Kawasaki Med. J. Vol. 6: No. 1·2, 35-48, 1980

CLINICAL APPLICATION OF THE F-WAVE
I. MEASUREMENT OF F-WAVE CONDUCTION VELOCITY (FWCV)

Kenji MORIMOTO

Division of Neurology, Department of Medicine,
Kawasaki Medical School,
Kurashiki 701-01, Japan

Accepted for Publication on May 21, 1980

Abstract

Measurement of motor nerve conduction velocity (MCV) and F-wave conduction velocity (FWCV) of the ulnar nerve were taken in each group of diseases, and the following results were obtained.

1. Normal values of MCV from wrist to elbow (W-E) was 57.4±3.9 m/sec (mean±SD), FWCV from wrist to spinal cord (W-C) 61.7±3.8 m/sec, and FWCV from elbow to spinal cord (E-C) 65.1±4.4 m/sec.

2. In the patients with Guillain-Barre syndrome (GBS), MCV (W-E) proved to be 57.1±6.5 m/sec, FWCV (W-C) 52.0±4.8 m/sec, and FWCV (E-C) 48.5±7.2 m/sec, showing a significant slowing in the proximal segment.

3. In the patients with diabetes mellitus (DM), MCV (W-E) proved to be 50.0 ± 6.9 m/sec, FWCV (W-C) 50.8±6.1 m/sec, and FWCV (E-C) 51.8±6.8 m/sec, indicating the slowing in both distal and proximal segments.

4. In the patients with hematological disease, MCV (W-E) proved to be 51.3±6.7 m/sec, FWCV (W-C) 52.5±6.0 m/sec, and FWCV (E-C) 54.3±9.3 m/sec.

5. In the patients with other neurological diseases, the interesting findings were obtained.

6. FWCV reflects the behaviors of conduction velocity in the proximal segment of peripheral motor nerve, especially that in the radicular portion, and by comparing it with MCV (W-E) it is possible to clarify the level of lesion, and it serves as an aid to clarify the onset mechanism and disease condition of neuropathy in diseases of various kinds.

INTRODUCTION

The F-wave was first described by Magladery and McDougal[1], and it is the muscle response elicited by the supramaximal stimulation of peripheral nerve trunk in its innervated muscle following M wave. The origin of the F-wave

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is still debated, but most investigators agree that it results, at least in part, from backfiring of the motoneuron. Namely, the impulse by peripheral nerve stimulation travels toward the spinal cord and activates the anterior horn cell antidromically, and again descends the same nerve segment orthodromically eliciting the response of its innervated muscle, the F-wave. With the conventional method it was limited to the measurement of MCV of distal segment, but by measuring of FWCV with F-wave as its parameter, it has become possible to conduct electrophysiological investigation of the proximal segment of peripheral motor nerve. Recently this FWCV measurement is being applied to clinical practice, and here the results of FWCV measurements in various diseases are reported.

SUBJECTS AND METHODS

Thirty cases of various neurological diseases, 15 of diabetic cases, and 15 cases of hematological disease were investigated. In addition, as the normal control 16 healthy individuals (12 males, 4 females) in the age ranging 19 to 27 years old were used.

The subjects were made to assume a quiet supine position with the upper limb to be tested turned slightly outward and fixed there, then by giving electrical stimulation to the ulnar nerve M wave and F-wave were elicited. The stimulation was given with surface electrode to wrist and elbow, and muscle action potentials were elicited from the needle electrode inserted into abductor digiti minimi muscle with the electromyograph, Sanei Biophysiograph 130 System. In this instance, the presence of M and F waves on the oscilloscope was confirmed, then a digital computer, Signal Processor 7TO7A was connected and further recordings were taken with XY recorder. In case F-wave was big and distinct, it was not added, and in case F-wave was indistinct or very small, it was added several times, then recorded. As F-wave even under the same condition has some variation in its latency, the recording were taken at least over 10 times repeatedly, and F-wave with the shortest latency was selected for FWCV measurement. Analytical time was 50 msec.

The calculation formula of FWCV is the method of Kimura as follows : FWCV in meters per second = (Distance from stimulus point to C_7 spine) $\times$ 2 (in millimeters) divided by (F latency-M latency)-1(in milliseconds).

The approximate length of the nerve from the stimulus points to the spinal cord was obtained by measuring the distance from the stimulus points to the C_7 spinal process. Surface measurements were taken along the course of the nerve from the wrist to axilla with the subject in a sitting positon and the arm abducted to 90 degrees. The hand was pronated for the measurement so that stimulus points were easily accessible to the examiner standing behind

the subject. Moreover, the latency of M and F waves was set as the time from stimulus artifact to the beginning of response.

