Case report

Mid-palm recording technique, a new electrodiagnostic approach in Martin-Gruber anastomosis

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ABSTRACT

Introduction: Median and ulnar motor nerve conduction study recording from median-innervated 2nd lumbrical and ulnar-innervated 1st palmar and 2nd dorsal interossei is designated in electrodiagnostic evaluation of carpal tunnel syndrome. In this technique, both responses are recorded by the same surface recording electrode placing over their shared motor point in mid-palm. To the best of our knowledge, this technique has never been utilized in demonstration of Martin-Gruber anastomosis.
Case reports: By applying this technique to the conventional CMAP comparison method, the authors accordingly demonstrated Martin-Gruber anastomosis in four cases. Three presented with focal mononeuropathies of the upper limbs, including a severe carpal tunnel syndrome, a mild to moderate carpal tunnel syndrome, and an ulnar neuropathy. One was a normal individual.
Significance: This report described a new electrophysiological pattern of MGA. Adding the mid-palm recording to conventional CMAP comparison method provides a broader view in terms of innervation of MGA. This technique is not complicated and can be added to the electrophysiological investigation for complete studying of the innervated muscles in MGA.
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1. Introduction

Martin-Gruber anastomosis (MGA) is the most common median and ulnar nerve anastomosis of the upper limb with the pooled prevalence of 19.5% (Roy et al., 2016). In MGA, the axons traveling with the median nerve cross over to join and travel with the ulnar nerve to innervate intrinsic hand muscles. MGA can be demonstrated by anatomical or electrophysiological studies. The anatomical studies, mostly done in cadavers, display MGA by direct demonstration of neural connection. The electrophysiological studies, on the other hand, may indirectly demonstrate median and ulnar nerve anastomoses in the forearm when dual innervation of an intrinsic hand muscle was found. Two electrophysiological investigations, the compound muscle action potential (CMAP) comparison method and the collision technique, have been desig-nated to demonstrate MGA (Kimura et al., 1976; Oh, 1993; Sander et al., 1997).

The CMAP comparison method is more simplified and less time-consuming. With this method, supramaximal stimulations are delivered to median nerve at the wrist (MW), median nerve at the elbow (ME), ulnar nerve at the wrist (UW) and ulnar nerve at the elbow (UE). Conventionally, the CMAPs are consecutively recorded from thenar, hypothenar and 1st dorsal interosseous (1DI) muscles by surface recording electrodes. In every recording site, CMAPs evoked from four sites of stimulation are compared. By this technique, three well-recognized electrophysiological types of MGA, classified by the innervated muscles, have been demon-strated. Type 2 (1DI) is found to be the most common, followed by type 1 (hypothenar) and less common type 3 (thenar) (Khosrawi et al., 2015; Roy et al., 2016).

Median and ulnar CMAPs can also be recorded from median-innervated 2nd lumbrical (2 Lum) and ulnar-innervated 1st palmar and 2nd dorsal interossei (1PI/2DI) by placing an active surface recording electrode over their shared motor point in mid-palm and a reference electrode distally. This technique has been useful in the diagnosis of carpal tunnel syndrome (Boonyapisit et al., 2002; Kaul and Pagel, 2002). However, it has never been utilized

to demonstrate MGA. The objective of this report was to demonstrate the electrodiagnostic pattern of MGA with mid-palm recording in four cases. Three cases presented with focal neuropathies of the upper limb (severe carpal tunnel syndrome, mild to moderate carpal tunnel syndrome, and ulnar neuropathy at the elbow) and one case was a normal individual. This study was approved by the institutional review board. All patients provided signed, informed consent.

2. Electrophysiological study

In addition to routine nerve conduction study and electromyogram, the CMAP comparison method was done in all cases. Recording electrodes were placed over four recording sites: (1) *thenar* (motor point of abductor pollicis brevis (APB) (G1) and the first metacarpophalangeal joint (G2)), (2) *hypothenar* (motor point of abductor digiti minimi (ADM) (G1) and the fifth metacarpophalangeal joint (G2)), (3) *1DI* (motor point of 1DI (G1) and the first metacarpophalangeal joint (G2)) and (4) *mid-palm* (the shared motor point of 2Lum/1PI/2DI, just lateral to the midpoint of the third metacarpal bone (G1) and distal to the second metacarpophalangeal joint (G2)). Median and ulnar nerves were stimulated at the wrist and the elbow, as in standard nerve conduction study. Stimulus intensity was gradually increased in each stimulus, by less than 5 mA to its supra-maximal intensity. Stimulus duration was constantly maintained at 0.1 ms. At each stimulation site, the stimulus intensity was just reached its supramaximal threshold or was slightly above it. Still, the stimulus did not spread to co-stimulate another nerve. For each recording site, the amplitude, area and morphology of CMAPs, evoked from four sites of stimulation, were compared. All studies were carried out with Viking on Nicolet EDX (Viking software version 22).

