recurrent thenar nerve innervates the abductor pollicis brevis and the lateral half of the flexor pollicis brevis and opponens pollicis muscles. Generally, although carpal tunnel syndrome results in sensory abnormalities, the symptoms do not include such abnormalities of the lateral portion of the thenar eminence, as the sensory branch to that area does not go through the carpal tunnel. This is why anesthesia of the lateral portion of the thenar eminence, along with other sensory symptoms, may suggest a more proximal process, either in the median nerve or at the plexus or root levels, or even the brain or spinal cord. Other entrapment syndromes of the proximal median nerve at the antecubital fossa and in the forearm, resulting in an almost pure motor anterior interosseous nerve syndrome exist, but are not the subject of this chapter.

As in all medical issues, history is paramount. Carpal tunnel syndrome usually presents with nocturnal paresthesias in any combination of the lateral 3 digits of either hand, more commonly the dominant one [1]. Causes of the nocturnal predominance is not clear, but may relate to the patient being more aware of the symptoms when the distractions of the day are at a minimum. Occurring frequently upon awakening in the morning and although the patient may insist he does not sleep on the arm in question- ALL people twist and turn at night and may compress the forearm, upper arm, or wrist during sleep, thus increasing the risk of compressing the median nerve in the narrow carpal tunnel. Weakness alone is rarely the presenting symptom of carpal tunnel syndrome, as sensory nerve fibers are more prone to chemical and physical injury than motor nerves. Many an amyotrophic lateral sclerosis (ALS) victim has been referred to the electromyographer's attention with a wasted, atrophic hand, without sensory symptoms. Carpal tunnel syndrome occurs in the dominant hand in the vast majority of cases and electrophysiologically can found to be bilateral in 15% of the time, even if the side not in question is asymptomatic. This is why bilateral electrophysiologic testing for carpal tunnel syndrome is so important, both to delineate the severity of the process on the side which is the subject of the patient's complaint, and as a baseline for further potential therapy in the less, or non-symptomatic side.

As alluded to above, history of glucose intolerance, uremia, hypothyroidism and hand or wrist trauma is of paramount importance to know. Some patients suffer from carpal tunnel syndrome, denying a history of diabetes, are found on examination to have it by their surgeon or neurologist, even prior to their PCP or endocrinologist making the diagnosis. Diabetes is the most common accompanying medical problem occurring when carpal tunnel syndrome is diagnosed. Diabetes causes both hypoxemic damage to nerves in the body as well as direct hyperglycemic damage to both axon and myelin. A glucose tolerance test or hemoglobin A1c should be tested in all patients with carpal tunnel symptoms, as well as tests of thyroid and renal function.

Along with a detailed history of any accompanying medical problems, as well as determining

that the predominant symptoms affect the lateral portion of the hand up to and perhaps including the medial 4th digit, examination of the patient with carpal tunnel syndrome generally begins with inspection. Isolated atrophy of the lateral thenar eminence is a clinical key that the problem is originating in the median nerve at its distal extent at the carpal tunnel. Accompanying forearm atrophy can be seen with more proximal median nerve problems. If indeed there is atrophy of the lateral thenar eminence (abductor pollicis brevis [APB] muscle), then inspection of other C8 and T1 innervated muscles is paramount to rule out a lesion at these nerve root levels or the lower trunk of the brachial plexus.

Weakness of the distal tips of digits 2 and 3 and of the thumb along with weakness of forearm pronation can point to a pure anterior interosseous nerve lesion in the forearm.

EMG/NCV

This test is upwards of 85% sensitive for carpal tunnel syndrome and more than 95% specific for it [2]. A basic understanding of the technique follows.

