The role of different neurophysiological tests in the differential diagnosis of diabetic axonal neuropathy and lumbosacral radiculopathy

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ABSTRACT

الأهداف: تقييم دور الفحص الفسيولوجية العصبية في التشخيص لتمييزي بين اعتلال الأعصاب المحورية السكرية (DAN)، واعتلال الجذور العصبية القطنية العجزية (LSR).

الطريقة: أجريت هذه الدراسة في مستشفى الكاظمية التعليمي – بغداد – العراق، خلال الفترة مابين يوليو 2006م وحتى فبراير 2007م. تم فحص 27 شخصا سليما، 44 مريضاً بداء السكري، و 36 مريضا مصاباً باعتلال الجذور العصبية القطنية العجزية (LSR). قمنا بقياس مستوى خضاب الدم المرتبط بالغليكوسيل (HbA1c)، أخذت صورة إشعاعية، وتصوير بالرنين المغنطيسي (MRI) للمنطقة القطنية العجزية (LSR)، ومختلف الفحوص الكهروفسيولوجية.

النتائج: كان مدى جهد الفعل للعصب الرَّبلي الحسِّي (SNAP) وسعة الموجة لجهد فعل العصب الرَّبلي الحسِّي على سعة الموجة لجهد فعل العصب الكعبري الحسِّي (SRAR) مُخْتَزَلاً في 56.3% و 71.8% على التوالي في المرضى المصابين بداء السكري، ولكن ليس في مجموعة اعتلال الجذور العصبية القطنية العجزية (LSR). مدى جهد الفعل العضلي المُرَكب للعصب الشظوي العام (CMAP) كان منخفضاً في المرضى المصابين بداء السكري DAN–70.45% مقابل 35.5% عند مرضى اعتلال الجذور العصبية القطنية العجزية (LSR)، استطالة فترة كمون موجة ف الدنيا Fmin–56.8% عند مرضى داء السكري (DAN) مقابل 32.25% عند مرضى اعتلال الجذور العصبية القطنية العجزية (LSR)، استمرارية موجة ف (Fp) كانت منخفضة في 72.7% عند مرضى داء السكري (DAN) مقابل 45.2% عند مرضى اعتلال الجذور العصبية القطنية (LSR). من ناحية أخرى كان الفرق في سرعة توصيل موجة ف الدنيا والقصوى Fc–71% عند مرضى اعتلال الجذور العصبية القطنية (LSR) مقابل 11.4% عند مرضى داء السكري (DAN).

خاتمة: تبين أن سعة الموجة لجهد فعل العصب الرَّبلي الحسِّي (SNAP) أكثر أهمية من سعة الموجة لجهد فعل العصب الكعبري الحسِّي (SNAP) لوحده في التفريق في تشخص الحالتين في المجموعتين. يبدوا أن الاعتلال الشظوي في سرعة توصيل موجة ف الدنيا والقصوى (Fc) و (Fp) اختبار ذو أهمية كبيرة للتمييز بين مرضى اعتلال الأعصاب المحورية السكرية (DAN)، ومرضى اعتلال الجذور العصبية القطنية العجزية (LSR) على التوالي.

Objective: To evaluate the role of different neurophysiological tests in the differential diagnosis of diabetic axonal neuropathy (DAN) and lumbosacral radiculopathy (LSR).

Methods: This study was conducted at Al-Kadhimiya Teaching Hospital, Baghdad, Iraq, from July 2006 to February 2007. Twenty-seven healthy subjects, 44 type 2 diabetics, and 36 LSR patients were studied. The HbA1c level, plain x-ray, and MRI of the lumbosacral region and different electrophysiological tests were assessed.

Results: The sural sensory nerve action potential (SNAP) amplitude values were reduced in 56.3%, and the sural/radial amplitude ratio (SRAR) values were reduced in 71.8% in the diabetic patients, but not in the LSR group. The peroneal compound muscle action potential (CMAP) amplitude was low in 70.45% DAN patients versus 35.5% LSR patients. Peroneal F-minimum (Fmin) values were prolonged in 56.8% DAN versus 32.25% LSR patients. The F-persistence (Fp) values were low in 72.7% of DAN, versus 45.2% of LSR patients. However, the F-chronodispersion (Fc) was abnormal in 71% of LSR versus 11.4% of DAN patients.