3. Case presentation

3.1. Case 1

A 50-year-old right-handed housekeeper presented with numbness of both hands for two years. Examination showed decreased sensation along the radial side of both palms. She had weakness and atrophy of the right APB and positive Phalen's test, bilaterally. Electrodiagnostic study was remarkable for absent bilateral median-digit II SNAPs, markedly prolonged bilateral median-APB distal motor latencies (R, 7.40 ms; L, 6.82 ms (reference value <4 ms)), reduced right median-APB distal CMAP amplitudes (R, 2.9 mV; L, 7.3 mV (reference value >4 mV)) and active denervation potentials found at the right APB. Sensory and motor nerve conduction studies of bilateral ulnar nerves and sensory nerve conduction study of bilateral superficial radial nerves were normal. These findings were consistent with severe right median mononeuropathy and moderately severe left median mononeuropathy at the wrists. The MGA (thenar, hypothenar, 1DI and mid-palm) was demonstrated on the right side. (Fig. 1A, Supplementary Fig. 1 – Appendix 1.)

3.2. Case 2

A 45-year-old man presented with numbness of both hands and weakness of right thumb for six months. Examination showed decreased sensation to pin along the radial side of right palm and at the left digit III. He had mild weakness of the right APB. Sensory nerve conduction study was remarkable for prolonged bilateral median-digit II distal latencies and reduced right median-digit II SNAP amplitude. Motor nerve conduction study showed prolonged bilateral median-APB distal latencies (R, 8.18 ms; L, 4.43 ms) and

reduced right median-APB distal CMAP amplitude (R, 1.2 mV; L, 7.8 mV). Sensory and motor nerve conduction studies of bilateral ulnar nerves and sensory nerve conduction study of bilateral superficial radial nerves were normal. Findings were consistent with severe right median mononeuropathy and mildly to moderately severe left median mononeuropathy at the wrists. The MGA (thenar, 1DI and mid-palm) was demonstrated on the left side. (Fig. 1B, Supplementary Fig. 2 – Appendix 2.)

3.3. Case 3

A 59-year-old woman has had numbness along the ulnar side of left hand, and weakness (grade IV) and atrophy of the left ulnar-innervated muscles for one year. Electrodiagnostic study showed reduced left ulnar-digit V and left dorsal ulnar cutaneous SNAP amplitudes, reduced left ulnar-ADM distal CMAP amplitude (4.7 mV (reference value >6 mV)) and slow conduction velocities (forearm, 39.6 m/sec; elbow, 23.1 m/sec (reference value >49 m/sec)). Sensory and motor nerve conduction studies of bilateral median and right ulnar nerve were normal. MRI of the elbow showed a 1 cm ganglion cyst, compressing the left ulnar nerve at the elbow. The MGA (hypothenar, 1DI and mid-palm) was demonstrated on the left side. (Fig. 1C, Supplementary Fig. 3 – Appendix 3.)

3.4. Case 4

This was a 25-year-old woman without neurological complaint. She had normal neurological examination. The MGA (1DI and mid-palm) was demonstrated on the right side. (Fig. 1D, Supplementary Fig. 4 – Appendix 4.)

4. Discussion

In MGA, the anastomotic fibers usually destine to innervate normally ulnar-innervated muscles (Rubin and Dimberg, 2010). The most common muscle found by the electrophysiological study is 1DI, followed by ADM (hypothenar) and adductor pollicis (thenar) (Khosrawi et al., 2015; Roy et al., 2016). Combination of those muscles has also been reported (Erdem et al., 2002). Very rarely, the anastomotic fibers may terminate in normally median-innervated muscles (Amoiridis, 1992). In our cases, the electrodiagnostic study revealed the innervation of mid-palm muscles, mainly 1PI and 2DI, by the anastomotic fibers. The 1PI and 2DI are normally ulnar-innervated. Thus, it may be expectable that fibers from MGA may innervate them. However, to the best of our knowledge, 1PI/2DI innervation by MGA has never been shown by the electrophysiological study. It might be because motor nerve conduction study (MNCS) recording from mid-palm is not routinely performed. This technique is employed to assess carpal tunnel syndrome (Boonyapisit et al., 2002; Kaul and Pagel, 2002). In such cases, to compare median and ulnar distal motor latencies, stimuli are delivered to median and ulnar nerves at the wrist, not at the elbow. Consequently, MGA cannot be discovered.