RESULTS

1. Normal values (Table 1)

Normal values of MCV (W–E) was 57.4±3.9 m/sec (mean ± SD), FWCV (W–C) 61.7±3.8 m/sec, and FWCV (E–C) 65.1±4.4 m/sec, showed higher conduction velocity in the proximal segment than in the distal segment.

TABLE 1. MCV and FWCV in normal subject

No	Sex	Age	Distance (cm)		Latency (msec)				MCV (m/sec)	FWCV (m/sec)	
					Wrist		Elbow				
			W-E	E-C	F	M	F	M	W-E	W-C	E-C
1	M	24	26.0	45.0	27.5	2.5	22.5	7.0	57.8	59.1	62.0
2	M	24	24.0	45.0	25.0	2.7	20.5	6.6	61.5	64.8	69.8
3	M	22	26.1	44.8	27.8	2.8	23.5	7.4	56.7	59.0	59.3
4	M	24	26.0	44.0	26.0	2.4	21.0	7.0	56.5	61.9	67.7
5	M	21	26.0	46.1	26.5	2.8	21.5	7.5	55.3	63.5	70.9
6	M	20	24.6	45.0	26.5	3.0	22.5	7.3	57.2	61.9	63.4
7	M	20	28.6	46.0	26.4	2.7	21.5	7.5	59.6	66.3	70.8
8	F	19	21.2	39.4	23.5	2.6	20.0	6.4	55.8	60.9	62.5
9	M	26	24.6	42.0	24.5	2.5	20.5	6.6	60.0	63.4	65.1
10	F	19	24.0	43.0	25.5	2.6	21.1	6.7	58.5	61.2	65.1
11	F	19	23.2	40.4	26.3	2.6	21.6	7.1	51.6	56.0	59.8
12	F	19	24.0	43.0	27.5	2.3	22.5	7.0	51.1	55.4	59.3
13	M	27	26.0	42.2	25.0	4.0	21.0	8.0	65.0	68.2	70.3
14	M	20	24.0	45.0	25.0	3.1	21.0	6.9	63.2	66.0	68.7
15	M	22	24.0	50.0	29.3	2.1	24.7	6.7	52.2	56.4	59.1
16	M	22	27.8	49.9	28.5	3.1	23.6	8.0	56.7	63.7	68.4
Mean			25.1	44.4	26.3	2.7	21.8	7.1	57.4	61.7	65.1
S.D.			1.8	2.8	1.5	0.4	1.2	0.4	3.9	3.8	4.4

2. Patients with GBS (Fig. 1)

The patients comprised 8 cases, of which with 3 cases follow-up study was done over 2 times. Moreover, in two cases F-wave could not at all be elicited. Table 2 shows the results of 6 cases F-wave could be elicited. Here the initial examination was done within 10 days after the onset of disease. MCV(W–E) proved to be 57.1 ± 6.5 m/sec (no significant difference between patients and normal control), FWCV(W–C) 52.6 ± 4.8 m/sec ($P<0.001$), and FWCV(E–C) 48.5 ± 7.2 m/sec ($P<0.001$), showing a significant slowing in the proximal segment in spite of normal conduction velocity in the distal segment. As the result of follow-up study a clear-cut improvement of FWCV was observed, while MCV(W–E) decreased gradually (Fig. 2).

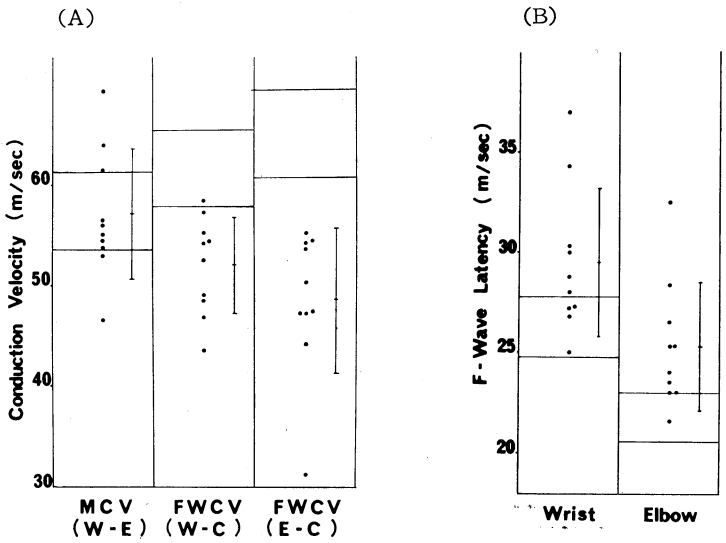

Fig. 1. Comparison between GBS and normal control
A : MCV and FWCV
B : F-wave latency at wrist and elbow

