ROLE OF H-REFLEX LATENCY STUDIES IN THE DIAGNOSIS OF SUBCLINICAL DIABETIC NEUROPATHY

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Summary : Motor conduction velocity (MCV) in the median, ulnar and peroneal nerves and H-reflex studies have been conducted in 50 diabetics aged 20-65 years and 25 controls. MCV in the upper limb was below the normal range in 16% of diabetics. 28% diabetics showed abnormal MCV in the peroneal nerve. H-reflex abnormality consisting of either prolonged latency or its complete absence could be observed in 54% of diabetics. The results indicate the greater sensitivity of H-reflex in the detection of sub-clinical diabetic neuropathy. Greater prevalence of neuropathy in the early onset diabetes than in the late onset type is also suggested.

Key words : diabetes neuropathy H-reflex motor conduction velocity

INTRODUCTION

Neuropathy is a common complication of diabetes mellitus. Depending on the duration of diabetes, neuropathy may affect as many as 93% of the patients (4). Histologically, Schwann cell damage, leading to demyelination has been observed not only in distal but also in proximal segments of nerve fibre (2). In some cases axonal degeneration has also been reported (2). Another important feature of diabetic neuropathy is that the large diameter afferent fibres are affected earlier than the motor fibres (9). This may explain why motor conduction velocity (MCV) studies have not been helpful in the diagnosis of diabetic nauropathy in its early stages. Sensory conduction study could be more helpful but it is technically a difficult procedure (6). Since the H-reflex involves conduction in the proximal segments of both large diameter afferents (Ia) and efferent fibres, its study may be helpful in the diagnosis of subclinical diabetic neuropathy. A linear inverse relationship between the sensory conduction velocity and H-reflex latency has also been observed (16).

MATERIAL AND METHODS

Fifty diabetics aged 20 to 65 years, admitted to the medical wards and 25 age and sex-matched controls were investigated. The mean height of the subjects in the two groups was similar. Subjects with symptoms of S_1, S_2 radiculopathy were excluded. The diagnosis of diabetes mellitus was confirmed by oral glucose tolerance test (13). Electrophysiological investigations included the H-reflex latency studies from the soleus muscle and MCV studies in peroneal, median and ulnar nerves.

For the purpose of recording the H-reflex, the patient was made to lie prone comfortably on a couch. The knee was kept semiflexed at 120° and the ankle at 100°-120°, with ihe leg supported by heavy pillows, so as to keep the soleus muscle relaxed. Silver disc electrodes were taped on the soleus muscle; one on its belly, on the midline 2 cm below the lower level of gastrocnemius muscle and the other 5 cm distally, on the Achilles tendon, acting as the active and indifferent recording electrodes respectively. The tibial nerve was stimulated at the mid-popliteal fossa, by a single pulse from the EMG stimulator (Medicor, Budapest) with the cathode of the bipolar skin electrode placed proximally. The site of stimulation was identified from the lowest threshold for evoking the H-response. The H-response was observed on the EMG oscilloscope and photographed simultaneously for accurate calculations later. The H-reflex was identified by the already established criteria (15). The latency from the stimulus artefact to the first deflection from the baseline was taken as the H-reflex latency.

The motor conduction velocity in the peroneal nerve (fibular head to ankle), ulnar nerve (elbow to wrist) and median nerve (elbow to wrist) was studied by the standard methods (6).

RESULTS

The results of MCV and H-reflex latency studies are shown in Table I. In the diabetic group, mean MCV in the ulnar and median nerves was essentially similar to that in the controls. However MCV in the peroneal nerve was significantly lower than in the controls (Student's 't' test ; $P = < 0.05$). A study of individual cases revealed mostly borderline abnormality in MCV in median and ulnar nerves in 8 diabetics (16%) but a more severe reduction in MCV in peroneal nerve in 14 diabetics (28%).

The H-reflex latency was greater than 36 ms in 19 cases (Table II). In another 8 cases, the H-reflex could not be elicited, making a total of 27 cases (54%) in whom the H-reflex abnormality was evident. In the 8 cases showing absence of the H-reflex

the MCV was significantly reduced not only in peroneal but also in median and ulnar nerves.

TABLE I : Motor conduction velocity and H-reflex latency in diabetics and controls (mean±SD).

	*Controls**	*Diabetics*	*P value*
Motor conduction velocity (m/sec)			
Median nerve	56.72±6.41	53.55±8.62	>0.05
Ulnar nerve	55.89±5.97	54.07±8.97	>0.05
Peroneal nerve	50.17±4.73	42.39±9.22	<0.05
H-reflex latency (msec)	30.19±2.15	36.08±5.18	<0.001

*Normal range : MCV=median nerve, 45-70m/sec, Ulnar merve 45-68m/sec, peroneal nerve 40-58m/sec. H-reflex latency=25-35 msec.

