MARTIN-GRUBER ANASTOMOSIS AND CARPAL TUNNEL PHYSIOLOGIC IMPLICATIONS AND PITFALLS

Daniel Dumitru, M.D., Ph.D.

INTRODUCTION

Focal compressive median nerve sensory and motor neuropathies about the wrist comprise the typical pathophysiologic basis for the clinical term carpal tunnel syndrome (CTS). In this particular discussion we are primarily concerned with the electrophysiologic aspects of CTS when manifesting in patients who also happen to have a Martin-Gruber Anastomosis (MGA). There are a number of interesting and insightful presentations of this combination. A detailed appreciation of the various electrophysiologic manifestations of CTS with the MGA provide the practitioner with a wealth of insight into median nerve anatomy and pathophysiology.

We will further explore a number of additional electrophysiologic presentations of CTS without an MGA that nevertheless may be challenging to beginning practitioners. Although CTS is the most frequently encountered focal neuropathy in clinical practice, and considered rather straightforward from an electrodiagnostic medicine perspective, this is not always the case. As with the MGA, critically evaluating the various manifestations of particularly motor CTS, can provide us with considerable insight into the pathophysiologic mechanisms of the peripheral nervous system that extends beyond CTS.

MARTIN-GRUBER ANASTOMOSIS

Anatomy. It is generally accepted that all of the hand intrinsic muscles are innervated by the ulnar nerve with the exception of five muscles innervated by the median nerve: abductor pollicis brevis (APB), opponens pollicis (OP), superficial head of the flexor pollicis brevis (FPB), and the first two lumbrical muscles. One of the more “common” anomalous innervation patterns affecting the hand, and is the subject of this discussion, is referred to as the Martin-Gruber anastomosis.

There are a number of misconceptions regarding this anomaly, but in reality the innervation pattern is rather straightforward. The Martin-Gruber anastomosis is comprised of a communicating neural branch most commonly, though not always, between the anterior interosseous nerve (AIN) and ulnar nerve typically in the mid-forearm region (Fig. 1). This communicating branch conveys efferent motor fibers originating from the C8 and T1 nerve root levels as well as muscle afferents from the innervated musculature. Once these C8 and T1 fibers cross over to the ulnar nerve, they are now fully
realized ulnar fibers. That is, they innervate only the routine ulnar hand muscles. Specifically, they do not innervate the above noted five median innervated hand muscles. In short, the fibers comprising the MGA may be thought of as ulnar fibers that for whatever reason initiated with the median nerve and then crossed over to the ulnar nerve. There is a suggestion that very rarely a few cutaneous ulnar sensory fibers may also traverse this pathway, but it is almost exclusively a motor efferent and muscle afferent pathway with no cutaneous sensory distribution.

The MGA is known to be inherited in an autosomal dominant pattern with somewhere between 10-25% of the population possessing this anomaly. If a person has this anomaly, they are likely to have it bilaterally as high as about 70% of the time. The exact distribution and total number of these ulnar cross-over fibers to specific ulnar innervated muscles in any one person is truly unknown and quite variable. We will return to this important point again when the electrophysiologic consequences of the Martin-Gruber anomaly is discussed in detail. At this point, it is important to clear up a misconception that one may read in some textbooks. It is commonly stated that the ulnar destined MGA cross-over fibers innervate the muscles of the thenar eminence. This is a deceptively misleading statement at best. What is meant, but not specifically stated, is that these so called thenar eminence muscles are in fact the ulnar and not median innervated thenar eminence muscles. In other words, the noted thenar eminence musculature are in fact the adductor pollicis (AP), deep head of the flexor pollicis brevis, and possibly the first dorsal interosseous muscle which may contribute some bulk to the thenar eminence and NOT the APB, OP, or superficial head of the FPB muscles. It bears repeating that the fibers passing through the MGA innervate only typically ulnar innervated hand muscles and have absolutely nothing to do with the usually median innervated hand intrinsic musculature.

**Clinical Implications.** A number of relevant clinical implications of the MGA are important to practitioners of electrodiagnostic medicine with respect to lesion location and clinical hand function. Recall that in the MGA there is an anatomic bridge of C8/T1 primarily motor efferent/afferent fibers crossing over from the median to ulnar nerves at approximately the
mid-/forearm level. One can anticipate a number of clinical scenarios based upon various lesion locations of the median and ulnar nerves.

In a patient with an MGA and a complete lesion of the ulnar nerve in or about the post-condylar groove, a resultant clinical picture can be anticipated. Since the motor innervation to the ulnar innervated forearm muscles (medial two digits' flexor digitorum profundus and flexor carpi ulnaris muscles) will be dysfunctional, fourth and fifth finger flexion will be weak. However, the typical "ulnar claw" hand will likely not be overtly manifest. This is because some degree of innervation of the ulnar innervated hand intrinsic muscles will have preserved function with little tone in the FDP to the 4th and 5th digits to cause distal phalanx flexion, i.e. clawing. In particular, the interossei will maintain some degree of metacarpal and interphalangeal stability to resist the pull of the extrinsic finger extensors thereby avoiding an overt clawing of the medial two digits and abduction of the fifth digit. This is not an indication of an all "median" hand, but rather some degree of sparing of hand intrinsic function. Of course, the degree to which the hand avoids a claw deformity is directly dependent upon how many hand intrinsic neural fibers participate in the MGA and subsequently which muscles are innervated. One cannot assume that there are a large number of cross-over fibers, nor can one assume that all of the ulnar innervated muscles are innervated to the same degree by the cross-over fibers. A needle EMG will likely reveal denervation in all ulnar innervated muscles, no motor units in the ulnar forearm musculature, but motor units with variable recruitment in the ulnar innervated hand intrinsic muscles. The preservation of motor units with denervation potentials in the hand intrinsic muscles is a result of a mixture of nerve fibers traveling through both the main ulnar trunk and the MGA. An absent ulnar sensory nerve action potential (SNAP) should be observed with an absent compound muscle action potential (CMAP) from the abductor digiti minimi (ADM) with above/below elbow stimulation. However, a variable amplitude CMAP may result from wrist stimulation and depends upon the total number of crossover fibers through the MGA innervating the ADM.

If an individual with a MGA sustains a complete laceration of the ulnar nerve at the wrist, then all of the ulnar innervated hand intrinsic muscles are completely denervated. In this instance, the patient will lose all function of the ulnar hand intrinsic muscles and display an ulnar claw hand deformity. Obviously, the ulnar SNAP and CMAP are absent with denervation detected in the ulnar hand intrinsic but not extrinsic musculature. These findings are a result of the MGA having no effect on hand intrinsic muscle preservation since the lesion at the wrist is distal to the MGA cross-over location.

On the other hand, if a complete axonal loss lesion of the median nerve at the antecubital fossa is sustained, typical clinical but unexpected EMG findings are noted. From a clinical perspective, the patient will clearly have dysfunction of the usually...
median innervated forearm and hand muscles. However, performing a needle EMG of the ulnar innervated hand intrinsic muscles most likely will reveal denervation with preserved motor units. This finding is a result of some of the MGA fibers participating in most of the hand intrinsic innervation. That is, there is usually some MGA as well as non-MGA ulnar nerve fibers providing innervation to most if not all of the typically ulnar innervated hand intrinsic muscles, but to a variable degree. In some sense, patients with an MGA have hand intrinsic muscles that are “dually” innervated with both MGA and non-MGA ulnar nerve fibers.