Motor nerves carry large myelinated fibers which conduct electrical impulses at 40–50 m/s. Sensory nerves carry large, medium, small and unmyelinated nerves which conduct electricity at anywhere from 50 m/s down to 2–3 m/s. Standard EMG nerve conduction study testing evaluates the large, fast conducting fibers of motor and sensory nerves. Electrical studies for entrapment neuropathy involve testing mixed motor and sensory nerves, isolated motor nerves, and isolated sensory nerves. The standard protocol for evaluating a patient with carpal tunnel syndrome is to place a recording electrode over the abductor policies brevis muscle (Fig. 1.3). A reference electrode is placed at a local, bony, non-electrically active reference area. A separate ground wire is applied to the skin for electrical safety. The median nerve is stimulated in the antecubital fossa and at the wrist with a handheld stimulation device. If one were to measure the distance between the stimulating and the recording electrode on the APB muscle, and divide that distance by the time it takes an electrical impulse generated from the stimulating electrode to the recording electrode, a falsely low velocity for motor nerve conduction would result. This is because one is measuring not only the conduction in nerve fibers, but also across the neuromuscular junction and across the muscle membrane, which conduct at extremely low velocities. Therefore the technique of measuring velocity in motor nerves involves stimulating the nerve at two separate points, recording at one particular point, and subtracting the distance from the distal stimulation point to the recording electrode, from that of the proximal stimulus point to the recording electrode, and dividing that by the difference in time it takes the impulse that is generated from the initial point of stimulation from the distal point of stimulation. This results in deriving a conduction velocity along

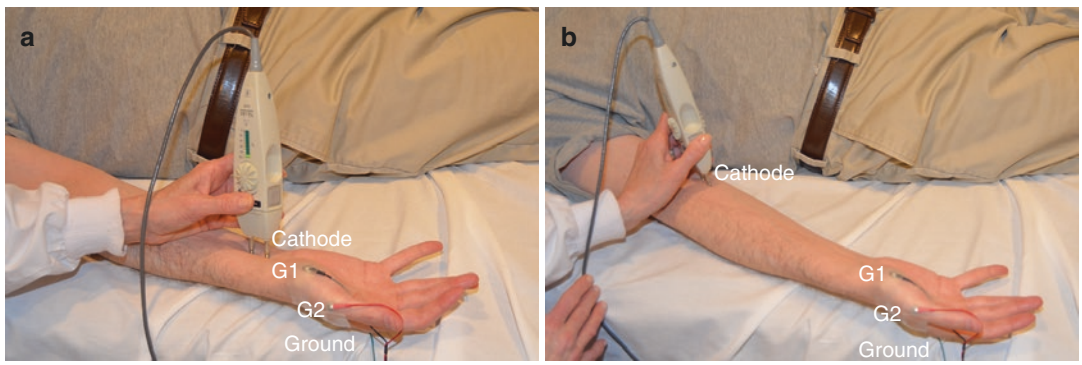

Fig. 1.3 Median motor study. (**a**) Distal stimulation site over median nerve at wrist, recording abductor pollicis brevis. (**b**) Proximal stimulation site at antecubital fossa

the forearm segment of the median nerve itself, subtracting out the time it takes the impulse to go from the distal stimulating point to the recording electrode, which is adulterated by neuromuscular junction and muscle membrane influences. This results in an accurate conduction velocity in the median motor nerve itself. This concept is also applied to stimulating all other motor nerves such as the ulnar nerve and other motor nerves in the body. Sensory nerve conduction velocities are a more simple practice. Since the nerve impulse does not traverse a neuromuscular junction or muscle membrane substance, simply stimulating a sensory nerve at one point, recording at another point, and dividing the distance between the two points by the time it takes the impulse to go from one point to the other results in measuring conduction velocity in the sensory nerve. The examiner must always be attentive to the effects of temperature on conduction velocities, and even on the amplitude of both sensory and motor responses. A surface skin temperature of 30 ° C generally reflects a normal body temperature which reflects the true parenchymal nerve temperature itself. A rule of thumb is that for every degree Celsius below body temperature measured for a nerve, there is a 2 m/s decrease in conduction velocity. Too many studies in general neurological practice reveal low conduction velocities which are due to low surface skin temperature and not to true pathology, frequently rendering a false positive diagnosis of peripheral neuropathy, resulting in inappropriate patient management.