TABLE 2. MCV and FWCV in GBS

No	Sex	Age	Distance (cm)		Latency (msec) Wrist		Elbow		MCV (m/sec)	FWCV (m/sec)	
			W-E	E-C	F	M	F	M	W-E	W-C	E-C
1(a)	F	28	22.4	39.0	25.0	2.6	21.5	6.1	64.0	57.3	54.2
(b)	"	"	23.0	41.0	27.3	4.4	24.0	7.7	69.7	58.4	53.6
(c)	"	"	22.6	40.0	27.2	3.1	23.0	7.3	53.8	54.2	54.4
2(a)	F	40	24.0	43.2	28.8	3.1	25.3	7.0	61.5	54.4	47.2
(b)	"	"	22.4	43.0	30.3	4.4	26.5	8.4	56.0	52.5	50.3
3(a)	F	29	22.5	34.0	26.8	2.5	23.0	6.5	55.0	48.5	44.2
(b)	"	"	22.8	37.0	28.0	2.6	23.5	6.9	53.0	49.0	47.4
4	M	50	24.0	37.0	37.0	3.4	32.5	7.8	54.5	43.6	31.2
5	M	23	26.0	48.0	30.0	2.2	25.3	6.8	56.5	55.2	55.2
6	M	21	27.0	44.8	34.3	2.6	28.4	8.4	46.6	46.8	47.2
Mean		32	23.7	41.1	29.5	3.1	25.3	7.3	57.1	52.0	48.5
S.D.			1.6	4.2	3.7	0.8	3.2	0.8	6.5	4.8	7.2

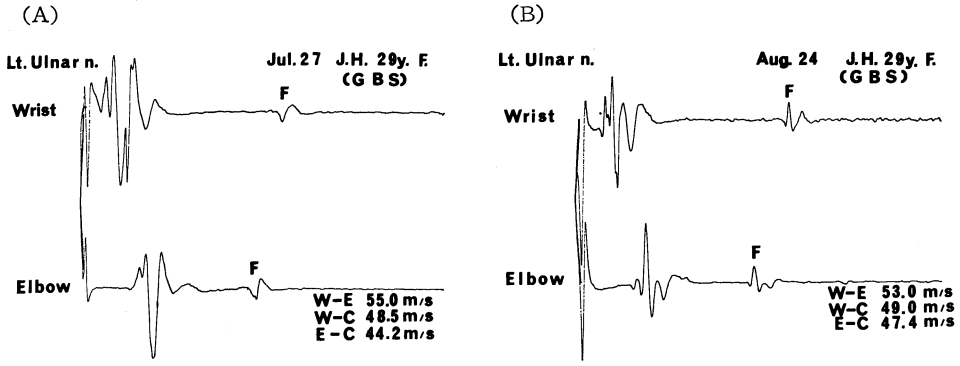

Fig. 2. Recordings of M and F waves in GBS
A : Ist recording on July 27
B : IInd recording on Aug. 24

3. Patients with DM (Table 3 and Fig. 3)

Those cases showing subjectively or objectively sign and symptom of neuropathy were excluded. MCV(W-E) proved to be 50.0 ± 6.9 m/sec ($P < 0.001$), FWCV(W-C) 50.8 ± 6.1 m/sec ($P < 0.001$), and FWCV(E-C) 51.8 ± 6.8 m/sec($P < 0.001$), indicating the slowing in both distal and proximal segments.