It is this author’s experience that most patients with an MGA have the above noted “dual” innervation of all ulnar hand intrinsic muscle fibers through both MGA and non-MGA ulnar nerve fibers. This observation is based on the careful assessment of hand intrinsic muscles in patients with proximal median nerve lesions and an MGA. In my opinion, those “studies” that characterize a specific percentage of MGA fibers contributing to the innervation of various hand intrinsic muscles is more commensurate with fantasy than science. Specifically, if one is performing surface recordings on patients with an MGA and attempting to record from specific ulnar hand intrinsic muscles, volume conducted responses from nearby muscles will contaminate the recorded results. The same situation applies to needle recording electrodes. Some practitioners are under the false assumption that placing an EMG recording electrode (monopolar/concentric) into a muscle guarantees no volume conducted contamination from nearby muscles. This is just not so. As noted above, if you want to clearly document what muscles are innervated by the MGA, then patients with a complete axonal loss lesion to the median nerve at or proximal to the antecubital fossa must be thoroughly assessed.

ELECTRODIAGNOSTIC MEDICINE PRESENTATIONS AND IMPLICATIONS

Introduction. A number of patients with signs/symptoms, and electrodiagnostic medicine sensory abnormalities consistent with carpal tunnel syndrome (CTS) are presented. In other words, all of the presented patient scenarios discussed have focal slowing of the median sensory component across the carpal tunnel region. Therefore, we will explore the various MGA presentations in patients with presumptive CTS and discuss why these electrophysiologic manifestations are present. Specifically, we will only be discussing the various motor findings in CTS with MGA.

Normal Findings. In an individual with no clinical or electrophysiologic sensory findings for CTS, a normal median CMAP with no prolongation of the distal motor latency (DML) is to be expected. Further, the forearm motor nerve conduction velocity (NCV) and CMAP amplitude are also without evidence of slowing or reduced amplitude/temporal dispersion respectively (Fig. 2). It is important to have a clear appreciation for normal median motor parameters so
Figure 2. A normal median CMAP from wrist and antecubital fossa stimulation are shown. The DML is 3.4 ms with a distal amplitude of 7.8 mV while the proximal response is 7.8 ms with an amplitude of 7.0 mV and a forearm NCV of 58 M/s. Note the clearly defined negative take-offs with a slightly reduced proximal CMAP amplitude.

as to immediately recognize when any abnormal median motor presentations arise.

Martin-Gruber Anastomosis Without CTS. It is certainly possible to readily appreciate when an MGA is present even if the patient does not present with, or actually have any median nerve abnormalities about the wrist (Fig. 3). Stimulating the median nerve at the wrist will result in the anticipated normal median motor CMAP parameters of DML and amplitude. Following activation of the median nerve about the antecubital fossa, however, the electrical impulses travel toward the hand along both the main median nerve trunk as well as through the MGA to cross over to the ulnar nerve. Because there is no delay across the carpal tunnel region, the motor fibers through the main branch of the median nerve arrive at the median innervated thenar muscles without delay and result in muscle depolarization with an ensuing negative onset CMAP. The MGA cross-over fibers arrive at the various ulnar innervated muscles located beneath the median innervated thenar muscles somewhat later. This “somewhat later” is a subtle but critical difference in action potential arrival times. The MGA cross-over fibers must traverse a longer pathway (cross over fibers plus deep branch of the ulnar nerve across the palm) to arrive at the ulnar innervated thenar eminence muscles compared to those pure median nerve fibers which travel a much more direct and hence shorter path. This marginally longer pathway requires somewhat more time which delays the CMAPs from the underlying ulnar innervated hand intrinsic muscles relative to those from the median nerve. The net result is that the ulnar innervated CMAPs’ initial deflections are not observed since the median CMAPs have already produced a large initial negative deflection. The median innervated muscles’ CMAPs initial takeoffs nullify and overwhelm the ulnar innervated CMAPs initial take off which may be positive in nature. Irrespective of the ulnar

Figure 3. Presentation of a MGA in a patient with a motor CTS at the wrist. Note the larger proximal compared to distal CMAP with both CMAPs displaying an initial negative onset. All median nerve parameters are normal except for the comparatively larger proximal CMAP amplitude (Compare this recording with that from Figure 2).
CMAPs take off (positive or negative) we continue to observe the anticipated initially negative median CMAP. However, the CMAPs negative spikes from the ulnar innervated muscles do summate with those CMAPs from the median nerve. The net result is a median CMAP derived from antecubital fossa stimulation with an initial negative deflection, but a larger amplitude than that derived from the wrist (Fig. 3). Again, this comparatively larger proximal median nerve CMAP is a result of the summation of both median innervated and ulnar innervated (through the MGA) muscle CMAPs. The calculated conduction velocity for the median nerve is within normal parameters since there is no slowing at the wrist and both the proximal and distal motor latencies reflect only median innervated muscles. Although the CMAP latencies reflect only median innervated muscles, the CMAP amplitudes do not. The distal CMAP amplitude is pure median nerve while the proximal CMAP (antecubital fossa stimulation) is a mixture of both median and ulnar innervated (MGA fibers) hand intrinsic muscles. The net electrophysiologic findings in a person without CTS but an MGA is a: 1) normal DML, 2) normal forearm conduction velocity, but 3) larger proximal compared to distal CMAP amplitude.

A word of caution is necessary at this point. Failure to appreciate the presentation of an MGA without a CTS can result in significant confusion and waste of time. This is because we typically anticipate some small but nevertheless present degree of temporal dispersion for the proximally derived CMAPs compared to their distally obtained counterparts. Documenting a larger proximal compared to distal CMAP usually suggests that our distal compared to proximal response was obtained with a comparatively submaximal stimulus. The implication of a submaximal stimulus delivery is not only a smaller amplitude CMAP, but potentially an erroneously longer latency if the fastest conduction fibers are not activated. In the above noted MGA without CTS, we may be tempted to return to the wrist and continue to deliver a larger stimulus until we obtain a distal CMAP with an amplitude comparable to that recorded for the antecubital fossa. No doubt, in some circumstances we may indeed have been submaximal at the wrist. Therefore, delivering slightly more current than previously may result in the anticipated larger distal compared to proximal median CMAP with a possibly shorter DML. In this case, all is well. However, if an MGA is present and a supramaximal current was delivered at the wrist, attempting to obtain a larger distal CMAP can potentially result in an electrophysiologic mess! Specifically, returning to the wrist and attempting to deliver ever increasing current intensities in the hope of obtaining a comparatively larger distal CMAP may only result in co-activating the ulnar nerve at the wrist. This is a distinct possibility particularly in those persons who have small wrists thereby predisposing the median and ulnar nerves to be in relatively close anatomic approximation. This anatomic proximity will predispose a large current intensity to spread from the median to ulnar nerve locations...
which may be only a few centimeters apart. The result of this action is typically a larger distal CMAP with is now contaminated with ulnar innervated muscles. At first, one may believe this to be a good thing since we are after all attempting to get a comparatively larger amplitude, and if there is an MGA are those really ulnar fibers going to ulnar muscles? Yes they certainly are, but when we co-stimulate the ulnar nerve at the wrist we could be activating most if not all of the ulnar nerve wrist fibers which consist of both the main trunk ulnar nerve fibers as well as those cross-over fibers from the MGA. In other words, we cannot activate only the MGA crossover fibers. One may attempt to circumvent this occurrence by assuming a gradual increase in current delivery at the wrist until a change in waveshape is observed and then back off a bit on the delivered current. This may occasionally work, but more likely than not, we will be unable to accurately appreciate just when the ulnar co-activated fibers begin contributing to the response, and to what degree. We are more likely to observe a gradual increase in CMAP amplitude as the wrist current delivery is also gradually increased and an abrupt change in waveform configuration or amplitude simply does not occur. Therefore, we may frequently not be able to discern just when we are truly delivering a just supramaximal response with no cross contamination from the ulnar nerve. Attempting to record from the median innervated lumbrical muscles is no guarantee of success either. At times there is just no escaping the physiologic consequences of volume conduction.