In addition to velocities, the resulting waveform generated on the oscilloscope results in an amplitude measured from the baseline to the electrical peak of a biphasic waveform in units of microvolts, in the case of a motor nerve referred to as a compound motor action potential (CMAP), and in the case of a sensory nerve referred to as a sensory nerve action potential (SNAP). Each of these potentials is the algebraic sum of the number of either the number of sensory nerves recorded or muscle cells depolarized, measured within the nerve bundle itself or the muscle being tested. Any electromyography laboratory must have a reference set of normal values from which clinical test results are compared. It is impractical for each laboratory to determine its own normal values, which historically had been painstakingly measured. Historical normal values for amplitude and velocity in motor and sensory nerves can be applied as long as the EMG/NCV machine is set to the same filter settings, and the same type of electrodes and stimulators are used as were used to derive the original normative textbook values. One cannot overemphasize the importance of controlling skin temperature in providing accurate results, as mentioned above. A paradoxically slow conducting nerve with large amplitude should be a clue to that the test was done with inadequate temperature control. The study may then need to be repeated. Other than amplitude and velocities, the parameters measured in standard nerve conduction studies, include other data points - the “distal latency”, and the “F response”. Historically the F response, (which stands for “foot”), where the measure was first derived, is a method of stimulating motor nerves distally at a very high level of stimulus duration and amperage, and doing so repeatedly with recording over the muscle innervated by the nerve in question, and deriving approximately 10–20 responses which occur because of “back firing” of motor nerves near or in the anterior horn cell. The amplitude of these responses are very low normally, and appear much later on the oscilloscope than the CMAP, however when the latency of these responses is measured, it provides a general overview of the most myelinated nerves conducting along the proximal, middle, and distal segments of the motor nerve, and if very prolonged, can be a clinical sign of severe demyelination proximal to the point of stimulation. For entrapment neuropathies, this particular test has less use than the regular recording of amplitude, velocity and distal latency. Distal latency refers to the time it takes an electrical impulse from normal stimulation of a motor nerve at the point closest to its recording electrode to reach its innervated muscle. This value is usually on the order of less than 10 ms, and there are normative values for each motor nerve tested. Prolongation of distal latencies suggests distal demyelinating pathology in a nerve.

For sensory nerve action potentials, merely the recording of the amplitude of the SNAP and the velocity are adequate for the clinical report to the referring physician. Thus, an electrical study for any entrapment neuropathy or even for peripheral neuropathy, myopathy radiculopathy, or plexopathy should contain a data sheet which lists the amplitude and velocity of any sensory nerve tested as well as the amplitude, distal latency, F response latency and conduction velocity along any motor or sensory nerve measured.

The most sensitive indicator electrically of carpal says tunnel syndrome is a slowing in the conduction velocity and or decreased amplitude of the sensory nerve action potential of the median sensory branch to digits 1 or 2 [3]. Electrodes are placed at the distal end of digit two as well as at the wrist. Stimulation of the sensory nerve can be accomplished either at the finger, with recording at the wrist, or at the wrist with recording at the finger. Stimulating the nerve in its physiologically "correct" or orthodromic" direction or stimulating the mixed median nerve at the wrist with recording at the finger "antidromic" direction will result in a waveform representing the sensory nerves to the finger tested. The antidromic method may result in muscle artifact; however, it is a more sensitive method for recording sensory nerve action potentials. Either method is reasonable in practice as long as artifact is controlled for. The next most sensitive indicator of carpal tunnel syndrome is a prolonged distal latency when measuring the motor conduction as described above. Frequently, electrical studies will reveal some combination of sensory nerve slowing to digit 2 and a prolongation of the median motor distal latency. The normal median motor distal latency is <4.5 ms. The normal median sensory conduction is 50 m/s. Generally no median motor nerve conduction slowing is noted in the forearm in carpal tunnel syndrome, but it may occur as an artifact of an extremely prolonged distal latency (greater than 10 ms) as retrograde demyelination can occur from severe damage to the nerve within the carpal tunnel. In this case the sensory nerve action potential to digit 2 is usually found to be absent. In cases of extremely mild carpal tunnel syndrome the entire electrical study is normal- however, with the patient reporting typical carpal tunnel syndrome symptoms a more sensitive test that can be performed is to stimulate the median mixed motor and sensory nerve directly in the palm and recording over the median nerve at the wrist and determining the conduction velocity in that short segment of this median mixed motor/sensory nerve. If it is decreased, and all other data are normal, there is clear evidence of carpal tunnel syndrome. This portion of the study should be performed when the patient's symptoms are typical for carpal tunnel syndrome as mentioned, and the remainder of the test is normal. As part of a general electrical study for carpal tunnel syndrome, the ulnar motor nerve and ulnar sensory nerves should also be evaluated. When a generalized neuropathic process is not present, a normal ulnar motor and sensory nerve set of values serves as a nice control when median neuropathy at the wrist is electrically noted. This provides more confidence for the electromyographer to present to the surgeon of the lesion truly being at the carpal tunnel and not being part of a more generalized peripheral neuropathic process not requiring surgical intervention.

