



Editorial

Electrodiagnosis, a real added value for the evaluation of upper limb paresthesiae and pains



Nerve conduction studies (NCS) and electromyography (EMG) in carpal tunnel syndrome: do they add value? is a frequent question that arises from practitioners and more especially hand surgeons. Four experts in neurophysiology, from all over the world: Masahiro Sonoo, Daniel Menkes, Jeremy Bland, and David Burke (Sonoo et al., 2018), give their point of view on this polemic, but not recent topic (Seror, 1998).

Carpal tunnel syndrome (CTS) is an association of symptoms and signs, related to median nerve compression at wrist. However, some patients may have typical complaints without demonstrable median neuropathy at the wrist (MNW), and others MNW with atypical or no complaints. CTS symptoms and signs had been the source of numerous descriptions, studies and publications (Bland, 2000; D'Arcy and McGee, 2000; de Krom et al., 1990; Graham et al., 2006a; Graham et al., 2006b; Kiernan et al., 1999; Seror, 1998, 1987). Some scores arose from these, with the ambition to recognize and diagnose CTS, without any complementary examination.

CTS patients frequently report symptoms outside the median nerve territory, what factually enlarges the spectrum of the present topic to: *NCS and EMG in upper limbs paresthesiae and pains: do they add value?* In fact in true life, the patients do not come with a flyer where it is written "I have CTS". So, practitioners must evaluate, by history and clinical examination the diverse causes: that are essentially entrapment syndromes, and root disorders, to determine if electrodiagnosis (EDX = NCS + EMG) or cervical spine MRI is the most appropriate tool to confirm their diagnoses. The electromyographer studies also the possible brachial plexus lesions, and some rare pathologies (Lewis Sumner syndrome, hereditary neuropathy with pressure palsy, IgM-anti-Mag neuropathy, chronic inflammatory demyelinating polyneuropathy, amyloidosis neuropathy) (Seror, 2008), when the radiologist will only search a disc-related nerve root impingement. When a patient comes for his first neurophysiological evaluation for CTS symptoms, I believe that one must proceed to a complete EDX, with NCS and EMG. Here I differ from the practice of the authors of the target article. As EMG is the only neurophysiological method to evaluate root lesions, and that numbness and paresthesiae usually involve at least the 3 first digits, EMG should examine at least the biceps and triceps muscles, but also a C8T1 muscle, out of the median nerve distribution (1st interosseous or adductor digiti mini) and the abductor pollicis brevis muscle of the most symptomatic upper limb.

NCS evaluate the function of median nerve at wrist, when ultrasonography (US) shows morphological changes of median nerve

(enlargement), in the same way that thyroid US evaluates the anatomical abnormalities, when blood investigations evaluate the hormonal function (Seror, 2008). EDX will evaluate all diagnoses defined above, with a single examination, when additional US along the ulnar nerve track from hand to Erb point, cervical spine X ray/MRI, and brachial plexus MRI/US are required. The time and the cost of these multiple imaging investigations, will largely exceed those of the EDX that is frequently blame to be expansive, without taking in account, that the practitioner, who will perform the EDX provides clinical added value, linked to his neurological experience, which is usually missing to radiologists. Naturally, US and other imaging examinations of carpal tunnel will never answer the question of the rare neuropathies defined above, which sometimes also escape to the diagnostic sagacity of some neurophysiologists.

CTS being the most frequent cause of nocturnal intermittent paresthesiae of the upper limbs, the simple history offers quite 80% of diagnostic sensitivity (Bland, 2000; Seror, 1998). If history reports that complaints have been much improved by a corticosteroid injection in carpal tunnel the probability of CTS increases. But this attitude do not offer a high specificity, as ulnar lesion at elbow, and sometimes also root disorder have similar history. Of course, one can certainly manage without EDX, when these tests are not available in some countries, but certainly not in industrialised countries such as Japan, USA, Australia, UK or France. Indeed, according with the evidence based medicine, the current practice cannot ignore the evaluation of the functional status of the median nerve before determining the best treatment to use. Failure to comply with this evidence, may expose the practitioner, and especially the surgeon, to a malpractice and a forensic procedure, when no improvement or a complication occur after the surgical decompression.

The most sensitive NCS methods may not be able to demonstrate MNW, because in any pathology, there is a continuum from normal function, to the most altered function (no recordable potential). CTS symptoms may be present with very mild MNW that NCS may miss, and absent in severe cases. CTS symptoms may also occur in normal subjects when a deleterious position is maintained a sufficient time. Kiernan et al. have demonstrated that maximal wrist extension increasing the pressure inside the carpal tunnel, produces a complete conduction block in 49 mn for 6 normal subjects, and in 28 mn for 7 CTS patients. Both controls and patients experienced mild paresthesiae during the wrist extension test, and intense paresthesiae when wrist extension was released,

and conduction block recovered. Pressures varies in normal subjects from 0 to 5 mmHg for neutral position, to 30–40 mmHg for stress position and from 20 to 40 mmHg to 80–100 mmHg in CTS patients (Gelberman et al., 1981; Kuhlmann et al., 1978). It is only during the night, that these positions are maintained for a long period, what explains why CTS complaints are maximal in the last part of the night or at awakening. This is certainly related to the longer duration of paradoxical sleep phases, at the end of the night; phase which is characterized by a very low reactivity to external stimuli, and very low muscular tonus. Then, one can find rare cases mimicking true CTS, with no permanent MNW, what I call “positional or functional CTS”, and at the opposite of the spectrum, cases with severe MNW who are complaining only since 1 or 2 months. In these positional CTS, the possibility of “central sensitization” may be raised.

If the median nerve function doesn't determine alone the choice of treatment, it seems evident, that different methods should be proposed to a CTS with a mild MNW without axonal loss, and a severe MNW with 95% or more axonal loss. Only NCS can evaluate the axonal loss and conduction block of median nerve fibers. This functional evaluation is very important, as there is a poor correlation between the severity of clinical symptoms, signs or scores, except for thenar atrophy, and the severity of the MNW assessed by EDX. One can only agree performing NCS before invasive treatment, but another question arises: what is the right delay between EDX and surgery, to provide a good benchmark for post-surgical evaluation, when a poor result or an aggravation of the complaints occur after surgical decompression? The best would be less than 1 month, but less than 3–6 months seems to be an acceptable compromise. In fact, the longer is the delay between EDX and surgical procedure, the more EDX parameters may worsen, and then can mask the post-surgical neurophysiological improvement. In my practice this is not infrequent when the patient has experienced continuous, worsening of his complaints despite one or 2 corticosteroid injections along these 6–12 months.

On the whole, as you have understood our eminent colleagues have done a difficult exercise of synthesis that will always find alternative opinions for some details, but considering the heart of the matter, their highlights and conclusions are in line with the common practice, and actual recommendations.

Conflict of interest

The author has no conflict of interest to disclose.

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