There may be some instances in which the practitioner will have to rely on the fact that the majority of patients with CTS have their sensory fibers affected. If the sensory fibers are spared, it may be worth spending only a limited amount of time attempting to problem solve an MGA without a motor CTS.

**Martin-Gruber Anastomosis With CTS: Classic Presentation.** In this portion of the discussion, we are assuming the patient has a classic demyelinating lesion of the median nerve motor fibers localized to the wrist. Stimulating the median nerve at the antecubital fossa results in a CMAP with a prolonged distal motor latency exceeding the reference population norms (Fig. 4). However, activating the median nerve at the antecubital fossa results in a number of anticipated findings. First, the CMAP arising from the elbow stimulation is noted to initiate not with a negative, but positive deflection (Fig. 4). Second, the magnitude of the proximal CMAP is larger than that of the distal CMAP. Finally, attempting to calculate a nerve conduction velocity (NCV) results in a rather high number and may typically exceed 60 M/s and not infrequently approaches 100 M/s. Documenting the above three findings is strong electrophysiologic evidence of the presence of a MGA in association with a median motor neuropathy at the wrist, i.e. motor CTS. How do we explain these findings and correlate them with pathophysiology?

We noted above a larger proximal compared to distal CMAP. A larger
In some sense, we are not only activating median nerve fibers, but also a portion of the ulnar nerve fibers destined to join the main trunk of the ulnar nerve proximal to the wrist but distal to the elbow. However, these ulnar nerve fibers Do Not innervate any median muscles. Rather, these C8/T1 cross-over fibers innervate some or all of the typically innervated ulnar hand intrinsic muscles to varying degrees. Since the active recording electrode does not “know” what we want to record, but will detect any electrical activity within its recording territory, any activated ulnar muscles as well as the median innervated muscles will contribute to the ensuing CMAP. As long as there are depolarizing ulnar muscles close enough to be picked up by the active electrode, this activity will summate with the median innervated muscles’ electrical activity to produce a “hybrid” CMAP. Those ulnar innervated muscles likely capable of contributing to the median CMAP include: deep head of the flexor pollicis brevis (FPB; most likely), adductor pollicis (AP), and possibly first dorsal interosseous (FDI). The pathway to these muscles is longer than that for the median fibers to the median innervated thenar muscles and hence should arrive at the recording electrodes location after the median nerve action potentials activate the median thenar musculature (see above explanation for MGA without CTS). However, in the patient with a demyelinating median motor neuropathy at the wrist, the median nerve action potentials induced at the antecubital fossa are delayed at the wrist. This delay allows the ulnar destined cross-over MGA fibers to

Figure 4. A classic MGA with CTS is shown with a prolonged median DML (4.7 ms) and amplitude of 8.5 mV as well as a proximal CMAP that has an initial positive deflection with a comparatively larger amplitude (9.5 mV). The NCV is measured at 75 M/s by using the proximal CMAP’s take-off to the initial positive deflection.

comparative CMAP usually implies more fibers were activated and subsequently contribute to the response (Fig. 4). We will assume the response at the wrist is supramaximal and a slight increase in current delivery at the wrist over the median nerve do not result in a progressively larger distal CMAP. Therefore, the only logical conclusion regarding the larger proximal CMAP is that more fibers contributed to this CMAP than were activated for the wrist CMAP.

We know that the MGA conveys some number of C8/T1 motor efferents and afferents through the ulnar nerve at some location approximating the mid-forearm. Stimulating the median nerve at the antecubital fossa activates all of the fibers in the median nerve at this location including those cross-over fibers to the ulnar nerve.
arrive at, and depolarize those ulnar innervated thenar muscles (FPB deep head and AP) prior to activation of the median innervated thenar muscles. The active recording electrode detects the ulnar muscles depolarizing and begins to record their CMAP. However, the ulnar innervated muscles’ motor points (end-plates) are not typically electrically coincident with the active recording electrode, i.e. the active recording electrode is off the ulnar muscles’ end-plate zone. The net result is a CMAP with an initial positive deflection. Soon thereafter, the median innervated CMAP musculature begins to depolarize with an initial negative deflection but it is too late to be recorded, i.e. it is occurring later in time than the already detected ulnar muscles’ positive deflection. At this time, both the ulnar and median innervated muscles are generating a negative spike and they both summate to result in a larger CMAP compared to that obtained at the wrist from pure median nerve activation. Therefore, the antecubital CMAP has an initial positive deflection because the ulnar innervated thenar muscles were activated before the median innervated thenar muscles (due to median action potential slowing at the wrist), and the active electrode is off the motor point for the ulnar thenar activated muscles. The larger amplitude for the proximal compared to distal CMAP occurs because there are more muscles (median and ulnar innervated) contributing to the proximal CMAP. Because of the proximal CMAP’s initial positive deflection, we are not able to record the actual delayed arrival of the median nerve through the wrist. If we measure the delayed median wrist DML and subtract it from the marginally delayed onset of the initially positive ulnar response contributing to the elbow CMAP, we arrive at an erroneously fast NCV. Again, this is because we cannot “see” the actual delayed pure median nerve fibers’ arrival at their respective thenar muscles. That is, the latency for the wrist median nerve CMAP has an initial negative take-off while the onset for the elbow derived CMAP has an initial positive deflection and using the onset for this positive deflection is all that we can realistically measure. Somewhere within this positive deflection is the true median nerve onset but it is hidden from us with no clear indication for a true median proximal latency. For this reason, the NCV in a MGA with CTS is absolutely meaningless. We are comparing the “same” nerve CMAP (median) with two different subcomponents (median and ulnar), and we do not do that for any other motor response. Reporting an MGA “median” forearm velocity is of no diagnostic benefit.

