A neurophysiological grading scale for carpal tunnel syndrome

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SHORT REPORT

ABSTRACT: Different ways of expressing the severity of carpal tunnel syndrome (CTS) are found in the existing literature and in clinical records. This paper documents the distribution of patients on a scale based upon the nerve conduction study findings, which are largely independent of the exact normal values used in any given laboratory and demonstrate a highly significant linear relationship between the neurophysiological grading and a numerical score derived from the clinical history. Patients with more characteristic stories of CTS generally have higher neurophysiological grades. The scale is as follows: normal (grade 0); very mild (grade 1), CTS demonstrable only with most sensitive tests; mild (grade 2), sensory nerve conduction velocity slow on finger/wrist measurement, normal terminal motor latency; moderate (grade 3), sensory potential preserved with motor slowing, distal motor latency to abductor pollicis brevis (APB) < 6.5 ms; severe (grade 4), sensory potentials absent but motor response preserved, distal motor latency to APB < 6.5 ms; very severe (grade 5), terminal latency to APB > 6.5 ms; extremely severe (grade 6), sensory and motor potentials effectively unrecordable (surface motor potential from APB < 0.2 mV amplitude).

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A NEUROPHYSIOLOGICAL GRADING SCALE FOR CARPAL TUNNEL SYNDROME

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Because many neurophysiological approaches exist for assessing median nerve function, there is no widely accepted means of grading the severity of neurophysiological changes in carpal tunnel syndrome (CTS). Many surgical series merely categorize the nerve conduction studies as normal or abnormal. Campbell[4] divided patients into four groups by the degree of prolongation of the median terminal latency. Some authors[10] grade the severity of changes in each test modality separately. Stevens[24] suggests three grades based on the sensory nerve action potential (SNAP) and motor conduction findings. Others have used a single neurophysiological measurement—either sensory[8,11,23] or motor[12,26]—as an indicator of severity. Authors seeking to identify patients with "severe" CTS have used a surface motor potential amplitude of <2.0 mV,[9] an absent median SNAP,[17] or the presence of denervation potentials in the thenar eminence on needle electromyography. Combined clinical and neurophysiological severity scales[18] and purely clinical severity scales based on either subjective questionnaires,[16] physical findings such as the length of time to obtain a positive Phalen test,[5] or symptoms and examination findings together[6] have also been suggested, as have other tests such as magnetic resonance imaging[2] or ultrasound.[15]

In 1997, Padua et al.[20] published a grading scheme very similar to that already in use in Canterbury, the difference being that their severe grade corresponds to a combination of two of the grades used here. More recently, a study correlating their scale with clinical variables has been published.[21] They used a clinical severity scale denoted "Hi-Ob" and based upon a combination of historical and examination findings, and also a symptom severity and functional status scale[16] and were able to show significant correlations between these scales in 740 patients with clinical CTS.

A widely accepted severity scale for the neurophysiological changes of median nerve entrapment

Abbreviations: APB, abductor pollicis brevis; CTS, carpal tunnel syndrome; SNAP, sensory nerve action potential

Key words: carpal tunnel syndrome; clinical history; nerve conduction studies; severity

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at the wrist would greatly facilitate comparison of the severity of the disease in patient groups seen in different places, and this paper provides further evidence of the validity of a scale of the form suggested by Padua et al.

MATERIALS AND METHODS

Since 1990, data have been collected on patients referred to the neurophysiology department in Canterbury with a suspected diagnosis of CTS. Although values for motor and sensory conduction are included in the electrodiagnostic report, the description of overall findings usually includes an indication of severity such as "mild" or "severe." In an attempt to make the use of these terms more consistent, a numerical scale based upon certain widely accepted assumptions was introduced. First, sensory abnormalities precede motor abnormalities. Second, an absent response is worse than a recordable one. Third, slower conduction velocities are worse than faster ones, and finally, the index finger sensory potential is less sensitive than a variety of more recently devised tests. Grade 0 then denotes no neurophysiological abnormality; grade 1, very mild CTS, detected only in two sensitive tests (e.g., inching, palm/wrist median/ulnar comparison, ring finger "double peak"); grade 2, mild CTS (orthodromic sensory conduction velocity from index finger to wrist < 40 m/s with motor terminal latency from wrist to abductor pollicis brevis [APB] < 4.5 ms); grade 3, moderately severe CTS (motor terminal latency > 4.5 ms and < 6.5 ms with preserved index finger SNAP); grade 4, severe CTS (motor terminal latency > 4.5 ms and < 6.5 ms with absent SNAP); grade 5, very severe CTS (motor terminal latency > 6.5 ms); and grade 6, extremely severe CTS (surface motor potential from APB < 0.2 mV, peak-to-peak).