In routine MNCS, MGA may be suspected when the median MNCS generates a larger response with ME stimulation, or the ulnar MNCS generates a smaller response with UE stimulation. In healthy hands, a generally acceptable proximal and distal CMAP amplitude difference that may consider MGA may be at least 1.0 mV or over 25% change (Oh, 1993; Roy et al., 2016; Sander et al., 1997). However, in some cases with minor change of CMAP amplitude, the change of area or morphology should also be taken into consideration. Further electrodiagnostic study must be carried out to find other characteristic features of MGA (Sander et al., 1997).
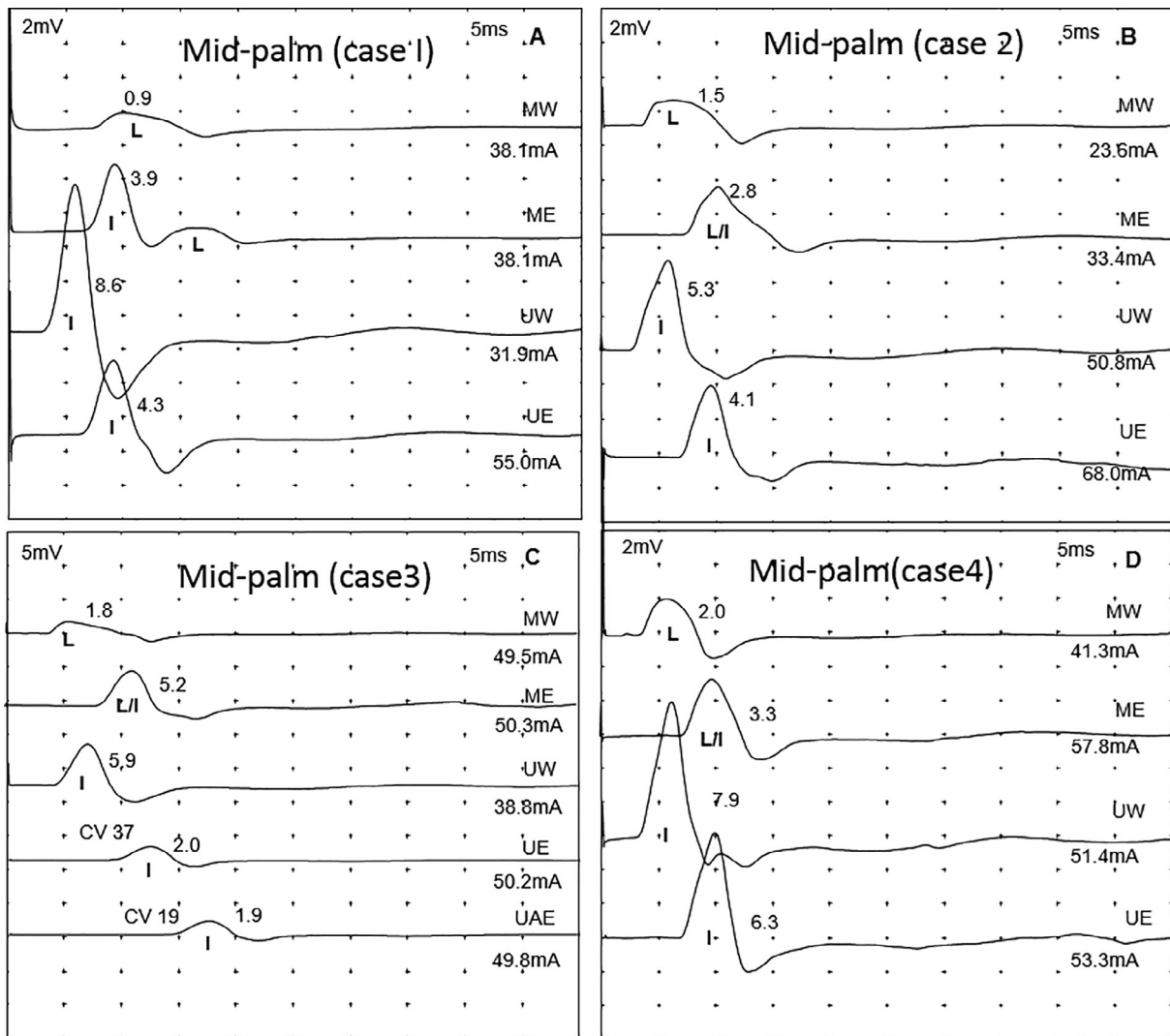
Fig. 1. Median and ulnar motor nerve conduction study with mid-palm recording. Fig. 1A–D showed the findings of cases 1, 2, 3 and 4 respectively. The second tracing in Fig. 1A showed CMAP with two components (bifid); the prior and the latter were responses of 1PI/2DI and 2Lum, respectively. Note that adding up the amplitude of 1PI/2DI responses evoked from ME (3.9 mV) and UE stimulations (4.3 mV) was close to the amplitude evoked from UW stimulation (8.6 mV). The second tracings in Fig. 1B, 1C and 1D showed superimposed responses, contributed by 2Lum and 1PI/2DI. In picture 1B, one may appreciate a triangle-shaped component, contributed by 1PI/2DI, on the top of the response. MW = median nerve stimulation at the wrist, ME = median nerve stimulation at the elbow (antecubital fossa), UW = ulnar nerve stimulation at the wrist, UE = ulnar nerve stimulation at the elbow (3 cm distal to medial humeral epicondyle), UAE = ulnar nerve stimulation above the elbow (7 cm proximal to medial humeral epicondyle), L = response of 2nd lumbrical muscle, I = response of 1st palmar and 2nd dorsal interosseous muscles, the numbers adjacent to each response indicated the CMAP amplitude in millivolt and the conduction velocity (CV) in meter per second. Stimulus intensities were shown in milliampere.