Electrodiagnostic Medicine Presentations Of Atypical MGA And CTS

Same Or Smaller Proximal Median CMAP. As noted above, one of the classic manifestations of a MGA in a patient with CTS is a comparatively larger proximal than distal median CMAP (Fig. 4). Again, this is because the ulnar cross-over fibers distal to the elbow, but proximal to the wrist are activated at the antecubital
Figure 5. A median CMAP with a prolonged DML (5.7 ms) is shown. The proximal CMAP displays an initial positive deflection but its amplitude is smaller than the CMAP derived from the wrist (wrist: 10 mV vs antecubital fossa: 9 mV). The calculated NCV is 95 M/s.

stimulation site. Their corresponding ulnar innervated thenar muscles depolarize and summate with the activated median thenar muscles thereby generating a larger amplitude than the wrist CMAP which is solely generated by median fibers activated at the wrist. However, strictly adhering to the concept that a MGA in CTS must generate a proximally derived CMAP that is larger from the elbow compared to wrist, despite documenting a proximal but not distal CMAP with a positive deflection, will result in confusion and a possible erroneous diagnosis. It is certainly possible to observe patients with a CTS and MGA that have proximal CMAPs that are either the same magnitude as the CMAP derived from the wrist or somewhat smaller (Fig. 5).

In the above scenario (Fig. 5), we do observe a negative onset median wrist CMAP with a DML that is prolonged compared to our reference population. Further, there is also documented a CMAP with an initial positive deflection with antecubital fossa median nerve stimulation. However, the antecubital CMAP is not larger than the wrist CMAP. The combination of a wrist derived median nerve initially negative onset prolonged distal CMAP with a proximal initially positive onset CMAP all supports the conclusion of a MGA. Why then, is the proximal CMAP amplitude not larger than the distal CMAP? There are a number of possible explanations for this type of MGA presentation.

In order to properly address the above question, we must first dispel the notion that there is a large population of fibers crossing over from the median to ulnar nerves. We simply are not sure of exactly how many nerve fibers actually participate in the MGA in any given patient. Further, we have no idea in any one patient which muscles and to what degree are innervated by the MGA fibers. Far too many novice practitioners assume that there are “tons” of cross-over fibers innervating every ulnar hand intrinsic muscle. This is simply not the case. Each patient is unique with respect to the total number of cross-over fibers and additionally each ulnar hand intrinsic muscle receives a variable number of these fibers. Some muscles may receive more cross-over fiber input than others. Also, recall that the further the generator site is from the recording electrode, the smaller the amplitude of the recorded response.
So, let’s put the above information together to help understand why the current CTS and MGA scenario under consideration does occur. Recall that our active recording electrode is located over the motor point of the median innervated thenar musculature. If any of the ulnar MGA cross-over innervated hand intrinsic muscles are within the recording territory of the active electrode, they will be recorded. Since the ulnar innervated fibers are activated prior to the median thenar muscles (recall slowing at the wrist for the median fibers; see above explanation) and their respective motor points are not aligned with the active electrode, a positive deflection is detected first. The ensuing negative spike of the ulnar CMAP will then summate with the negative CMAP spike of the median innervated thenar musculature. However, if the negative spike of the ulnar derived CMAP is small, it may contribute very little to the negative spike of the median CMAP resulting in either a similar or possibly lower amplitude than that typically anticipated for the proximally derived CMAP. The volume conducted initial positive deflection does not have to be very large to be detected since it occurs prior to the median CMAP. There are several reason why the MGA derived ulnar CMAP negative spike may be small enough to not significantly contribute to the proximally derived CMAP. First, if the muscles preferentially innervated by the MGA fibers are sufficiently far from the active recording electrode, the entire volume conducted CMAP will be relatively small. This may occur if the AD or FDI as opposed to the deep head of the FPB is the preferentially innervated and hence activated muscle. Second, if the MGA cross-over fibers only innervate a small percentage of the muscle contributing to the volume conducted CMAP, even if in close proximity to the recording electrode, a resulting small CMAP will be generated. Third, a combination of the above two factors may be operational. Finally, there is another very real possibility to consider which we will introduce at this juncture and return to again in another scenario. In particular, if the delay across the wrist for the median fibers is so pronounced, the MGA cross-over fibers can produce a volume conducted ulnar generated CMAP that occurs sufficiently prior to the median derived CMAP from the antecubital fossa site, it will contribute little to the overall antecubital fossa CMAP magnitude. In other words, the negative spike of the cross-over ulnar derived CMAP is beginning to manifest prior to that for the median derived fibers resulting in a less than optimal situation for negative spike waveform summation. If the negative spikes for the two separately derived nerve fiber populations to not summate optimally, the resulting waveform will fail to be larger than that at the wrist with a somewhat commensurate prolongation in the overall (initial positive deflection to negative spike return to baseline) proximal compared to distal CMAP duration and area under the curve. Further, the more delayed the median fibers are, the more likely the terminal “downward going” positive phase of the MGA ulnar CMAP may coincide with the delayed “upward going” negative median CMAP and phase
cancel thereby reducing the overall CMAP negative spike magnitude. Any or all of the above explanations may be operational in any given patient to generate an antecubitaly derived CMAP that is similar or smaller in amplitude compared to the pure CMAP from the wrist.

The above explanations provide a very real reason why many patients with a MGA and CTS to not display a proximally derived CMAP with a larger magnitude than that derived at the wrist. Therefore, documenting a patient with a sensory CTS and a motor study with a proximally derived CMAP with an initial positive deflection without a larger amplitude than that for the wrist site is still very consistent with a MGA. Of course, the wrist DML is still prolonged and calculating a forearm NCV remains a questionable practice as the velocity may be erroneously fast or even negative in nature if the wrist latency is so prolonged that it exceeds the antecubital fossa derived CMAP latency (initiation of the positive deflection subtracted from a longer wrist latency; see below).

**Normal DML And Proximal CMAP With An Initial Positive Deflection.**

This scenario of a MGA and CTS is perhaps the most challenging for practitioners to grapple with because it challenges a number of our unexplored assumptions. Let us explore a patient presentation exemplifying this particular manifestation of an MGA and CTS (Fig. 6). In this instance we have a patient present with the classic signs and symptoms for CTS. A

![Figure 6](image)

Figure 6. A median nerve CMAP from the wrist is documented with a normal DML (3.8 ms) and amplitude (8.8 Mv) while a proximal CMAP with an initial positive deflection is shown with an amplitude of 8 mV). In this case the amplitude is less but it could easily be larger as well. The calculated forearm NCV is 52 M/s.

electrophysiologic sensory assessment reveals focal slowing of the median sensory fibers across the wrist consistent with a typical sensory CTS. The motor studies are then performed with a number of interesting findings. The median nerve is stimulated at the wrist and reveals a thenar CMAP with an initial negative deflection and more importantly a DML that is well within our reference population and amplitude that is also quite normal. So far, there is no electrophysiologic indication of motor involvement in this CTS presentation, i.e. no evidence of median nerve motor fiber demyelination across the wrist or axonal loss based on CMAP latency and amplitude respectively. However, stimulating the median nerve in the antecubital fossa results in a CMAP with an initial positive deflection with
an amplitude that may be larger, the same, or even smaller than that derived from the wrist (Fig. 6). Now what?