After the median and ulnar motor and sensory responses are recorded, needle EMG is then performed.

EMG or electromyography, is the process of inserting a needle electrode in a muscle and recording electrical impulses on an oscilloscope either at rest or while directing the patient to perform a muscle contraction. Normal muscle generally does not reveal unusual electrical potentials at rest. With activation of any muscle tested a number of motor units can be assessed to that muscle for any variance from normality. Motor unit action potentials (MUAPs), are representative of the algebraic sum of individual muscle cell action potentials representing the sum of all voltage changes from all muscle cells innervated by the nerve activated. No externally applied electrical shocks are required. The duration, amplitude and number of phases of the visualized potentials can determine whether a process resulting in weakness or spasm is on a neurogenic or a myogenic basis.

Individual muscles are innervated by a number of different motor nerves. The motor unit is defined as a motor nerve and all the muscle cells it innervates. If there is damage to a particular nerve, other nerves innervating other muscle cells within the muscle itself eventually will take over innervation of those muscle cells. The resulting motor unit action potential generated by a voluntary contraction will then appear larger than normal on the electromyography oscilloscope, since any individual surviving motor nerve will innervate more muscle cells than it usually does. Therefore, a chronic neurogenic process can be defined as an increased amplitude in motor unit potentials seen. In addition, the surviving motor neurons fire at a frequency which is faster than usual in order to maintain a specific force since other motor nerves are damaged and cannot do so. This denervation and reinnervation process is time-dependent, and a nerve conduction study performed immediately after nerve injury will not reflect this. Reinnervation of muscle cells which have been "abandoned" by their damaged motor nerves occurs approximately after 2 weeks for arm innervated muscles and 3 weeks for leg innervated muscles and 1 week for facial innervated muscles. This reflects the time it takes for reinnervation to occur as the axons of the reinnervating nerves must grow to innervate the muscle cells which have been denervated. Reinnervation may not be complete process. After mechanical trauma to the carpal tunnel, when weakness ensues, one may perform a baseline nerve conduction study near the time of trauma, however it is more important to perform a study more than 2 weeks after the trauma to determine what reinnervation has occurred. In addition, proper localization of the process to the carpal tunnel can only occur after such reinnervation occurs. In moderate to severe cases of carpal tunnel syndrome, the APB muscle will show changes of acute and/or chronic neurogenic change, such as increased amplitudes of the motor unit action potentials (MUAPs), and if the process is subacute to acute, spontaneous electrical discharges from denervated APB muscle cells will occur and are easily noted with the patient's muscle at rest (fibrillations and positive sharp waves) and the EMG electrode inserted into the APB muscle. All these changes require at least 2 week's delay in performing the electrical test after any particular acute inciting event thought to cause carpal tunnel syndrome has occurred. If the patient has had carpal tunnel symptoms for weeks, months, or years- then certainly the electrical study can be accomplished at any time.

