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Sympathetic skin response in idiopathic and diabetic carpal tunnel syndrome

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Abstract

Background: In carpal tunnel syndrome (CTS), certain changes were expected in sympathetic skin response (SSR) because median nerve carries postganglionic unmyelinated fibres.
Purpose: To investigate the median and ulnar SSR in idiopathic and diabetic CTS without autonomic dysfunction in hands and to find possible relations with electrophysiological features of these diseases.
Patients and methods: SSRs were elicited by electrical stimulation on the supraorbital nerve and recorded from the median and ulnar territories in the hand from 20 diabetic patients with only CTS (29 hands), 24 idiopathic CTS patients (42 hands) and 13 normal subjects (26 hands). Hands with ulnar neuropathy at the wrist without symptoms and normal hands of unilateral CTS were excluded. In addition to classical parameters and comparative methods, SSR waveform changes and percentile method was used in finding abnormality.
Results: Median SSRs had significant delayed latency compared to ulnar latency in both CTS patients but this was not important clinically (1.38/1.37 s for idiopatic CTS; 1.43/1.36 s for diabetic CTS). Median and ulnar SSR amplitude, area, median/ulnar latency difference, amplitude and area ratio were compared and only median/ulnar latency difference and median/ulnar latency ratio were found different between the three groups. Four idiopathic CTS hands were outside of the limits or absent (9.5%). SSR waveforms were significantly different from normal subjects in CTS patients. P type SSR replaced M type in idiopathic CTS and N type in diabetic CTS.
Conclusions: Findings regarding SSR parameters suggest that unmyelinated C fibers were affected in CTS. These values were not useful because they were too small. Data on SSR were not normally distributed and the percentile method seems to be more convenient for finding any abnormality in clinical practice. Also, SSR waveform analysis could give us valuable data and should add to the SSR examination parameters.

Keywords: Carpal tunnel syndome; Sympathetic skin response; Diabetes mellitus; Waveform; SSR; DM

1. Introduction

Sympathetic skin response (SSR) is a transient change in the electrical skin potential evoked by a variety of internal and external stimuli [1]. The polysynaptic reflex arc includes large myelinated afferent sensory fibres, central relays coordinated in the posterior hypothalamus or upper brainstem reticular formation and an efferent pathway through the spinal cord, sympathetic preganglionic and postganglionic nerve fibres, with sweat glands as effectors [2]. Although it has some limitations, it has been employed to assess autonomic function in neuropathies, especially in diabetic neuropathy [3]. In carpal tunnel syndrome (CTS), certain changes are expected because the median nerve carries postganglionic unmyelinated fibres [4]. Sympathetic skin response itself has no value for detecting CTS [5] but its value for assessing autonomic disturbances in CTS is conflicting [1,6–9]. Some of these

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study designs did not include diabetic patients [7,8] and SSR changes in diabetic CTS are not known. Therefore, the aim of this study was to investigate the median and ulnar side SSR in idiopathic and diabetic CTS patients without polyneuropathy or ulnar neuropathy to find possible alterations in median SSR with related CTS in these patients.

2. Patients and methods

SSRs were recorded from median and the ulnar innervated areas in patients with idiopathic CTS (23 female; 1 male), patients with CTS and diabetes mellitus (18 female; 2 male) and normal subjects (12 female; 1 male). Patients with clinical CTS diagnosis by other physicians or neurologists and confirmed with electrophysiological methods were enrolled in the study and other conditions were excluded. Electrophysiological examinations for CTS were done by different authors but all SSR recordings done by same author (NK). For clinical diagnosis, the AAEM guideline was used [10]: paresthesia of the hand and weakness or clumsiness of the hand provoked or worsened by sleep, sustained hand or arm position or repetitive actions of the hand or wrist. All patients and controls gave written informed consent and the local ethic committee approved the study. Controls and patients who had not been previously diagnosed as diabetic underwent laboratory screening tests including liver function tests, renal function tests, and fasting blood glucose, sedimentation rate, rheumatoid factor, and thyroid function tests. Then, they were defined as idiopathic CTS (mean age 46.7 ± 11.9 years; min. 24 years, max. 70 years), or diabetic CTS (mean age 56.7 ± 9 years; min. 37 years, max. 70 years) by the 1985 World Health Organization criteria [11]. Put simply, this means symptoms of diabetes mellitus plus a random plasma glucose concentration of at least 200 mg/dl, a fasting plasma glucose level of 140 mg/dl or higher (two times), or one of plasma glucose level at 30, 60, 90 min and 2-h plasma glucose level of 200 mg/dl or more during a 75 gram oral glucose tolerance test. Thirteen normal subjects (mean age 41.9 ± 9.8 years; min. 20 years, max. 57 years) were included for upper limits of the SSRs and normal hands of other patients were excluded. Limits for nerve conduction values (mean $\pm$ 3S.D.) were derived previously from 18 normal subjects (10 women, 8 men; mean age 42.6 ± 2.7 years; min. 40 years, max. 50 years) in conditions similar to this research laboratory using a Nihon Kohden EMG machine (Neuropack 2). All CTS patients and controls were questioned and examined for dysautonomic features related to CTS such as swelling of the fingers, dry palms or blanching of the hand, and no abnormalities were found.