TABLE 3. MCV and FWCV in DM

No	Sex	Age	Distance (cm)		Latency (msec)				MCV(m/sec)	FWCV (m/sec)	
					Wrist		Elbow				
			W-E	E-C	F	M	F	M	W-E	W-C	E-C
1	M	19	25.5	45.0	44.3	4.5	37.8	11.2	38.0	36.3	35.2
2	F	30	23.5	43.0	26.3	2.8	22.3	6.8	58.8	59.1	59.3
3	M	31	27.6	44.0	30.5	3.3	25.5	8.2	56.3	54.7	54.0
4	M	44	24.3	44.0	28.0	3.6	23.5	8.0	55.2	58.4	60.7
5	F	46	23.5	37.0	25.5	3.0	21.5	6.9	60.3	56.3	54.4
6	M	48	25.5	41.0	31.5	3.1	26.0	8.5	47.2	48.5	49.7
7	M	50	25.0	39.5	33.3	3.2	26.3	10.3	35.2	44.3	52.7
8	M	50	24.5	40.0	35.0	3.3	29.5	8.8	44.5	42.0	40.6
9	M	50	26.0	42.0	31.0	2.8	25.5	8.4	46.4	50.0	52.2
10	M	60	25.0	39.5	27.8	3.2	22.8	8.0	52.1	54.7	57.2
11	M	60	25.5	39.0	31.8	3.5	26.8	8.5	51.0	47.3	45.1
12	M	62	26.0	42.0	30.5	3.1	24.8	8.7	46.4	51.5	55.6
13	F	65	22.0	41.0	26.0	2.5	21.5	6.5	55.0	56.0	58.6
14	M	65	27.0	41.0	30.8	3.0	26.0	8.2	51.9	50.7	48.8
15	M	71	26.7	42.3	31.0	3.7	26.0	8.9	51.3	52.5	52.5
Mean		50	25.2	41.4	30.9	3.2	25.7	8.4	50.0	50.8	51.8
S.D.			1.4	2.0	4.4	0.5	3.9	1.2	6.9	6.1	6.8

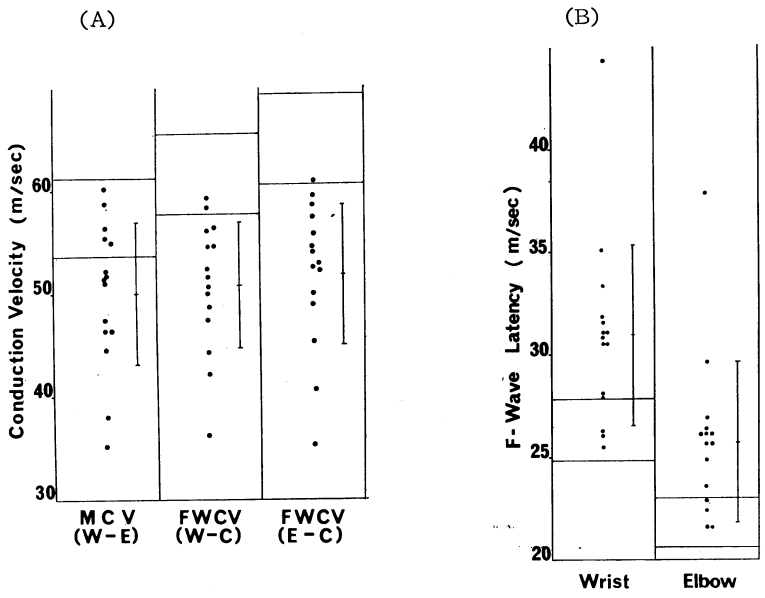

Fig. 3. Comparison between DM and normal control
A : MCV and FWCV
B : F-wave latency at wrist and elbow

4. Patients with hematological disease (Table 4 and Fig. 4)

TABLE 4. MCV and FWCV in hematological disease

No	Disease	Sex	Age	Distance (cm)		Latency (msec) Wrist		Elbow		MCV (m/sec)	FWCV (m/sec)	
				W-E	E-C	F	M	F	M	W-E	W-C	E-C
1	ALL	F	45	22.0	44.0	32.5	3.6	27.9	8.3	46.8	47.3	47.3
2	ALL	F	55	22.4	42.0	27.3	3.2	22.8	7.7	49.8	56.1	59.6
3	CGL	F	51	22.0	42.5	26.5	3.6	22.5	7.4	57.9	58.9	60.3
4	CGL	F	57	22.2	40.0	29.0	3.0	25.5	6.5	63.4	49.8	44.4
5	MM	M	57	23.0	42.0	35.8	3.8	31.0	8.4	50.0	41.9	38.9
6	MM	F	67	22.2	40.0	32.0	3.2	27.0	7.0	58.4	44.7	42.1
7	MM	F	67	18.1	41.0	23.5	3.0	19.8	6.5	53.1	61.1	66.7
8	MM	F	73	20.0	37.0	26.3	4.0	21.0	9.0	40.0	53.5	67.3
9	Hodgkin	F	64	20.0	39.0	29.0	3.2	25.0	7.0	52.6	47.6	45.9
10	Hodgkin	M	65	23.6	42.0	31.3	4.2	26.3	9.2	47.2	50.3	52.2
11	Lymphoma.	M	34	28.0	47.0	30.5	3.1	25.0	8.6	50.9	56.8	61.0
12	Lymph. sar.	F	53	24.2	41.5	31.5	2.4	25.3	8.4	40.3	46.8	52.2
13	APL	F	37	22.4	44.5	26.3	2.7	22.0	6.8	54.6	59.2	62.7
14	AMOL	M	69	27.0	45.0	31.8	4.9	26.0	10.8	45.8	55.6	63.4
15	AGL	M	68	26.0	45.0	30.8	2.9	26.3	7.3	59.1	57.8	50.0
Mean			57.5	22.9	42.2	29.6	3.4	24.9	7.9	51.3	52.5	54.3
S.D.				2.6	2.6	3.2	0.6	2.9	1.2	6.7	6.0	9.3