Conclusion: The SRAR was found to be more significant than the sural SNAP amplitude alone in the differential diagnosis of the 2 groups. Abnormal peroneal Fc and Fp seems to be valuable tests in the detection of LSR and DAN patients.

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Long-term damage, dysfunction, and failure of various organs especially eyes, kidney, nerves, heart, and blood vessels have been documented to occur in diabetes mellitus (DM).[1] The neuropathies are among the most common of the long-term complications of diabetes, affecting up to 50% of patients.[2] The patients usually complain of burning pain paraesthesia and hyperesthesia, and experience symmetrical sensory loss to all modalities in a stocking distribution. The ankle reflexes are usually reduced or absent, and knee reflexes may also be reduced in some cases.[3] Lumbosacral radiculopathy (LSR), is one of the major causes of acute and chronic low back pain. It results from lumbosacral nerve root compression and/or inflammation that has progressed enough to cause development of neurological symptoms in the areas supplied by the affected nerve root(s).[4] The lumbosacral region is the most common spinal area afflicted with root disease.[5] The typical radicular pain is a well-defined stabbing or shooting pain in the buttocks that radiates into the lower extremities.[6] In most LSR patients, the pain radiation extends to the ankle level and symptoms distal to this are mostly numbness.[7] On many occasions, people suffer from LSR and diabetic axonal neuropathy (DAN) simultaneously and the clinical differentiation is often difficult between them, as the 2 conditions may present with numbness and tingling of feet, and helpful clinical clues such as back pain, proximal weakness, or radiating pain into the legs may be absent.[8] In such cases, the electromyographer looks for the presence of paraspinal or proximal muscle denervation and normal sensory conduction studies to indicate radicular disease, and reduced distal sensory and/or motor amplitudes on nerve conduction studies, and a distal to proximal gradient of reinnervation or denervation on electromyography to indicate DAN.[9,10] Unfortunately, these criteria are often inadequate, because in radicular disease, and in elderly patients, the amplitudes of sensory nerve action potentials may be reduced as an age-related phenomenon, and paraspinal denervation may be absent or so widespread as to be of uncertain relevance.[10] However, in peripheral neuropathy, including DAN; distal sensory and motor amplitudes might remain within the normal range despite being reduced for that individual.[11] More importantly, despite the huge available data concerning the neurophysiological assessment in the 2 disease entities, still there were conflicting results. Thus, the intention of the present study is to investigate the role of various neurophysiological tests in differentiating between DAN and LSR and to evaluate the limits of these various tests in a particular patient group.

Methods. This study was conducted at Al-Kadhimiya Teaching Hospital, Baghdad, Iraq, from July 2006 to February 2007. The approval of each participant in the study was taken prior to the study. Twenty-seven normal healthy volunteers were randomly selected and included in the present study. They were 18 males and 9 females with an age range from 40-66 years (mean ± SD = 48.11 ± 9.83 years). The patients were selected from those attending the Diabetic Clinic and the Clinic of Rheumatology and Physical Rehabilitation at Al-Kadhimiya Teaching Hospital, Baghdad. They were divided into 2 groups. The first group comprised 44 type 2 DM patients (26 females and 18 males) with an age ranging from 43-65 years (mean ± SD = 51.56 ± 6.01 years). The duration of DM varied from one year up to 16 years (mean ± SD=6.64 ± 3.34 years). The following inclusion and exclusion criteria were considered in the selection of diabetic patients; symptoms of numbness, tingling or burning sensation of feet by history, no history of low back pain, no history of neurological claudication, no previous history of a known neurologic disorder, uremia, vitamin B12 deficiency or alcoholism, and no features of LSR on plain x-ray study. The second patient group comprised 36 LSR patients (20 males and 16 females) with an age ranging from 39-63 years (mean ± SD = 49.9 ± 9.82). The duration of illness varied from an acute insult of 3 weeks duration up to 10 years of chronic history of low backache radiating to the lower extremities. The following were the inclusion and exclusion criteria for the subjects; low back pain with or without neurological claudication, ability to walk without crutches, plain x-ray study of the lumbosacral region showing features of LSR (decreased intervertebral space and straightening of the vertebrae), MRI showing features of disc prolapse or spinal canal stenosis, no history of DM, alcoholism, vitamin B12 deficiency, S1 radiculopathy, or any other neurological disease that affects nerve function.