We have a somewhat confusing presentation where the patient clearly has a sensory CTS, but what about the motor fibers, is there a motor CTS as well? Let us return for a moment back to basic anatomy and physiology. If we detect a CMAP with an initial positive deflection, then the fibers generating that positive deflection originated away from the active recording electrode’s location, i.e., a volume conducted response. The wrist stimulation generated a CMAP with an initial negative deflection, therefore, we know we are over the median innervated muscles’ motor point and definitely should under no circumstances attempt to relocate the active recording electrode. The initial positive deflection from the proximally derived CMAP suggests that an ulnar activated muscle (there are no other nerve/muscle options) arrived within the proximity of the electrode’s recording territory prior to the median nerve’s fibers, and this implies we have a MGA operational. However, we have stated above that MGA cross-over fibers must travel a longer pathway than the native median nerve fibers and should always arrive at the thenar eminence after, and never before, the median fibers traversing a shorter pathway unless there is slowing at the wrist. In this author’s opinion the only logical explanation is that the MGA ulnar fibers did indeed arrive at the thenar eminence recording electrode prior to that for the median fibers. Therefore, slowing of conduction must exist for the median nerve fibers across the wrist region despite a DML within our reference population. We must conclude that despite a “normal” DML, the antecubital fossa initial positive deflection CMAP must take priority and define a motor CTS.

The question at hand persists: How can we conclude that there is slowing across the wrist in the face of a clearly “normal” distal motor latency? I would submit that the answer lies in the manner in which we statistically derive our reference population. In any large data set there are always a mean and standard deviations defining that population. This large conglomeration of data works quite well most of the time, but we also know that there is an unavoidable subset of patients within the larger set that are defined as false positives and false negatives. Specific to our particular situation, there will always be individuals with a DML that falls within our two standard deviations for “normal” but in reality have a DML that is abnormal for them, i.e. a false negative. We do not have any one person’s pre-morbid DML. It is entirely reasonable to assume that a nerve’s DML affected by a progressive lesion (e.g. CTS) does not start out at a very short or “normal” value and then instantly “jump” into the abnormal range. Rather, as disease progression continues, there is a gradual lengthening or delay of the DML. Depending upon where in that disease process we happen to examine a particular patient, we may or may not observe a DML that has exceeded our mean and two standard deviations for our reference values. For example, if a person starts our with a DML of
2.9 ms prior to incurring CTS and then has a DML of 3.9 ms when we happen to examine them, the documented value is still within our “normal” values. However, there is a 25% increase in the patient’s DML. This patient’s prolonged value may continue to be within our “normal limits” but is outside of the patient’s pre-disease value. The cross-over MGA fibers can serve as our internal control in these patients informing us of a relative slowing of median fibers across the wrist for this particular patient thereby avoiding a potential false negative conclusion. If the patient in question is not treated, and subsequently examined at some point in the future, their DML will no doubt exceed the upper limit of our reference population and present in a more typical pattern. It is important for the practitioner to keep the above in mind when a patient presents with signs and symptoms consistent with a CTS and displays an abnormal sensory study with the above noted motor findings. I believe it is reasonable to conclude that the patients has both a motor and sensory CTS. Of course, even if you don’t believe any of the above, the patient should still be treated for CTS since we have already stated the sensory studies as well clinical presentation are consistent with a median neuropathy at the wrist. The above electrophysiologic presentation is not an uncommon finding should one choose to look for it. It is the author’s experience that this particular presentation is always accompanied by a sensory study abnormality on NCV. It is certainly possible, although rare, that a patient with a pure motor CTS might display only the above motor findings.

Shorter Proximal Compared To Distal CMAP Latency (Negative NCV). In some patients with a sufficiently delayed distal median CMAP, it is possible for the proximally activated MGA fibers to generate a CMAP with a shorter latency as measured from the initial positive deflection’s take-off (Fig. 7). Because of the MGA’s CMAP positive deflection, we cannot ascertain the exact take-off of the later arriving median fibers. In this instance, the difference between the two latencies is actually a negative number which results in a negative NCV. Again, this makes no physiologic sense, but an interesting observation nevertheless. As noted above, reporting an NCV in a patient with a MGA and CTS is of little value and even more so when that velocity suggests the action potentials are traveling back in time!

Figure 7. In this instance of a CTS with an MGA we observe a markedly prolonged distal response (DML: 8.1 ms/3.8 mV) while the proximal response has an initial positive onset that is actually shorter than the distal response (PML: 6.9 ms/3.7 mV). The calculated NCV is -144 M/s.
Figure 8. In this patient, the wrist median response demonstrates a DML of 13.4 ms (5.7 mV) while the proximal response reveals a PML of 9.3 ms (measured to onset of the positive deflection). The important aspect of this patient is the actual separation of the MGA CMAP from that of the median CMAP. The proximal median CMAP is 5.1 mV. The calculated NCV is -51 M/s.

Separation Of The MGA And Median Fibers. It is rarely possible to actually observe the contributing component of the MGA as a distinct waveform (Fig 8). In those patients who have a profound degree of demyelination with an accompanying exceedingly long DML, the MGA component of the proximal CMAP can separate out from the median derived CMAP. The end result is two distinct and subsequently occurring CMAPs. The first smaller CMAP with an initial positive deflection represents those MGA cross-over fibers generating a volume conducted CMAP. The second and large CMAP with a negative deflection represents the pure median component of the proximal CMAP. It may be possible to calculate both the erroneous and true conduction velocities in these patients (Figs 8 & 9). One can also observe that the MGA component is usually quite small in magnitude when compared to the median CMAP for reasons already discussed above. The practitioner may be perplexed by the two distinct CMAPs with proximal stimulation and attempt to re-adjust the active recording electrode. This is not a wise course of action since the distal response clearly defines the active electrode as being in the proper location and over the median innervated muscles' motor point. Recognizing that a very prolonged wrist DML indicates profound demyelination which in turn permits separation of the MGA and median nerve fibers' respective CMAPs can save quite a bit of time and confusion.

Initial Negative Deflection. The combination of a MGA and CTS does not always present with an initial positive deflection. It is certainly possible for a patient with a CTS and MGA to have both a distal and proximal CMAP with an initial negative and not positive deflection.
The explanation for this finding is rather straightforward. There is no rule that muscles with different innervations overlying each other cannot have either anatomic and/or electrical alignment of their respective motor points. Specifically, the deep head of the FPL for example, may be situated in the thenar eminence in such a way as to have its end-plate region coincidentally aligned with those of the APB. In this instance, when the median nerve is activated at the antecubital fossa, those fibers innervated by the cross-over MGA ulnar fibers will depolarize the deep head of the FPL and generate an initial negative and not positive deflection. The CMAP waveforms generated from both the median and cross-over ulnar fibers will summate to generate a larger CMAP at the elbow compared to wrist stimulation. Also, the calculated NCV may be erroneously fast or even negative since the median fibers at the wrist should be slowed in the CTS. In short, the median DML will be prolonged, while the proximal CMAP could be larger, the same, or smaller than that at the wrist with a corresponding abnormal forearm NCV secondary to the MGA fibers bypassing the wrist. One could easily miss the presence of the MGA in this instance since there is no initial positive deflection and the large amplitude for the proximal CMAP may or may not be present while the fast forearm velocity could be considered “normal” for this patient unless it is negative. If the proximal motor latency is less than the DML, however, then clearly a MGA is likely present.