In cases 2, 3 and 4, ME stimulation generated higher-amplitude CMAPs than MW stimulation (Fig. 1B–D). With ME stimulation, the electrical impulses not only descended through the median nerve to activate 2Lum but also propagated via the anastomotic fibers to activate 1PI/2DI. When two potentials occurred simultaneously, they were superimposed with each other, resulting in a higher-amplitude CMAP. In case 1, median distal latency was markedly prolonged due to severe CTS. Such prolongation of median distal latency created morphological change of CMAP evoked by ME stimulation (Fig. 1A). This response comprised two components (bifid shape). The prior was the potential of 1PI/2DI contributed by anastomotic fibers, while the latter was the potential of 2Lum, contributed by the median nerve. The amplitude of the latter component was smaller. This could be explained by the effect of phase cancellation from the following positive deflection of the prior component. Calculated conduction velocity of the median nerve became erroneously negative (−211 m/sec). The explanation was similar to the reason previously described in type 3 MGA with sev-

ere CTS (Rubin and Dimberg, 2010). The electrical impulse from the ME stimulation descended and crossed over via the MGA to the ulnar nerve to supply normal 1PI/2DI without passing the marked delay at the carpal tunnel. But, in contrary to the classic type 3 MGA with CTS, if the amplifier sensitivity was increased, no initial positive deflection would be observed, indicating that this was a true response of 1PI/2DI, not a volume-conducted potential from other distant muscles. The amplitude of this component was also approximately close to the difference of CMAP amplitude evoked from UW and UE stimulations.

In all cases, UE stimulation generated smaller-amplitude CMAPs than UW stimulation (Fig. 1A–D), resembling partial motor conduction block. Besides MGA, this pattern could be found in other demyelinating neuropathies. Thus, such finding must be interpreted carefully. To confirm MGA, a true response of 1PI/2DI must be elicited by ME stimulation. Technical cautions were made that the stimuli delivered to ME did not spread to co-stimulate ulnar nerve. In our study, to avoid overstimulation, stimulus intensity

was gradually increased by less than 5 mA. To be more confident that ulnar nerve was not co-stimulated, the stimulus probe was then slightly moved medially (towards the ulnar side). Without change of stimulus intensity, the obtainable responses were smaller and then disappeared. Case 3 was an example of combination of MGA and ulnar neuropathy. When two conditions coincide, MGA may be unrecognized (Cho et al., 2013). The slow conduction velocities of both forearm and elbow segments and reduction of CMAP amplitude below the elbow might be merely misinterpreted as a result of ulnar neuropathy. However, in this case, ME stimulation generated a superimposed response of 2Lum and 1PI/2DI (Fig. 1C). Subtracting it with CMAP amplitude evoked from MW stimulation (2Lum) represented the 1PI/2DI CMAP contributed by the anastomotic fibers. This was also approximately close to the difference of CMAP amplitude evoked from UW and UE stimulations, which indicated MGA.

In all four cases, MGA was found to be unilateral. Combination with 1DI innervation (type 2 MGA) was found in all cases. Adding the mid-palm recording to conventional CMAP comparison method provides a broader view in terms of innervation of MGA. Whether it increases electrodiagnostic yield is not known. It might be beneficial in some, such as cases with unobtainable thenar, hypothenar or 1DI responses. Further study in a larger number of cases is warranted. MGA frequently makes confusion, not only in neurological examination, but also in the interpretation of nerve conduction study. It may mimic a conduction block of the ulnar nerve, or results in confusing findings of the median MNCS. Awareness of the existence of different electrophysiological patterns of MGA is essential to avoid misinterpretation of the nerve conduction study.

5. Conclusions

A new electrophysiological pattern of MGA with the mid-palm recording was presented. MGA could be diagnosed with this recording. This technique is not complicated and can be added to the electrophysiological investigation for complete studying of the innervated muscles in MGA. Whether it increases electrodiagnostic yield of MGA needs further study.

Competing interests

None.

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Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.cnp.2020.03.001.

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