The choice of a motor terminal latency measurement of 6.5 ms to divide grade 4 from grade 5 was arbitrary and based upon a personal impression that most patients with values exceeding 6.5 ms had absent median SNAPs. During the last 9 years a variety of sensitive tests have been used. We find orthodromic sensory conduction from the ring finger[27] and comparison of the motor terminal latencies from the median and ulnar nerves at the wrist to the second lumbrical and interossei[22] to be two of the most reliable. A practical definition of an "absent" motor response was adopted when no peak-to-peak signal greater than 0.2 mV could be identified.

Data have also been collected on the clinical symptoms of these patients. A single-page questionnaire lists questions about features of the history in multiple-choice format, and the answers have been used to derive a logistic-regression model of this simplified clinical history. In generating this model, only the presence or absence of any neurophysiological evidence of CTS was used as the dependent variable, so the resulting probability estimate contains no embedded prior knowledge of the neurophysiological severity. The features of the history used in deriving the symptom score and the weights attached to them are shown in Table 1. This statistical model correctly predicts the finding of abnormal median nerve conduction at the wrist in 80% of CTS cases, although in this population it also has an appreciable false-positive rate (submitted for publication). To test whether the neurophysiological grading has any relationship to the clinical history, the symptom scores were compared against the neurophysiological grade. As CTS is frequently bilateral, with unequal involvement of the two hands, the hand with the worse neurophysiological grading was used for comparison with the symptom score.

RESULTS

Since the introduction of the scale, 8501 patients have been tested for CTS. The numbers in each grade were: grade 0, 3629; grade 1, 684; grade 2, 944; grade 3, 1359; grade 4, 568; grade 5, 930; and grade 6, 387. The relationship between symptom score and neurophysiological severity score is shown in Figure 1. The linear regression line is highly significant (Pearson $r = 0.4728$; $P < 0.0001$) though within any given neurophysiological grade, the range of symptom scores is wide. Comparison of each pair of adjacent groups (e.g., grade 0 versus grade 1, grade 1 versus grade 2, etc.) with Student's *t*-tests shows that

Table 1. Weights used in the symptom score.

Feature	Weight
Constant term in regression equation	−3.3817
Nocturnal awakening	0.5584
Morning symptoms	0.5135
Symptoms while driving	0.2709
Relief from shaking hand when awakened	0.4795
Distribution of symptoms	
Symptoms mainly in thumb, index, and middle fingers	0.9165
Symptoms mainly in little and ring fingers	−0.8625
Symptoms mainly in middle and ring fingers	0.8797
Symptoms affecting all five fingers	0.3751
Ambidextrous	−0.6880
Benefit from splinting	0.4664
Duration of symptoms >1 year	0.2113
Laterality of symptoms	
Symptoms worse on right side	−0.3667
Symptoms worse on left side	−0.3804
Age (per year)	0.0408

each group is symptomatically distinct from the next, with $P < 0.05$ in all cases.

DISCUSSION

Only very occasionally did we find a patient in whom the combination of findings— such as a motor terminal latency > 6.5 ms with a normal SNAP (coded as grade 5) or an absent SNAP with normal motor conduction (coded as grade 2)—made the correct grading uncertain. A comparison of this patient population with that recently described by Padua et al.[21] is interesting. The most striking difference is in the proportion of patients with normal nerve conduction studies, 5.1% of hands in the Italian series, 43% of patients, and 56.1% of hands in the current data. This probably results from a difference in referral patterns. The patients studied in Italy were all clinically diagnosed as CTS, whereas we encourage referral of any patient in whom the diagnosis is considered even a remote possibility. Many of the patients without neurophysiological evidence of CTS in this series have other, obvious explanations for their symptoms. As the grading schemes are so similar, it is easy to compare these two series by combining Canterbury grades 4 and 5, and there is a trend towards more severe disease in Canterbury. The Canterbury population is slightly older, mean age 54 years compared with 51 years, and this may partially account for the difference in disease severity. Padua et al. showed in their data a positive correlation between age and severity of nerve conduction abnormality, noted also by other authors.[14,25]

Although there are theoretical reasons, stated earlier, for accepting that the ranking of severity by this method is valid, i.e., that higher grades indicate worse nerve function than do lower grades, it is useful to compare it against another measure. The comparison with symptom score reveals a strong linear relationship and shows that the neurophysiological ranking does correspond to a clinical variable, in agreement with other studies.[21,28]