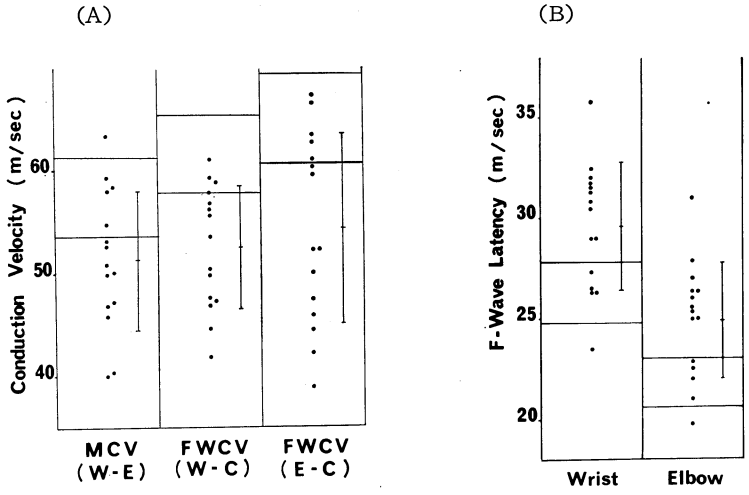

Fig. 4. Comparison between hematological disease and normal control
A : MCV and FWCV
B : F-wave latency at wrist and elbow

Of 15 patients 4 comprised multiple myeloma, 4 of malignant lymphoma and 7 of leukemia. Clinically none of them showed any finding of neuropathy.

MCV(W-E) proved to be 51.3 $\pm$ 6.7 m/sec ($P<0.01$), FWCV(W-C) 52.5 $\pm$ 6.0 m/sec ($P<0.001$), and FWCV(E-C) 54.3 $\pm$ 9.3 m/sec ($P<0.001$).

5. Other neurological diseases (Table 5)

A. Hirayama's disease

In measuring 2 cases, a significant slowing was observed only in the proximal segment (Fig. 5A)

B. Charcot-Marie-Tooth disease (CMT)

In measuring 3 cases of CMT mean MCV (W-E) proved to be 37.3 m/sec, mean FWCV(W-C) 42.3 m/sec, and mean FWCV(E-C) 46.6 m/sec, showing a marked decrease in the conduction velocity of the entire peripheral nerve area (Fig. 5B).

C. Thoracic outlet syndrome

Both of two cases were the patients with scalenus anticus syndrome, showing a conduction velocity decrease in the proximal segment.

D. Patients with neuropathy of various kinds

Respective cases had a decrease of conduction velocity in the distal or proximal segment.