**Figure 10.** It is certainly possible for a MGA in a patient with a CTS to produce a proximal CMAP with an initial negative deflection if the motor point of the ulnar innervated muscles just happen to align with the active recording electrode over the APB’s motor point. A) Median CMAP with a prolonged DML (10 ms/4.8 mV). B) Proximal CMAP with a shorter and initially negative onset (9.2 ms/5.2 mV). The calculated NCV is -287 M/s.

**ULNAR NERVE AND THE MARTIN-GRUBER ANASTOMOSIS**

**Anatomic/Physiologic Considerations.** We have discussed the consequences of a MGA with and without CTS, but what about the ulnar nerve in patients with a known MGA irrespective of CTS? As noted above, a variable number of “ulnar” destined fibers initially travel with the median destined nerve and then cross-over to the ulnar nerve somewhere in the mid-forearm. From the perspective of the ulnar nerve, there are now more ulnar fibers at the wrist compared to the elbow. This anatomic fact has physiologic implications and may be relevant during the electrodiagnostic medicine evaluation.

**Clinical Implications.** It should be obvious at this point that there are a
Figure 11. Recording from the ADM while stimulating the median nerve at the wrist and below elbow in a patient with a MGA. Note the somewhat excessive decline (26%) in amplitude for the proximal compared to distal response.

Figure 12. Activating the ulnar nerve at the wrist, below elbow, and above elbow sites results in no significant drop in amplitude in a patient with a MGA.

variable number of ulnar destined fibers that manage to bypass the usual anatomic ulnar nerve pathway until the forearm. This suggests that an axonal loss lesion of the ulnar nerve proximal to the mid-forearm, will result in some degree of ulnar innervated hand intrinsic musculature sparing. Confusion can arise if the MGA is not considered, as one could conclude that the patient has an all median hand. An all median hand is highly unlikely and an MGA should always be considered first.

Additionally, such a patient may continue to have some hand function if both an ulnar nerve lesion at the elbow is sustained in conjunction with a median nerve lesion at the wrist. The needle EMG can be rather enlightening in such patients and the interested reader is encouraged to consider additional implications. We are preferentially concerned with the NCV and not the needle EMG in patients with a MGA, and no suspected ulnar nerve lesion at any location.

Electrophysiologic Implications. As noted above, the ulnar nerve at the wrist will contain more nerve fibers than at the elbow. Stimulating the ulnar nerve at the wrist and elbow in patients without pathology could lead to an erroneous conclusion of conduction block of the ulnar nerve in the mid-forearm. Specifically, stimulating the ulnar nerve at the wrist and recording from the abductor digiti minimi (ADM) may result in a CMAP with a normal latency and amplitude. However, activating the ulnar nerve at the elbow can produce a CMAP with a smaller amplitude than anticipated (Fig. 11). This is due to fewer fibers being activated at the elbow compared to the wrist. Detecting a smaller amplitude than that anticipated for normal temporal dispersion can lead to an erroneous conclusion of conduction block. In this author’s experience, the above noted “significant” discrepancy between proximal and distal ulnar CMAP amplitudes in patients with a MGA is rather uncommon. It is more usual to not notice any significant difference in amplitude (Fig. 12). The reason for this lack of significant amplitude differences is a result of several factors. First, there is lack of
agreement on just how much an amplitude must decline between the wrist and below ulnar nerve activation sites for pathology to be present. It may be that in some patients with an MGA the amplitude decline due to the cross-over fibers does not rise to significant levels.

Second, there is a variable distribution to the hand intrinsic muscles from patient-to-patient and the ADM may simply have very few fibers traveling through the MGA. Also, there is an assumption that large numbers of fibers participate in the MGA and this simply may not be the case. There may be additional factors but these are the obvious ones.

**Median CMAP Wrist Initial Positive Deflection Pitfall (Just For Fun).** We have extensively discussed the reasons for an MGA generating a CMAP with an initial positive deflection from antecubital fossa stimulation. However, what if median nerve wrist stimulation produces an initial positive deflection, but antecubital fossa median nerve stimulation does not when recording from the APB (Fig. 13). One may initially and reasonably conclude that the active recording electrode is not on the motor point for the median thenar muscles. This is not necessarily wrong, but could lead to a “wild goose chase” and waste valuable time if the median nerve at the elbow is not immediately activated prior to concluding anything. I am specifically advocating a problem solving approach through critical thinking instead of a reflex response of moving electrodes. In particular, I would recommend that the detection of a CMAP from the thenar eminence with an initial positive deflection following supramaximal median nerve wrist stimulation result in the practitioner first proceeding to the elbow and again stimulating the median nerve without touching the active recording electrode. If a positive deflection is observed with median nerve stimulation at the wrist, one of three possibilities may exist: 1) active electrode is off the motor point, 2) MGA is operational and the active electrode may or may not be off the motor point, and 3) active electrode is on the APB motor point but the ulnar nerve is stimulated at the wrist.

Let’s consider the above possibilities. Again, the median nerve is activated at the wrist and an ensuing CMAP with an initial positive deflection arising from the APB is noted. Proceeding to the elbow and stimulating the median nerve may now generate a CMAP with an initial negative deflection. This is a rather common occurrence. This proximal CMAP’s initial negative deflection tells us that we are indeed on the motor point of the APB and a MGA is likely not present. The
practitioner should return to the wrist and again activate the median nerve with less current intensity, or locate the cathode and anode just medial to the flexor carpi radialis tendon. Both procedures are attempting to reduce the current delivery at the wrist to ensure optimal stimulation of the median nerve without co-activating the ulnar nerve. The positive deflection for the CMAP at the wrist without a comparable positive deflection at the elbow strongly suggests the ulnar nerve is activated at the wrist with ulnar innervated muscles in close proximity to the active recording electrode (deep head FPL) contributing to the median CMAP. The deflection is positive because of the early occurring volume conducted source currents feeding the ulnar intrinsic muscles overwhelming the median CMAP's initial negative deflection. Remember, in most instances the active recording electrode over the APB is not over the motor point for the ulnar innervated hand intrinsic muscles and a CMAP from these muscles will initiate with an initial positive deflection. When we either reduce the current intensity over the median nerve or move the cathode slightly away from the ulnar nerve, the CMAP associated with wrist stimulation immediately presents with an initial negative deflection as the ulnar nerve is no longer activated (Fig. 14). If the practitioner failed to obtain a proximal response and record an initial negative deflection prior to doing anything else, the tendency would be to move the electrode over the APB so as to better approximate the motor point. In fact, in this instance the practitioner would actually be making the situation worse by inadvertently moving the electrode off the APB's motor point. Of course, one would not return to the original location since it generated a positive deflection and subsequently relocate the active electrode everywhere except where it should be, i.e. the original location. This is the above noted "wild goose chase."

On the other hand, if a CMAP with a positive deflection is noted with both wrist and elbow stimulation, this may indeed represent a case where the active electrode is off the APB’s motor point. Relocating the electrode in small increments to better approximate the muscle's mid-belly may result in a negative deflection provided the above course of action is pursued first, i.e. lower the current intensity/relocating the cathode. At this point, if a negative onset CMAP results with wrist stimulation, then we know the active electrode is correctly positioned over the APB motor point. Now, proceeding to the elbow stimulation site should result in a CMAP with an initial negative deflection. If it does not, then a MGA should be considered irrespective of
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Figure 15. A median nerve response from the wrist and elbow are obtained in a patient with CTS. The DML is 5 ms (10 mV) and a forearm NCV of 53 M/s.

the DML (see above scenarios). I believe the above noted protocol is more productive and time saving than reflexively moving the electrode prior to understanding what is going on electrophysiologically.