Clinical examination. For DM patients, the lower limbs were inspected for the presence of ulcers and muscle atrophy. The Achilles tendon reflex was evaluated. The sensation of touch, pinprick, vibration, and proprioception sensations were tested in the proximal and distal parts of the limbs according to the dermatological distribution. For LSR patients, the back was inspected to assess posture while the patient was standing. Any scoliosis tilt was assessed. The lumbar spine was palpated to identify any tenderness and to evaluate the contour of the spine. Touch and pinprick sensations were assessed. Knee and ankle joint reflexes were evaluated. Straight leg rising test was performed.

Laboratory tests. All the LSR patients were tested to exclude DM by measuring fasting blood sugar (FBS) using the enzymatic oxidation test method (the kit labeled Glucose GOD/PAP, Cat. no. GL 364, Randox

Laboratories Ltd., 55 Diamond Road, Crumlin, Co. Antrim, UK) and glycated hemoglobin (HbA1c) using high pressure liquid chromatography (Variant Hemoglobin Testing System, Bio-Rad, Hercules, CA, USA). The normal range was calculated to be 4.1-6.5%.

Magnetic resonance imaging. Axial and sagittal sections of the lumbosacral spine were taken for LSR patients in the MRI department in Al-Kadhimiya Teaching Hospital using Philips MRI machine (Mod. Gyroscan NT/ Compact Plus R6.2, Philips Manufacturing Co., Ref. No. 5545, Eindhoven, Holland).

Nerve conduction studies. Nerve conduction study (NCS) measurements were performed with Counterpoint 4-channels electromyography machine, serial No. 169, Dantec, Denmark. In the LSR patients, the symptomatic limb(s) was usually examined. The following parameters were studied: sensory nerve conduction velocity (SNCV) for both the sural and superficial radial nerves, and the ratio between the sural and radial sensory nerve action potential (SNAP) amplitudes was calculated (sural/radial amplitude ratio, [SRAR]). The recording and stimulating electrodes were manipulated for both nerves to obtain the highest amplitude potential with a flat baseline. Ratios were calculated by dividing the highest sural amplitude obtained by the highest radial amplitude obtained.[12] In addition, the motor nerve conduction velocity (MNCV), the compound muscle action potential (CMAP) and different F wave parameters including F-minimum (Fmin), F-mean, F-maximum (Fmax), F-chronodispersion (Fc) and F-persistence (Fp) for the common peroneal nerve were studied. To record the F-wave, 20 records from each run were obtained. The mean of 3 runs was taken for consideration. In the control group, 27 of each of sural, radial, and common peroneal nerves were studied, whereas, 44 sural and radial nerves were studied in the DAN patients and 36 common peroneal nerves were studied in the LSR patients. The room temperature was maintained around 25°C during the examination. Nerve conduction tests were carried out according to the standard methods.[13]

The results were expressed as mean ± standard deviation (SD). The student (t) test was used to evaluate the differences between any 2 groups. The percent of abnormal values was calculated as above or below (mean±2.5 SD) the normal values for the control group. The probability limit (p-value) of less than 0.05 was considered to be statistically significant for the results under study. The SPSS software was used for statistical analysis.

Results. Fasting blood sugar was normal in all of the control and LSR subjects, while only 6 of the DM patients had normal FBS. The HbA1c level was normal in only 3 DM patients (below 6.5%). On MRI, reduction in the height of the lumbar vertebrae with central disc bulging was detected in 66.7%, lateral disc bulging was seen in 20%, intervertebral foramina compression was seen in 33.3%, and canal narrowing was observed in 13.3% of the LSR cases. Different degrees of lateral recesses and thecal sac indentation were observed in the cases.