TABLE II : Incidence of abnormalities in H-reflex observed in 50 diabetic patients.

		latency (msec)	*No. of cases*
A	Prolonged H latency	36-40	12
		41-45	4
		46-50	3
B	Absent H-reflex	—	8
		Total	27

The data on patients showing the H-reflex abnormality was further analysed taking into account the age of the patients at the onset of symptoms of diabetes and the duration of symptoms (Table III). Of the 13 cases with onset of diabetes before 40 years of age (early onset type) 11 showed abnormal H-reflex. On the other hand, only 16 out of

37 diabetics with onset of diabetes after 40 years of age (late onset type) showed abnormal H-reflex. The difference in the incidence of neuropathy between the two groups was highly significant (x^2 test : $P = <0.001$).

Clinical evidence of neuropathy, in the form of paraesthesias and absent ankle jerks, was present in six patients with duration of diabetes greater than five years. These cases were characterised by severe reduction in MCV, especially in the peroneal nerve and either absence of H-reflex (4 cases) of very much prolonged (>46 ms) H-reflex latency (2 cases).

TABLE III : Incidence of abnormal H-reflex in the two types of diabetes.

Duration of diabetes	*Early onset diabetes*		*Late onset diabetes*	
	Investigated n	*Affected n%*	*Investigated n*	*Affected n%*
<1 year	9	6(66)	15	2(13)
1-5 years	3	3(100)	9	4(44)
>5 years	1	1(100)	13	10(76)
Total	13	11(85)	37	16(43)

DISCUSSION

Normal mean MCV in the upper limb and significantly reduced MCV in the peroneal nerve observed by us (Table I) is consistent with the previous reports that the diabetic neuropathy affects the lower limbs earlier than the upper limbs (12). The degree of reduction in MCV in the peroneal nerve is found to be correlated with the severity of neuropathy as assessed clinically (9). MCV tends to be reduced in patients with subclinical neuropathy but the degree of reduction is greater when the neuropathy is overt (10). Our observations support these views.

H-reflex studies (Table II) have revealed evidence of neuropathy in a far greater number of diabetics (54%) than revealed by MCV (28%). Lachman *et al.* (8) could

detect abnormal H-latency in 18% of the patients in whom motor and sensory conduction velocities were within normal limits. Greater sensitivity of the H-reflex in the detection of subclinical neurepathy may be due to the fact that (a) longer pathway is tested so that borderline abnormalities in conduction velocities are amplified, (b) the neuropathy may affect the proximal sagments that are not measured by routine methods and (c) "utilisation time" of the anterior horn cells may be prolonged by the neuropathy (5).

H-reflex abnormality manifested either as a prolongation of latency or as complete abolition of the H-reflex (Table II). The absence of the H-reflex most probably reflects an elevation of the sensory threshold to the same level of motor threshold, so that due to collision of antidromic motor impulses, the H-response fails to appear (11). The absence of the H-reflex thus represents a severe form of neuropathy and in all the patients showing this feature, the MCV was abnormal not only in peroneal but even in median and ulnar nerves.

Recently type II (maturity onset) diabetes has been further classified (13) into early onset type (onset between 20 and 40 years) and late onset type (onset after 40 years). In contrast to the obese subjects in the late onset type, the early onset type diabetics are characterised by emaciation and are labelled as M type (malnutrition related) diabetics (1). In this small series, 26% of the patients had the onset of symptoms between 20 and 40 years of age and were non-obese. H-reflex studies revealed significantly greater prevalence of subclinical in this type than in the late onset type diabetics. A comparison of the results of those with duration of illness less than one year revealed H-reflex abnormality in 66% of the early onset type as against 13% of the late onset type (Table III). There seems to be no previous study in which the incidence of subclinical or clinical diabetic neuropathy in these two sub-types of maturity onset diabetes has been compared.

Our work contradicts the view that the H-reflex is a relatively poor indicator of peripheral neuropathy. According to Braddom and Schuchmann (3) H-reflex latency is prolonged only after a significant reduction in the distal sensory and motor conduction velocities. This concept may be true for neuropathies affecting only the distal segments of the peripheral nerves. In diabetic neuropathy the histological studies have revealed involvement of proximal as well as distal segments of the nerves (2). Rieu *et al.* (14) have also observed alterations in the H-reflex earlier than in the sensory and motor conduction.

Laboratory diagnosis of neuropathy in a patient without clinical sign or symptoms, is not merely of academic interest; it may have prognostic significance also. Kraft *et al.*

(7) reviewed 100 diabetics who had their nerve conduction velocities determined from 1 to 8 years previously. They observed that 44 cases who died had their conduction velocities considerably lower than the group of diabetics that survived.

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