CTS DML/NCV MISCONCEPTIONS

It is this author’s opinion that the “lowly” CTS evaluation can at times be anything but straightforward and thereby can teach us an incredible amount of electrophysiology as it relates to median nerve pathophysiology. A critical attention to detail can lead us into a deeper understanding of the electrophysiologic manifestations of disease processes. We cannot be afraid to ask hard questions as to why observations are as they are, and refrain from answering with: “Because this is the way I was taught.” Not uncommonly such simple questions such as Why?, frequently initiate a rewarding journey into the unknown. Electrodagnostic medicine is no different, and so for a short time let’s ask some “why” questions and see where they take us.

In all of the upcoming scenarios we are going to assume a patient has clinical signs and symptoms of CTS. Further, we will assume that there is sensory documentation of median nerve slowing across the carpal tunnel region. A number of questions will be asked regarding the motor findings in such patients that perhaps may not have occurred to many practitioners despite observing these findings. Hopefully we can address the following: “I always wondered about that but couldn’t find it addressed anywhere.”

Prolonged DML With A Normal Forearm NCV. The combination of a prolonged DML with a normal forearm conduction velocity is perhaps the most common finding in the majority of patients with CTS (Fig. 15). The explanation for this finding is rather straightforward. The slowing of conduction across the wrist is obviously due to some degree of demyelination. Stimulating the median nerve at both the wrist and elbow requires the traveling action potentials from both locations to undergo the same degree of slowing across the wrist segment. Since this slowing is common to both stimulation sites, it is eliminated from consideration for calculating the forearm NCV as this latency is common to both activation sites and is subtracted out. The net result is a true time for neural conduction within the forearm segment for the measured distance. For example, a DML of 5.0 ms is subtracted from a proximal motor latency (PML) of 9.5 ms to yield a difference of 4.5 ms over a forearm distance of 23 cm with a resulting NCV of 51 M/s (PML − DML = forearm
latency; 9.5ms – 5.0ms = 4.5 ms). The DML time across the wrist of 5.0 ms is common to both activation sites and thereby eliminated. The velocity of 51 M/s is likely as true a representation of the motor fibers' velocity in the forearm as is possible. The above description should pose no major stumbling blocks. However, this reasoning fails miserably at explaining a number of other electrophysiologic motor findings in patients with CTS. Let's consider additional scenarios and attempt some reasonable explanations.

**Prolonged DML And A Slowed Forearm NCV.** It is not particularly unusual to document patients with a sensory CTS and median motor findings of the anticipated slowed DML but with a commensurately slowed forearm NCV (Fig. 16). The above noted explanation of a normal forearm NCV simply does not work in this situation. How can we have a slowed DML with a slowed forearm conduction velocity as well if the distal segment is truly subtracted out as a common element to both stimulation sites?

An explanation in the literature put forward to explain the above scenario relies on the concept of the pathology of the median nerve at the wrist resulting in “retrograde” degeneration of the median nerve up the forearm killing off the faster conducting fibers. This explanation has never made any sense to this author. First, there is no documentation in the literature that a lesion of an axon distally leads to the proximal destruction of the axon along its entire course or at least a significant distance along its length (in this case the forearm length of 20-30 cm). A report in guinea pigs did reveal some degree of proximal neural degeneration but it was no more than a centimeter. Second, the electrophysiologic studies purporting to document this proximal degeneration was not convincing to this author and had experimental design flaws (see Bibliography for a more extensive discussion). Third, the assumption is that axonal loss must be present to induce this presumptive retrograde degeneration. Patients who display this type of presentation do not have reduced amplitudes and denervation of the median innervated hand muscles in this author’s experience to suggest axonal loss so as to produce retrograde degeneration (Fig. 16; CMAP with 7 mV amplitude). Additionally, significant demyelination is not sufficient to induce retrograde degeneration since many patients with very prolonged DML have acceptable forearm NCVs.

Rather, the explanation that seems to make the most physiologic sense to this author without invoking a finding that has not been documented, is to consider the basic pathophysiology of CTS and the manner in which the
CMAP is recorded. First and foremost, our active recording electrode for both stimulation (wrist/elbow) sites is distal (APB) to the presumptive site of pathology, i.e. the wrist. Therefore, we can only observe what passes through the carpal tunnel and only indirectly infer what is happening in the forearm. We know that the pathology in CTS occurs at the wrist for sure and likely not in the forearm up to and perhaps proximal to the elbow. An obvious question becomes: What type of pathology would produce a prolonged DML and a slow forearm conduction velocity given pathology localized to the wrist. The prolonged DML clearly implies slowing of neural conduction and this can result from: 1) demyelination, 2) conduction block of the faster conducting fibers, 3) axonal loss of the faster conducting fibers at the wrist, and/or 4) some combination of the preceding. If we consider pure demyelination with no conduction block or axonal loss, then the above noted explanation is operational for a prolonged DML and normal forearm NCV as noted above, i.e. the DML is “subtracted” out of the measurement. Hence this cannot be a viable explanation in and of itself. Some other process must be operational in addition to just pure demyelination. Suppose some degree of conduction block is present. This is certainly a not unreasonable consideration since demyelination can lead to a variable degree of conduction block and may indeed progress from demyelination with conduction slowing to more advanced demyelination with failure of action potential propagation, i.e. conduction block. If conduction block of the fastest conduction fibers were present, then these fibers would never arrive at the recording electrode. Again, this is not an unreasonable assumption since the largest fibers with the fastest conducting velocities are know to have the highest metabolic rates and likely to be compromised first. If the fastest fibers never arrived at the recording electrode because they are blocked at the wrist but are just fine in the forearm, then only those that do arrive through the wrist contribute to the NCV. Conduction block of the faster conducting fibers permit only documentation/measurement of the slower conducting fibers through the carpal tunnel, and hence directly account for the slowed forearm conduction velocity. Specifically, demyelination and conduction block can account for both a prolonged DML and slowed forearm conduction velocity. Similarly, if axonal loss were present at the wrist taking out the faster conducting/high metabolic rate fibers at the wrist, they again would not be able to be measured at the recording site even though they are just fine in the forearm. Only those more slowly conducting and still preserved fibers are available for measurement and hence account for the slowed forearm NCV. Note, this axonal loss is present only at the site of pathology and does not require axonal loss of the fastest fibers along the entire proximal course of the nerve. The net result is the same as for conduction block, i.e. a prolonged DML and slow forearm NCV. Finally, some combination of demyelination, conduction block, and axonal loss also explains the prolonged DML and slowed forearm NCV. None of the above explanations stretch credulity
since we are certainly familiar with a spectrum of disease progressing from mild to severe demyelination with eventual conduction block potentially progressing to axonal loss if the offending agent is not corrected. Hence, the need to intervene with either conservative and/or surgical strategies becomes all the more important to reverse any conduction block prior to progression to axonal loss. The documentation of a slowed forearm NCV may have some bearing on severity of disease and the need for more aggressive interventional strategies.