E. Fisher's syndrome

This syndrome was found in only one case, but MCV(W-E) proved to

TABLE 5. MCV and FWCV in other neurological disease

No	Disease	Sex	Age	Distance (cm)		Latency (msec)				MCV (m/sec)	FWCV (m/sec)	
						Wrist		Elbow				
				W-E	E-C	F	M	F	M	W-E	W-C	E-C
1	Hirayama	M	23	27.3	45.5	28.0	3.3	23.5	7.5	65.0	61.4	60.7
2	〃	M	25	24.8	42.0	31.5	3.5	26.5	8.2	52.8	49.5	48.6
3	C-M-T	M	20	26.5	44.9	34.8	3.1	29.0	8.8	46.5	46.5	46.8
4	〃	M	45	25.0	42.0	33.2	3.4	26.5	10.1	37.3	46.5	54.5
5	〃	F	58	22.5	38.0	41.3	4.5	33.0	12.2	29.2	33.8	38.4
6	K-W	F	15	21.5	35.0	23.5	3.4	19.3	7.7	50.0	59.2	66.0
7	ALS	M	50	26.1	44.5	27.0	3.1	22.5	7.6	58.0	61.7	64.0
8	〃	M	56	24.0	42.5	31.3	3.0	26.3	8.0	52.0	50.7	49.0
9	PMD (L-G)	M	12	24.0	40.0	26.3	2.6	22.0	7.0	54.5	56.4	57.1
10	〃	M	34	23.0	43.0	30.8	4.0	26.3	8.5	51.1	51.2	51.2
11	Thoracic outlet	M	53	25.0	40.0	30.3	3.7	25.8	8.5	52.0	50.7	49.0
12	〃	F	70	23.2	41.0	27.0	2.8	22.8	7.0	55.2	55.3	55.4
13	MS	F	22	21.4	39.8	23.0	3.1	19.3	6.6	64.8	64.9	68.0
14	PM	M	22	23.8	44.0	29.5	2.5	25.3	7.0	52.9	52.2	51.0
15	Neuropathy (Ca)	M	58	20.4	43.0	31.3	3.1	27.0	7.6	45.3	46.6	46.7
16	〃 (unknown)	M	30	24.6	37.2	28.0	3.3	22.8	8.6	46.4	52.2	56.4
17	〃 〃	M	45	26.0	41.0	31.5	5.0	24.8	11.7	38.8	52.5	67.8
18	〃 〃	M	28	24.7	38.0	26.3	3.1	21.3	8.0	50.4	56.5	61.8
19	〃 〃	M	40	22.5	42.0	32.0	3.4	28.3	7.6	53.6	46.7	42.6
20	〃 (CS_2)	M	25	24.3	44.5	27.5	3.4	22.8	8.3	49.6	59.6	65.9
21	Fisher	M	14	25.0	40.5	23.0	2.5	19.0	6.6	61.0	67.2	71.1

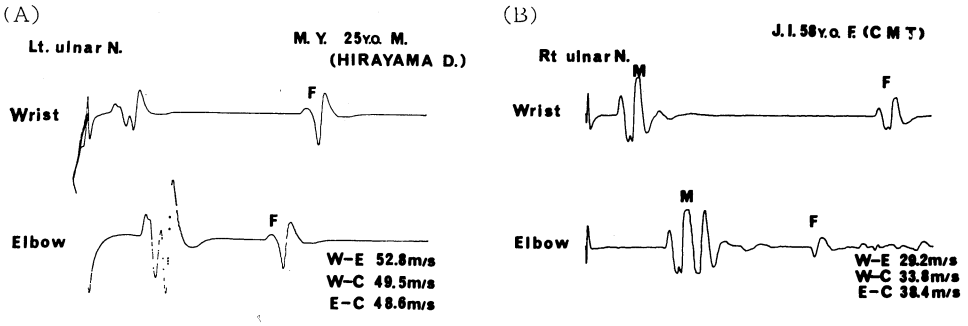

Fig. 5. Recordings of M and F-waves
A : Hirayama's disease
B : Charcot-Marie-Tooth disease

be 61.0 m/sec, FWCV(W-C) 65.0 m/sec, and FWCV(E-C) 70.1 m/sec, all being normal.

F. Amyotrophic lateral sclerosis (ALS), Polymyositis (PM), Kugelberg-Welander disease (K-W), Progressive muscular dystrophy (PMD), and Multiple sclerosis (MS).

ALS patients were 3 in number, of whom in one the measurement could not be taken as it was not possible to elicit F-wave, and one of the remaining two was normal and the other showed a slow conduction velocity in the proximal segment. All 3 cases of myopathy, PM and PMD, had a slight de-

crease in the conduction velocity. K-W patient showed a slow MCV(W-E), and MS patient showed normal.

DISCUSSION

1) The nature and origin of F-wave

In 1950, Magladery and McDougal[1] described a potential which was recorded at a greater latency than the direct motor response in small hand or foot muscles in man upon electric stimulation of the mixed peripheral nerve to the muscle, and designated it as F-wave. They further demonstrated the following characteristics of F-wave : (1) In order to elicit this response it is necessary to give a strong electric stimulation to be a supramaximal stimulus for motor fibers ; (2) the latency of this response becomes longer as the stimulus point is further away from the spinal cord ; (3) the latency and the wave shape of this response are variable ; (4) this response is absent when the nerve was stimulated distal to a complete procaine nerve block ; (5) this response was best recorded from small hand or foot muscles ; and (6) as this response appears by supramaximal stimulation, it differs from H wave which disappears by supramaximal stimulation. As for the origin of this F-wave, since M wave persists after procaine block but F-wave disappears, they considered that it is the result of an impulse discharged from the central nervous system, and either it is elicited by the impulse that has traveled antidromically the axon of motor neuron or it is a true reflex response. However, supposing it to be an antidromical impulse, the difference of latency of M wave and the difference of latency of F-wave obtained from two sites of the same nerve should be the same, but their data showed the difference, and the centripetal conduction velocity, i. e. conduction velocity of F-wave gave difference value from that of centrifugal conduction, i. e. conduction velocity of M wave, so that they assumed it to be of reflex origin. Thereafter many investigators challenged the interpretation of origin of F-wave, and in 1956 Dawson and Merton[2] suggested for the first time in their brief report that F-wave resulted from the discharge of motoneuron elicited by the antidromic stimulation of efferent fiber.