Use of a single measured value only as a proxy for overall severity is problematical. Studies that are sensitive to mild CTS may yield unmeasurable data in more severe cases, making statistical analysis difficult. Felsenthal[8] had to exclude 20 of 82 CTS hands from a study of the sensitivity of another neurophysiological technique for this reason, clearly biasing the sample. Measurements that allow comparison of severity in more marked cases, such as motor terminal latency to APB, may not detect mild CTS at all. The two must be combined in order to cover the entire range of severity. A scale such as this allows the inclusion of all patients in a study while retaining a numerical indication of relative severity.

Neurophysiological grading may have prognostic value. Padua et al.[19] attempted to relate the grading to the outcome of surgical decompression, but as only 37 hands in 33 patients were studied in total, there was little power to show differences. Nevertheless, the authors concluded that nerve conduction studies were of prognostic value despite several papers in the literature claiming the contrary.[1,3,7,13] Their 1997 paper classified 500 cases but made no attempt to relate the grading to outcome.[20] Further studies are needed.

For general use, the precise values used to define abnormal sensory and motor conduction in grades 2, 3, and 4 may be taken as whatever the individual laboratory considers to be abnormal for that test. The scale can then be stated as: grade 0, no abnormality; grade 1, CTS demonstrable only with most sensitive tests; grade 2, sensory conduction slow on finger/wrist measurement, normal terminal motor latency; grade 3, SNAP preserved with motor slowing, distal motor latency to APB < 6.5 ms; grade 4, SNAP absent but motor response preserved, distal motor latency to APB < 6.5ms; grade 5, terminal latency to APB > 6.5 ms; and grade 6, sensory and motor potentials effectively unrecordable.

Apart from the distinction between grades 4 and 5, which remains dependent on a single absolute value, this scale is then independent of the actual normal values used in a given laboratory. The measurements required to characterize a patient using this scale, above and beyond whatever sensitive tests a department chooses to use, are simple to perform.

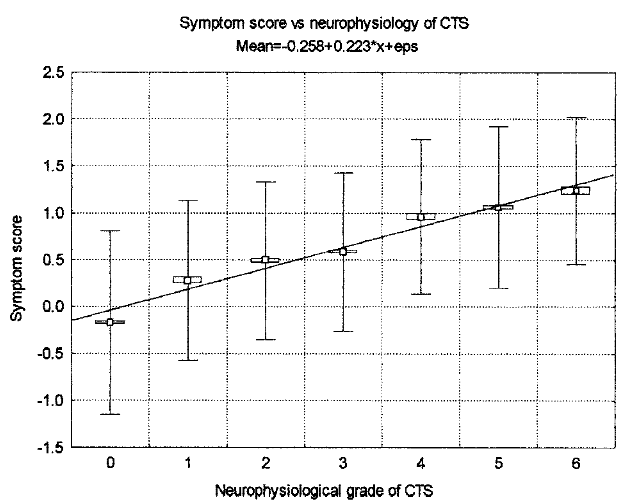

FIGURE 1. Relationship of symptom score to neurophysiological grade. Open circle represents mean value, bar line shows standard deviation, and open box shows standard error.

It would be of considerable assistance to anyone attempting to compare different studies on CTS if authors who use nerve conduction studies as a quantitative measure of severity would adopt a scale of this form.