Neurophysiological data. 1. The DM patients versus control subjects. The radial SNCV (p=0.0073) and its SNAP (p=0.0001) were significantly reduced. Similarly, sural SNCV (p=0.0091) and its SNAP (p=0.0238) were significantly reduced. Moreover, the SRAR was also reduced (p=0.0004) in the DM patients when compared with the values of the control group (**Table 1**). Absent sural response was observed in 15 (34.1%) patients, 2 of them were diabetics for <5 years duration, while the radial response was absent in only 3 (6.8%) patients. The peroneal Fmin, Fmax, and F-mean were significantly prolonged (p=0.0001) in the diabetic patients. Similarly, the Fc was also prolonged, but to a significant level (p=0.0073), while MNCV (p=0.0079), CMAP amplitude (p=0.0063), and Fp (p=0.0227) were significantly decreased in comparison with the control group (**Table 1**). The F wave response were absent in 4 (10%) of the nerves under study, the patients were diabetics for >5 years.

2. LSR patients versus control subjects. No significant difference was observed in the values of SNCV, and SNAP amplitude of the sural and radial nerves (**Table 1**). None of the LSR patients had absent sural and/or radial responses. Significantly reduced peroneal CMAP amplitude (p=0.0064), and significantly higher Fmin, Fmax, F-mean, and Fc values (p=0.0001) were found in LSR patients; except Fp, which was reduced as compared to the control subjects (p=0.0001). Conversely, the peroneal MNCV was not significantly different as compared to the control subjects (**Table 1**).

3. The DM versus LSR patients. The values of the SNCV (p=0.03), SNAP (p=0.0001) of sural nerve, and the SRAR (p=0.0001) were significantly reduced in the DM patients when compared to LSR patients. Significantly lower peroneal Fp (p=0.0038), and Fc (p=0.0001) were detected in the DM patients when compared with LSR patients. However, the radial SNCV and SNAP, peroneal MNCV and CMAP, Fmin, Fmax, and Fmean were different but not to a significant level in the 2 patient groups (**Table 1**).

By reviewing the above data and comparing the values of different parameters with the control group, the following results were obtained: In the DM group, 18 sural nerves (40.9%) showed reduced SNCV, and 8 (18.2%) showed reduced SNAP amplitude; these results were remarkably different from the LSR group values. In addition, similar differences were also evident in radial

Table 1 - Illustrates the values of the SNCV and SNAP amplitude of both sural and radial nerves, SRAR, and the MNCV, and F wave parameters of the peroneal nerve for the control subjects, DM, and LSR patients.

The parameter	Control group (n=27)	DM patients (n=44)	LSR patients (n=36)
		Mean±SD	
Sural SNCV (m/s)	57.14±4.87	42.43±7.72	50.95±5.65
Sural SNAP amplitude(μV)	8.8±2.43	3.17±1.06	9.3±2.3
Radial SNCV (m/s)	61±3.93	53.13±6.13	58.87±4.54
Radial SNAP amplitude(μV)	9.79±1.72	7.32±2.49	9.75±2.78
SRAR	0.9±0.16	0.38±0.09	1.01±0.31
MNCV (m/s)	47.96±4.15	41.55±6.54	46.34±5.2
CMAP amplitude (mV)	6.92±1.13	3.28±1.81	4.78±1.83
Peroneal Fmin (ms)	46.38±1.91	53.21±4.27	49.27±3.93
Peroneal Fmax (ms)	49.23±1.6	56.77±4.78	56.86±5.6
Peroneal F-mean (ms)	47.94±1.84	55.1±4.55	52.48±4.33
Peroneal Fp (%)	71.11±9.93	25.27±14.43	44.67±22.2
Peroneal Fc (ms)	2.84±1.06	3.56±1.68	7.59±3.39