Later in 1966 McLeod and Wray[3] in their experiment of baboon stated a possibility of F-wave being elicited after deafferentation, in 1967 Mayer et al.[4] stated that F-wave persisted also in the patient of chronic deafferentation, and in 1976 Sawnhey et al.[5] reported a possibility of F-wave being elicited by the stimulation of facial nerve which is a pure motor nerve. In 1973 Miglietta[6] reported the appearance of F-wave even in the patient undergoing dorsolateral longitudinal myelotomy, and in the same year Trontelz[7] stated

that by using single fiber EMG M wave and F-wave were initiated in the same nerve fiber. Many such reports have substantiated that F-wave is elicited by the antidromic stimulation of spinal motoneuron. The author has also observed F-wave being elicited by the stimulation of facial nerve. At the present stage, for the origin of F-wave a possible contribution of reflex component cannot be ruled out, but there seems to be no question that at least backfiring of motoneuron is related.

2) Measurement of FWCV

1. Normal values

As the peripheral nerve approaches closer to the proximal, its diameter becomes bigger, and conduction velocity becomes naturally faster in the proximal segment than in the distal segment. In my present study, I also obtained the result as MCV (W-E) < FWCV (W-C) < FWCV (E-C) just as I expected. Regarding normal values there are reports by Kimura[8], Eisen et al.[9] and my values were similar to that of Eisen et al. Namely, MCV(W-E) 57.2±4.5 m/sec, FWCV(W-C) 61.7±3.5 m/sec, and FWCV(E-C) 62.3±5.0 m/sec by their data. Compared with my data their value of FWCV(E-C) is slightly lower, but the difference in the values seems to be due to the difference in the age range as 21.8 years old in my average age as against 41.5 years old in theirs. In either report the proximal dominancy as observed faster velocity in the proximal segment as well as the value of FWCV itself are both commonly important points.

2. Patients with GBS and Fisher's syndrome

Excepting Case 1 with GBS and a case with Fisher's syndrome these cases are those of motor dominant polyradiculoneuritis accompanied by albuminocytologic dissociation of cerebrospinal fluid and they are clinically typical GBS. FWCV showed a significant decrease of conduction velocity in the proximal segment in all cases except Case 6. In GBS patients of the past it is well known that most cases show the slowing of MCV(W-E). On the other hand, in early stage there are many cases who do not show MCV(W-E) decrease, and actually even in my results a marked decrease of FWCV at the initial examination while MCV(W-E) was in normal range in most cases.

As to GBS Kimura et al.[10], King et al.[11], and Ishida et al.[12] have already reported conducton velocity decrease in the proximal segment. In GBS it is a well-known fact that pathologically demyelinating lesion can be observed at the radicular portion, but it is quite significant to demonstrate the involvement of radicular portion of peripheral nerve electrophysiologically that could not be accomplished by the conventional methods.

Follow-up study done more than twice amounted to 3 cases, where a

clear-cut improvement of conduction velocity was observed in the proximal segment, while conduction velocity of the distal segment decreased gradually. This fact clearly demonstrates that the fall in MCV(W-E) of GBS patients cannot be observed in an early stage and it appears in the recovery stage. Case 1, moreover, had the swelling of eye-lids and lips after upper respiratory tract infection, and at the time of visiting the hospital the bilateral facial nerve paresis was observed.

As for four limbs there was no abnormality other than slight dysesthesia of the finger tips, and at first the case was suspected of Melkersson-Rosenthal syndrome[13,14].

However, by the cerebrospinal fluid examination a marked albuminocytologic dissociation was observed, and further conduction velocity measurement showed MCV(W-E) to be normal, but a slowing of FWCV was recognized so that the case was finally diagnosed as the cranial form of GBS[15], and recovered without any residual signs and symptoms. Next, as to Fisher's syndrome[16,17] which calls an interest in its relation to GBS, though only one case, gave quite normal values.