Ulnar neuropathy most commonly occurs at the ulnar sulcus or at the cubital tunnel [4, 5]. When occurring at the ulnar sulcus historically the problem was referred to as the "tardy ulnar palsy", as it frequently was observed with some delay after some form of direct inciting trauma to the area had occurred [6]. Constant flexion and extension of the elbow is a clear risk factor for this process as well as the above-mentioned generalized peripheral neuropathic risk factors, distal humerus fracture, and olecranon fracture. The cubital tunnel is a virtual structure in the forearm through which the ulnar nerve passes after traversing the ulnar sulcus, piercing the aponeurosis between the two main heads of the flexor carpi ulnaris muscle. Compression of the nerve by these portions of the flexor carpi ulnaris muscle can result in the process referred to as the cubital tunnel syndrome. Although worth mentioning, but not the subject of this particular chapter, ulnar neuropathy may also occur at Guyon's canal at the wrist where the distal ulnar nerve enters the hand. Each of these processes is distinguishable, if not by clinical examination, then by nerve conduction study and electromyography.

The ulnar nerve is derived from the C8, T1 nerve roots and the lower trunk of the brachial plexus. The motor axons of the ulnar nerve to the flexor carpi ulnaris muscle generally branch from the main ulnar nerve trunk proximal to the cubital tunnel, and therefore in cases of cubital tunnel syndrome, examination via EMG of the flexor carpi ulnaris muscle is normal. Branches of the ulnar nerve to the flexor digitorum profundus muscles to digits four and five in the hand are generally not spared in cubital tunnel syndrome, and the finding of typical ulnar neuropathic symptoms with preserved flexor carpi ulnaris

function via EMG and abnormal EMG examination of the FDP muscle and other distal muscles via EMG can help localize the process.

Stimulation of the ulnar nerve in the electromyography laboratory with recording over either the 1st dorsal interosseous muscle or the abductor digiti minimi muscle of the hand, generally is carried out recording at 3 points, above the elbow, below the elbow, and at the wrist. As described for the median nerve, the segmental velocities of the ulnar nerve in the above-elbow to below-elbow segment, and the below-elbow segment to wrist are derived via the method mentioned above to filter out the effects of muscle membrane and neuromuscular junction slowing, which adulterate calculated nerve velocity values. Both the above- elbow to below- elbow segment and the below- elbow to wrist segment should conduct at 50 m/s. When performing ulnar motor nerve conduction studies (Fig. 1.4) the forearm should be bent at a 45° angle, which more closely approximates its normal anatomic course. Performing ulnar motor nerve conduction studies with the elbow joint at 180° can result in "telescoping" of the nerve and its myelin upon itself, resulting in a falsely low conduction velocity being recorded. The ulnar nerve, in addition to innervating the flexor carpi ulnaris and flexor digitorum profundus muscles, innervates the abductor digiti minimi as well as a portion of the flexor pollicis brevis muscles and all the interosseous muscles of the hand. The sensory nerve action potential of the ulnar nerve is generally derived by either orthodromic stimulation of the digit 5 sensory nerve with recording over the ulnar nerve at the wrist, or antidromically as described for the median sensory nerve.

Ulnar neuropathy at the ulnar sulcus is readily demonstrated by revealing slowing in the ulnar motor nerve segment in the above- elbow to below- elbow segment, with relative preservation of the conduction velocity in the motor ulnar nerve in the below- elbow segment to wrist. In addition there is usually abnormality of the ulnar sensory nerve action potential from digit 5 noted. Usually an absent sensory