SNCV - sensory nerve conduction velocity, SNAP - sensory nerve action potential, SRAR - sural/radial amplitude ratio, MNCV - motor nerve conduction velocity, DM - diabetes mellitus, LSR - lumbosacral radiculopathy, CMAP - compound muscle action potential, Fmin - F-minimum, Fmax - F-maximum, Fp - F-persistence, Fc - F-chronodispersion

nerve values; reduced SNCV in 13 (29.5%) and SNAP amplitude in 9 (20.5%). Twenty-three nerves (52.3%) of the DM group had SRAR less than 0.47, which is the lowest normal value in the control subjects. Thirty-one nerves (70.45%) of the DM patients, showed reduced peroneal CMAP amplitude below the lower limit of normal; versus 11 nerves (35.5%) in LSR patients. Increased peroneal Fc was noticed in 22 nerves (71%) of the LSR patients in comparison with only 5 nerves (11.4%) in the DM patients. Furthermore, in DM patients, 32 nerves (72.7%) showed reduced peroneal Fp, a percentage that was larger than 45.2% (14 nerves) of LSR patients. Prolonged peroneal F-mean latency was reported in 27 nerves (61.4%) of the DM in comparison with 16 nerves (51.6%) of LSR patients.

Discussion. Several hundred studies have been carried out to estimate the value of different electrophysiological parameters in the diagnosis and follow up of DAN or LSR. However, little research was conducted to establish the possible differences and the diagnostic yields between these parameters.[8,10] Differentiating DAN and LSR depending on electromyography and routine nerve conduction studies can be clinically challenging, especially when the 2 entities may present simultaneously in older patients.[14]

The DM patients versus control subjects. The SRAR were abnormal in 71.9% of the nerves, whereas 56.3% showed abnormal sural amplitude. This means that a total of 15.6% of the subjects were considered as having no sensory impairment depending on measurement of sural SNAP amplitude alone. There are limited studies dealing with SRAR. In their study, Tamura et al[15] and Turgut et al[16] stated that the SRAR was reduced in DM patients. Similarly, Bromberg and Albers[17] reported relative sparing of the sural sensory response amplitude as compared to the median in patients with acute and chronic inflammatory demyelinating polyneuropathies. Interestingly, their comparison group of patients with diabetic polyneuropathy demonstrated an increased median to sural amplitude ratio, a result consistent with the findings of the current study. The reduced SRAR reported in this study could be attributed to the fact that most axonal polyneuropathies are characterized by a distal to proximal gradient of severity, with the longest nerves of the lower extremities being affected earlier than more proximal upper extremity nerves.[18,19] Thus, an early reduction in sural amplitude relative to radial might be anticipated. It is worthy to state that whenever the SRAR was <0.4 it gave a 90% sensitivity and specificity.[9] This is also found in the present study as 90% of the diabetic patients had SRAR <0.44. The peroneal MNCV was abnormal in over 75% of DM patients, however, this is not absolutely true regarding those who were diabetics for less than 5 years as the peroneal nerve values were significantly different from that of the controls, as 13 nerves (61.9%) exhibited abnormal CMAP amplitude, whereas only one nerve (4.8%) showed reduced MNCV. The MNCV and CMAP amplitude may be normal or show mild abnormalities in the early stages of DM.[20,21] The peroneal MNCV data were normal except in one LSR patient. This is in agreement with the findings of Berger et al.[10] This apparently normal conduction is attributed to the proximal location of the lesion; as both the distal root ganglia and the peripheral processes arising from them

are spared, since degeneration proceeds centrally rather than peripherally. Moreover, the conduction is along surviving fibers that are conducting at their normal rate.[22] The CMAP amplitude was abnormal in 11 (35.5%) of the LSR patients. As stated by Wilbourn and Aminoff,[23] the sole component of the nerve conduction studies that may be affected in LSR is the CMAP amplitude. As it was noticed from the results of this study, the CMAP amplitude of the common peroneal nerve was reduced in 70.45% of diabetics (including those who were diabetic for ≤5 years) versus 35.5% of LSR patients. This finding is almost in agreement with that of Berger et al.[10] The obvious difference in the percentage of reduced CMAP amplitudes between the 2 disease entities could be attributed to the following: - Firstly, according to Sumner,[24] it may be due to the differences in the underlying pathophysiology between DM and LSR as degeneration of large diameter axons, common to most DM, provides an anatomical explanation for the low-amplitude, or even absent CMAP amplitudes. Secondly, although axonal degeneration may also occur in LSR, there often is segmental demyelination with preserved axonal integrity.[25,26] Finally, because most muscles are supplied by more than one nerve root, a substantial number of motor axons may remain intact and contribute to the nearly normal CMAP amplitude in LSR patients.[25] The F wave response parameters of the common peroneal nerves were abnormal in reference to the control group, a finding supported by the results of Adamova et al[8] and Weber.[27] Furthermore, in agreement with Weber,[27] the Fp was the most recorded abnormal parameter for the common peroneal nerve than other parameters (whether we compared all the diabetic nerve values as a whole or only with those of ≤5 years duration).