**Normal DML And A Slowed Forearm NCV.** If one performs a large number of CTS evaluations, it is only a matter of time before this type of electrophysiologic presentation is noted (Fig. 17). Again, as with all the other scenarios discussed, the patient is documented to have a sensory CTS by electrophysiologic techniques. The question at hand remains, does this patient have a motor CTS as well?

To be sure the above electrophysiologic finding is interesting and requires an equally interesting explanation. The normal DML surely suggests there is a lack of demyelination and its accompanying slowing of conduction. After obtaining the DML there is no suggestion of a motor CTS (remember the patient is symptomatic for CTS and has consistent sensory findings). However, upon calculating a slowed forearm conduction velocity the thought process should come to a grinding halt at this point. Why would a patient with a totally normal DML end up with a slowed forearm NCV (we are assuming only a CTS and no peripheral neuropathy). Our previous explanation of forearm slowing can be of assistance in this scenario as well.

Recall, if the response never gets through to the recording electrodes, it cannot contribute to the end result. In this particular case, what if there was blockade of the fastest conducting fibers just as for the above noted scenario of forearm slowing. Once again, we would only be able to detect the slower conducting fibers and only those fibers passing through the carpal tunnel would influence what we calculate for the forearm NCV. Therefore, if the faster conducting fibers were block secondary to conduction block at the wrist, we should have a resultant slowing of forearm conduction with a prolonged DML. Oops, we have a “normal” DML. Now what? No worries, let’s work our way carefully through this apparent enigma.

As noted, if we invoke the concept of conduction block for the fastest conducting fibers, then we can easily account for the slowing of forearm NCV. But, we must also
simultaneously account for the normal DML. At this point it becomes relevant to again invoke the concept of a reference population and a statistical distribution. We have no idea as to the patient's premorbid median DML. It may have been considerably shorter than that which is measured at the time of examination. We have already discussed how a patient's DML can increase a considerable degree and still remain well within two standard deviations of the mean, but nevertheless still be a false negative, i.e. abnormal for this patient. Also, as previously noted, this is an inevitable occurrence given enough patient encounters. So far we have invoked both conduction block of the fastest conducting fibers at the wrist as well as an actual prolongation (for this particular patient) of the DML that still remains within the "normal" range. In this author's opinion, it is the above combination of findings that fully explains this scenario of a normal DML with a corresponding slow forearm NCV. I can hear some practitioners stating: "But wait! The DML is normal and so should be the forearm NCV." The reasoning here may be that the fastest conducting fibers in the forearm should arrive at the APB before the slower conducting fibers and thereby possess the fastest DML. If the DML is normal then there should be preservation of the forearm NCV for this same reason. Therefore, conduction block of the fastest conducting forearm fibers cannot correlate with both a "normal" DML and a corresponding slowed forearm NCV.

The above rationale appears sound on first examination, but there is an unfounded assumption and hence flaw of reasoning that lies at the crux of the explanation. Quite simply, it is assumed that the fastest conducting forearm fibers will arrive at the APB prior to the more slowly conducting forearm fibers. In other words, there should be a direct correlation between forearm conduction velocity and DML. In other words, those persons with the fastest forearm NCVs should also have the shortest DML. Is this true? Let's take a look.

The above assumption that there is a direct correlation between forearm NCV and DML can be quite easily documented. We can record the DML and forearm NCV in approximately 50 asymptomatic individuals and evaluated them electrophysiologically to ensure that they have neither signs/symptoms or sensory findings to suggest a CTS. Plotting their forearm NCV and DML is rather straightforward. The end result is quite surprising. Specifically, there is absolutely no correlation between DML and forearm NCV (Fig. 18). An individual with a very fast NCV could just as easily have a lower limit of normal as well as a short DML. Similarly, patients with short DML could have very fast or borderline slow but still normal forearm NCVs. From a statistical standpoint, the correlation coefficient was very close to zero, i.e. no correlation between DML and forearm NCV. Upon reflection, there are several anatomic and physiologic reasons to account for this finding. Each individual axonal in the forearm irrespective of it diameter and hence velocity, must break up into
Figure 18. A graphic depiction of median nerve DML vs NCV in healthy individuals is shown. Vertical axis: NCV. Horizontal axis: DML.

Many small fibers with varying calibers in order to innervate each single muscle fiber. These small caliber fibers can have considerable variability in their respective conducting velocities and hence will take different amounts of time to reach their corresponding single muscle fiber. Also, there is no guarantee that the largest forearm fibers will continue to have the largest fibers along their entire course to the individual single muscle fibers. Further, there is a different distance to each muscle fiber from where each of the main nerve trunks divide into small axons and forms an end-plate. We know that each neuromuscular junction also has a variable and different time of delay prior to activating a single muscle fiber. Again, there is no guarantee that the fastest conducting forearm fibers will have the largest caliber intramuscular axon with the shortest distance to their corresponding fibers and also have the shortest time across the neuromuscular junction. Each nerve fiber also has its own latency of activation and there is no documentation that the largest fibers have the shortest latency of activation, in fact it may take more current to activate a nerve with a larger cross sectional area than a smaller one which means it will take longer for the action potential to get going. I think you can now better appreciate why there is in reality no physiologic correlation between the forearm NCV and DML. Therefore, in light of this explanation we can see why a “normal” DML is not necessarily “normal” for that individual patient, and the fastest forearm conducting fibers can be blocked without having to have a DML that is clearly abnormal. In other words, those faster conducting fibers in the forearm may actually have a longer DML than comparatively slower forearm conducting fibers (see Fig. 18). Further, the patient’s DML is actually prolonged as well but still within our reference population of “normal values”. All of the above combine to generate a so called “normal” DML with a slow forearm NCV. To be sure, a lot to think about, but nevertheless well worth pondering.

Closing Thought For Fun

Let’s suppose you are examining a patient’s upper limb and perform a routine ulnar motor study to the ADM and observe a smaller CMAP amplitude proximally than distally greater than anticipated (Fig. 19). If the ulnar CMAP latencies and NCV are normal, consider the possibility of an MGA. You may be able to predict the presence of an MGA prior to even assessing the median nerve. When you study the median nerve CMAP look for a larger amplitude proximally with or without an initial positive deflection. If you don’t observe an initial positive deflection then there is
Figure 19. The lower trace represents the ulnar CMAPs from the wrist and elbow with a greater than anticipated drop in amplitude proximally. A MGA was predicted, and when the median CMAPs were derived it is clear the proximal CMAP is larger than the distal (distal response was supramaximal). The median DML was normal and there is no proximal median CMAP with an initial positive deflection suggesting a MGA without CTS.

likely a MGA without a CTS and if you do see a initial positive deflection there is a CTS present.

SUMMARY

We have attempted to focus on a number of conundrums with respect to the apparently simple electrophysiologic evaluation of CTS. As noted above, all may not be as it appears on first inspection. There are a number of interesting findings that only become apparent when critical assessment of their physiologic meaning is sought. Hopefully, a number of the above examples have resulted in some level of discomfort with our current level of practice. These actual patient findings can serve to help us take our knowledge base to the next level. Even the simple CTS evaluation has a tremendous amount of information to impart to us.

BIBLIOGRAPHY