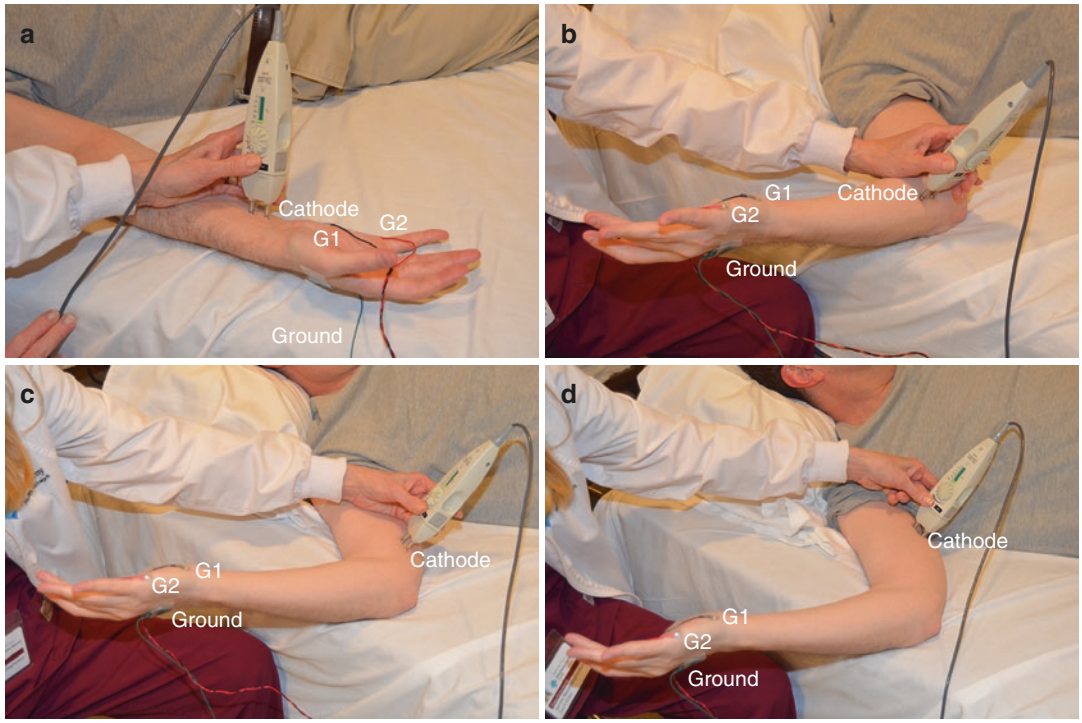

Fig. 1.4 Ulnar motor study. (**a**) Distal stimulation site over ulnar nerve at wrist. (**b**) Proximal stimulation site below elbow. (**c**) Proximal stimulation site above elbow. (**d**) Proximal stimulation site under upper arm

potential or low amplitude sensory potential accompanies ulnar neuropathy at the ulnar sulcus. In cubital tunnel syndrome, slowing in the ulnar motor nerve may not be readily perceptible. If ulnar neuropathy in general is suspected, then in addition to stimulating the ulnar motor nerve at the above elbow, below elbow and wrist segments, the ulnar nerve is serially stimulated at 1 cm intervals along the below-elbow to wrist segment in a separate graphical array available on any standard EMG nerve conduction study machine along perhaps 7–8 points. The conduction velocity along any of these 1 cm points can readily be derived, and a precise localization of pathology to a particular area in the below-segment to wrist may be identified, thus localizing in the process to the cubital tunnel. In addition, the ulnar sensory nerve action potential would be abnormal, and needle EMG of the flexor carpi ulnaris muscle (FCU) would be normal, with abnormalities seen in the needle examination via EMG of the flexor digitorum profundus muscle to digits 4 and 5, the abductor digiti minimi (ADM) muscle and the first dorsal interosseous (FDI) muscle. Thus, a combination of electromyography and ulnar nerve conduction studies, both motor and sensory, can be utilized to more precisely localize an ulnar nerve process.