This syndrome shows areflexia, ataxia and ophthalmoplegia as a triad and often reveals albuminocytologic dissociation of cerebrospinal fluid. Clinically, it has much similarity to the cranial form of GBS mentioned already, and there are reports suggesting of a variant form of GBS. However, by the results of MCV and FWCV it differs clearly from GBS, and its site of lesion seems to be located at least above the spinal cord level.

3. Patients with DM

Diabetic neuropathy has segmental demyelination as its main pathological change and shows a decrease in the nerve conduction velocity. The subjects that I studied this time all showed clinically no sign or symptom of neuropathy, but revealed the fall in MCV(W-E) as well as in FWCV. In diabetic patient there can be often observed an increase of protein in cerebrospinal fluid, which seems to be a reflection of radiculoneuropathy. My present results suggest this, and actually there were observed involvement of anterior root and loss of anterior horn cells[18,19]. Regarding the measurements of FWCV Conrad et al.[20], Narita et al.[21] and Argyropoulos et al.[22] have reported similar results, especially Conrad et al. have stated that F-wave latency is more sensitive than conventional nerve conduction velocity, and it is useful in identifying the initial phase of neuropathy. About the relation of the severity and the disease duration I did not conduct precise observation, but Narita et al. stated that the degree of FWCV decrease rather than the disease duration was correlated to the severity.

4. Patients with hematological disease

In patients with malignant tumor or hematoproliferative disease, sensory or sensoriomotor neuropathy is apt to occur, and as its cause a remote effect is pointed out. By the study of nerve conduction velocity also the results suggesting subclinical neuropathy are often reported. However, at present there is no report of FWCV in hematological disease. In the present study a significant decrease of nerve conduction velocity was observed in both distal and proximal segments. However, the average age is as high as 57.5 years compared with normal control so that it requires to take aging into consideration, hence it cannot at once be judged as abnormal. Nonetheless, there are many patients without any neuropathy clinically but with a significant decrease of nerve conduction velocity in the proximal segment, so that the presence of cases with subclinical radiculoneuropathy cannot be ruled out. Be it as it may, in patients of advanced age an important point to bear in mind is the presence of entrapment neuropathy of proximal segment due to cervical spondylosis.

In the present cases hardly any spondylotic change was observed on the cervical X-ray film, but a possibility of latent entrapment should always be taken into consideration.

5. Other neurological diseases

Hirayama's disease[23], otherwise known as juvenile muscular atrophy of unilateral upper extremity, is assumed to be the lesion of brachial plexus or root or spinal cord[24,25]. While there is no report of FWCV measurement in this disease, in the present results the slowing of nerve conduction velocity was observed only in the proximal segment. From this result it is not possible to conclude as the lesion of plexus or root, but at least the lesion of proximal segment of peripheral nerve was confirmed electrophysiologically. In patient of CMT Kimura[8] and Panayiotopoulos et al.[26] reported a decrease of nerve conduction velocity uniformly in each segment of peripheral nerve. The author also obtained similar results.

As to this uniform decrease of nerve conduction velocity in each segment, the question whether it is due to the progression of disease condition or it is decreased from the initial stage remains unclarified. However, assuming from the patient age, disease duration and values of MCV, and FWCV in the present 3 patients, it gives an impression that nerve conduction velocity decreases uniformly from early stage, and the degree of the decrease is correlated to the advance of disease condition. Of 3 ALS cases nerve conduction velocity decrease of proximal segment was observed in one case. Kinoshita et al. in their investigation of 17 ALS cases did not find any significant difference of the average value of FWCV from the control, but found a decrease of FWCV in some

cases.

As the early stage pathological change of ALS, numerous spheroids are found in the anterior horn of spinal cord[27], and this spheroid is considered to be swollen proximal axon[28]. It is unclarified whether it has a direct relation to the decrease of FWCV, but it is an interesting finding. Even in PM and PMD nerve conduction velocity decrease can be observed, but the presence of neural involvement is suggested in either case, so that the result is not inconsistent.

In conclusion, FWCV reflects the behaviors of conduction velocity in the proximal segment of peripheral motor nerve, especially that in the radicular portion, and by comparing it with MCV(W-E) it is possible to clarify the level of lesion, and it serves as an aid to clarify the onset mechanism and disease condition of neuropathy in diseases of various kinds.

Acknowledgment

The author would greatly thank Susumu Shibata, M. D., Dean of Kawasaki Medical School, for his cordial encouragement, Akira Terao, M. D., Professor of Medicine, and Teruo Shirabe, M. D., Associate Professor of Pathology for their valuable advice.

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