The LSR patients versus control subjects. Our data showed clear preserved sensory nerve parameters for both upper and lower limbs. According to Wilbourn and Aminoff,[28] the sensory nerve parameters rarely are affected, regardless of whether focal demyelination or axon degeneration has occurred, and even when there is a fixed sensory deficit on clinical examination. For the peroneal nerve values, F wave responses have historically been disappointing in the diagnosis of LSR. This may, partly, reflect a prior reliance on minimal latency.[10] The F-min was reported to be abnormal in 10 (32.25%) of the nerves under study, which was less than 18-65% reported by other researchers.[10,28,29] This could be attributed to: - Firstly, normalization of overall latency may occur when a long segment of normally conducting nerve compensates for impaired conduction across a shorter radicular segment. Secondly, it may also be related to dual root innervation, so that minimal latency reflects conduction through uninvolved roots, and to sparing of enough fast-conducting fibers to provide a normal minimal latency.[10] The significantly abnormal peroneal F-min, F-max and Fc were also recorded.[30,31] In addition, Fc was the most frequent parameter that showed abnormal percentage, even more than F-min; which agreed with other research,[8,26] however, it disagrees with Mebrahtu and Rubin[32] who found the Fc to have no substantial additional value in evaluating LSR over that of F wave latency. The findings of the latter authors were due to the wide range (0.2-23.4 ms) reported from studied LSR patients. Interestingly, in the more localized nerve lesions in LSR, the remaining normal segment dilutes the conduction delay across the much shorter segment. Thus, relatively mild abnormalities over restricted segments may reduce the Fp but minimally alter the F wave latency beyond its inherent variability.[13] This is proven in the current study as the Fp was abnormal in 14 (45.2%) nerves in comparison with 11 (35.5%) of Fmin in LSR patients.

The DM versus LSR patients. The recorded higher Fc percentage in LSR patients (22; 70%) in comparison with the DM patients (5; 11.4%) was also reported by Burger et al.[10] To the best of our knowledge, it was the only study considering this value between such patients groups. On the reverse to Fc, the F-min showed higher abnormal values in diabetic (56.8%) patients than LSR ones (32.25%). Similar findings were noticed by Adamova et al[8] and Burger et al,[10] although the latter group studied the posterior tibial nerve. Regarding the Fp, the DM patients had a higher percent of decreased values versus LSR patients, a finding that contradicts the results of Adamova et al,[8] who found no difference between the 2 patient groups. This contradiction might be due to the differences in the nerves studied and more importantly due to the higher tendency of the peroneal nerve to have decreased Fp than the posterior tibial nerve even in normal subjects.[33]

In conclusion, a simple ratio between sural and radial SNAP amplitudes can be a better indicator of peripheral neuropathy than sural amplitude alone. The CMAP amplitude of the common peroneal nerve seems to have a greater value in the detection of DAN than LSR patients. Almost all F wave parameters were different between the 2 conditions, yet, among all, the Fc is a highly reliable parameter that can aid in supporting the diagnosis of LSR, whereas Fp is a good index for diagnosing DAN. We recommend, the SRAR, and different peroneal F wave parameters to be involved in routine nerve conduction studies. Our future planning is to evaluate diabetic patients with and without radiculopathy to see whether the presence of neuropathy confounds the ability to recognize radiculopathy and vice versa. Fortunately, no study limitations were encountered.

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