REFERENCES

1. Al-Qattan MM, Bowen V, Manktelow RT. Factors associated with poor outcome following primary carpal tunnel release in non-diabetic patients. J Hand Surg 1994;19B:622–625.
2. Andre V, Zagnoli F, Andre M, Le Dreff P, Bellard S, Garcia JF. Clinical, electrophysiological and MRI correlations in carpal tunnel syndrome. J Radiol 1999;80:721–726.
3. Braun R, Jackson W. Electrical studies as a prognostic factor in the surgical treatment of carpal tunnel syndrome. J Hand Surg (Am) 1994;19:893–900.
4. Campbell EDR. The carpal tunnel syndrome—investigation and assessment of treatment. Proc R Soc Med 1962;55: 401–405.
5. Carneiro RS. Carpal tunnel syndrome: the cause dictates the treatment. Cleve Clin J Med 1999;66:159–164.
6. Chang M-H, Liao K-K, Chang S-P, Kong K-W, Cheung S-C. Proximal slowing in carpal tunnel syndrome resulting from either conduction block or retrograde degeneration. J Neurol 1993;240:287–290.
7. Concannon MJ, Gainor B, Petroski GF, Puckett CL. The predictive value of electrodiagnostic studies in carpal tunnel syndrome. Plast Reconstr Surg 1997;100:1452–1458.
8. Felsenthal G. Comparison of evoked potentials in the same hand in normal subjects and in patients with the carpal tunnel syndrome. Am J Phys Med 1978;57:228–232.
9. Finestone HM, Woodbury GM, Collavini T, Marchuk Y, Maryniak O. Severe carpal tunnel syndrome: clinical and electrodiagnostic outcome of surgical and conservative treatment. Muscle Nerve 1996;19:237–239.
10. Fuchs PC, Nathan PA, Myers LD. Synovial histology in carpal tunnel syndrome. J Hand Surg 1991;16A:753–758.
11. Ghavanini MRA, Haghighat M. Carpal tunnel syndrome: reappraisal of five clinical tests. Electromyogr Clin Neurophysiol 1998;38:437–441.
12. Glidden A, Bauer RB. Carpal tunnel syndrome: a comparison of nerve conduction studies before and after treatment. Harper Hospital Bulletin 1968;26:221–230.
13. Glowacki KA, Breen CJ, Sachar K, Weiss A-PC. Electrodiagnostic testing and carpal tunnel release outcome. J Hand Surg 1996;21A:117–122.
14. Kouyoumdjian JA. Carpal tunnel syndrome. Age, nerve conduction severity and duration of symptomatology. Arq Neuropsiquiatr 1999;57:382–386.
15. Lee D, van Holsbeeck MT, Janevski PK, Ganos DL, Ditmars DM, Darian VB. Diagnosis of carpal tunnel syndrome. Ultrasound versus electromyography. Radiol Clin North Am 1999;37:859–872.
16. Levine DW, Simmons BP, Koris MJ, Daltroy LH, Hohl GG, Fossel AH, Katz JN. A self-administered instrument for the assessment of symptom severity and functional status in carpal tunnel syndrome. J Bone Joint Surg 1993;75A:1585–1592.
17. Nolan WB, Alkaitis D, Glickel SZ, Snow S. Results of treatment of severe carpal tunnel syndrome. J Hand Surg 1992;17A: 1020–1023.
18. O'Duffy JD, Randall RV, MacCarty CS. Carpal tunnel syndrome in acromegaly—a sign of endocrine overactivity. Ann Intern Med 1973;78:379–383.
19. Padua L, LoMonaco M, Aulisa L, Tamburelli F, Valente EM, Padua R, Gregori B, Tonali P. Surgical prognosis in carpal tunnel syndrome: usefulness of a preoperative neurophysiological assessment. Acta Neurol Scand 1996;94:343–346.
20. Padua L, LoMonaco M, Gregori B, Valente EM, Padua R, Tonali P. Neurophysiological classification and sensitivity in 500 carpal tunnel sydrome hands. Acta Neurol Scand 1997;96:211–217.
21. Padua L, Padua R, Lo Monaco M, Aprile I, Tonali P. Multiperspective assessment of carpal tunnel syndrome. Neurology 1999;53:1654–1659.
22. Preston D, Logigian EL. Lumbrical and interossei recording in carpal tunnel syndrome. Muscle Nerve 1992;15:1253–1257.
23. Seror P. A La recheche d'une correlation electro-clinique au cours syndrome du canal carpien. Rev Rhum Mal Osteoartic 1987;54:643–648.
24. Stevens JC. The electrodiagnosis of carpal tunnel syndrome. Muscle Nerve 1997;20:1477–1486.
25. Stevens JC, Smith BE, Weaver AL, Bosch EP, Deen HGJ, Wilkens JA. Symptoms of 100 patients with electromyographically verified carpal tunnel syndrome. Muscle Nerve 1999;22: 1448–1456.
26. Stoehr M, Petruch F, Scheglmann K, Schilling K. Retrograde changes of nerve fibres with the carpal tunnel syndrome. J Neurol 1978;218:287–292.
27. Uncini A, Lange DJ, Solomon M, Soliven B, Meer J, Lovelace RE. Ring finger testing in carpal tunnel syndrome: a comparative study of diagnostic utility. Muscle Nerve 1989;12:735–741.
28. You H, Simmons Z, Freivalds A, Kothari MJ, Naidu SH. Relationships between clinical symptom severity scales and nerve conduction measures in carpal tunnel syndrome. Muscle Nerve 1999;22:497–501.