One complicating factor in the electrophysiologic examination of patients with entrapment neuropathies is that there are fairly common variations in nerve innervation of voluntary muscle, particularly median and ulnar innervated muscles, by anomalous median to ulnar nerve connections in the forearm. In a study of cadavers, the anatomist Martin found anastomoses in 15% of patients, and another anatomist Gruber did as well. The former in 1763 and the latter in 1870. These common anastomoses of median to ulnar nerves, complicating, at times proper diagnosis of ulnar neuropathy and median neuropathy, have become to be known as the Martin-Gruber anastomoses. There are three main types. Upwards of 15–20% of patients with carpal tunnel syndrome may have this anastomosis, and approximately 20% of normal controls do as well. The anastomoses involve axons leaving the main trunk of the median nerve or anterior interosseous nerve and crossing through the forearm to join the main trunk of the ulnar nerve, ultimately innervating intrinsic hand muscles. Although a significant number of axons may participate in the anastomosis, all of the axons of the median nerve are not involved. The most common anastomosis is a median-to-ulnar anastomosis with innervation eventually of the 1st dorsal interosseous muscle. This is the type 2 Martin-Gruber anastomosis. It can be inherited as an autosomal dominant trait. In routine nerve conduction studies this most common Martin-Gruber anastomosis may not be found because the ADM muscle is usually the routine site of ulnar nerve recording. The type 1 Martin-Gruber anastomosis exists as the median crossing fibers to the ulnar nerve terminating in the abductor digiti minimi muscle. This can be noted on routine nerve conduction studies as a low ulnar CMAP amplitude with above-elbow stimulation and a larger CMAP with below-elbow and wrist stimulation, simulating an ulnar neuropathy at the ulnar sulcus. When this is noted on a routine nerve conduction study, the electromyographer should simply, as a general check for the crossover, stimulate the median nerve at the elbow and record over the abductor digiti minimi muscle. Without the anastomosis, there should be no CMAP found. If the anastomosis exists to the ADM muscle, then a small CMAP would be seen. This simple test can avoid over diagnosing ulnar neuropathy at the elbow potentially avoiding unnecessary surgery. The most common type of Martin-Gruber anastomosis of median nerve fibers crossing to the ulnar nerve, innervating the FDI muscle, will not present on routine nerve conduction studies. If one were to test specifically for a deep ulnar branch neuropathy which characteristically causes interosseous wasting and pain from palmar compression (i.e. often in cyclists from handlebar pressure) the recording electrode is purposely placed on the FDI muscle, this type of Martin-Gruber anastomosis will reveal itself as a larger CMAP from the FDI upon wrist stimulation than upon elbow stimulation. The least common type of Martin Gruber anastomosis (Type 3) may manifest on routine median motor conduction studies with the recording

electrode on the APB muscle. It should be suspected when the CMAP of the thenar muscle is unusually larger after elbow stimulation than with wrist stimulation of the median nerve. In addition, if both carpal tunnel syndrome and this type of anastomosis co-exist, then not only will the median nerve stimulation at the elbow evoke a large CMAP with an unusual deflection pattern, but there would be and erroneously normal motor latency from the median nerve at the elbow with a prolongation of the distal motor latency and a factitiously high velocity noted in the forearm segment of the median nerve. The details of these anomalies can be left to the electromyographer, as well as the design of the study when such an anastomosis is present. These studies should be clearly explained in the summary portion of the electromyography report, not only for accurate documentation, but to avoid leading the surgeon down the path of over-diagnosing ulnar neuropathy at the ulnar sulcus and underdiagnosing median neuropathy at the wrist when the type 3 anastomosis occurs.

Conclusion

Electromyography and nerve conduction studies supplement the patient history, physical and neurological examination, and radiographic data to help the examining surgeon properly localize nerve entrapment processes, particularly when upper extremity median and ulnar nerve entrapments are suspected. These electrophysiological tests serve the patient and surgeon as well as to the correct place and timing of surgery in the management of these highly prevalent, uncomfortable, and often disabling conditions.

References

1. Padua L, Coraci D, Erra C, et al. Carpal tunnel syndrome: clinical features, diagnosis, and management. Lancet Neurol. 2016;15(12):1273–84.
2. Jablecki CK, Andary MT, Floeter MK, Miller RG, Quarterly CA, Vennix MJ, et al. Practice parameter: electrodiagnostic studies in carpal tunnel syndrome. Neurology. 2002;58:1589–92.
3. Buchtal F, Rosenfalck A, Trojaborg W. Electrophysiological findings in entrapment neuropathy of the median nerve at the wrist and elbow. J Neurol Neurosurg Psychiatry. 1974;37:340–63.
4. Brown WF, Yates SK. Cubital tunnel syndrome and ulnar neuropathy. Ann Neurol. 1980;7:289–90.
5. Ulnar neuropathy clinical presentation: history, physical examination, ulnar neuropathy at elbow. emedicine.medscape.com. 2016.
6. James GR, Grafton L. Diagnosis and treatment of tardy paralysis of the ulnar nerve: based on a study of 100 cases. J Bone Joint Surg. 1947;29:1087–97.