Electrophysiological examinations were performed on subjects lying supine on a bed in a room temperature of 26 °C. Skin temperature was kept at or above 32 °C during the electrophysiological examinations. Nerve conduction tests for CTS were performed using a Nihon Kohden EMG machine. These tests included a motor conduction study of median and ulnar nerves (stimulation-recording distance: 80 mm), an antidromic sensory conduction study from wrist to digit 1 (wrist–finger distance: 100 mm) and digit 2 (wrist–finger distance: 140 mm) for the median nerve, and to digit 5 (wrist–finger distance: 140 mm) for the ulnar nerve. If above tests were normal, palm–wrist segment conduction velocity measurements were made at digit 2 (digit–palm distance: 60 mm and palm–wrist distance: 80 mm) as a sensitivity test. Tests were performed bilaterally and surface electrodes were used for stimulation and recording. In diabetics, tibial motor nerve (ankle–poplitea stimulation, abductor hallucis record), peroneal motor nerve (ankle–fibula head stimulation extensor digitorum brevis record) and sural sensory nerve (antidromic stimulation 140 mm above the recording electrode placed near the lateral malleolus) were studied. Electrodiagnosis for CTS was made on at least two of the following: (1) Distal motor latency longer than 4.4 ms; (2) sensory nerve conduction velocity from the wrist to either digit 1 and 2 slower than 42.7 and 51.9 m/s, respectively and (3) median sensory latency longer than 2.2 and 2.6 ms or median sensory amplitude less than 9.9 and 8.7 μV for the first and the second fingers, respectively. Also, ulnar neuropathy was evaluated on the wrist side for Guyon's syndrome. Similar to CTS diagnosis, at least two of the following were required for the diagnosis: (1) distal motor latency longer than 2.9 ms; (2) sensory nerve conduction velocity from the wrist to digit 5 slower than 53.5 m/s and (3) ulnar sensory latency longer than 3.1 ms or ulnar sensory amplitude less than 1.7 μV. Diabetic patients were considered to have polyneuropathy if two or more nerves had at least one parameter abnormal: (1) tibial motor amplitude lower than 7.2 mV; motor distal latency longer than 5.1 ms; velocity lower than 38.2 m/s; (2) peroneal motor amplitude absent; motor distal latency longer than 6.3 ms; velocity lower than 37.2 m/s and (3) sural sensory amplitude lower than 8.5 μV; motor distal latency longer than 3.4 ms; velocity lower than 38.4 m/s.

Patient hands with CTS were grouped by degree of disease as reported in the literature [12]. Briefly; grade 0: no abnormality; grade 1: CTS demonstrable only with the most sensitive test; grade 2: sensory conduction slow on finger–wrist measurement, normal terminal 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 < 6.5; grade 5: terminal latency to APB > 6.5 ms; grade 6: sensory and motor potentials effectively unrecordable.

Mimicking conditions such as polyneuropathy, C6–8 radiculopathy or proximal median nerve entrapments were excluded by other motor and sensory nerve conduction examination, needle EMG of radicular myotomes and proximal median innervated muscles, respectively. Abnormal neurological findings including muscle weakness, sensory disturbances and deep tendon reflex abnormalities with abnormal electrophysiological examination results were used for the diagnosis of polyneuropathy. Patients were also questioned for systemic autonomic abnormalities such as incontinence, constipation, nocturnal diarrhea, sexual dysfunction, ortho-