HEMIPLEGIC AMYOTROPHY AND MOTOR NERVE CONDUCTION VELOCITY IN HEMIPLEGIC PATIENTS

I. HEMIPLEGIC AMYOTROPHY: ITS CLINICAL AND ETIOLOGICAL CONSIDERATIONS

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INTRODUCTION

It has been classically taught that amyotrophy or muscle degeneration is the result of myogenic or lower motor neuron lesions. However, careful clinicians have sometimes found muscle atrophy in the involved sides of hemiplegic patients. Over the past one hundred years, many attempts have been made to explain the mechanism of hemiplegic amyotrophy.¹⁻¹⁷ The etiology of this condition has been variously described as neural atrophy, disuse atrophy, atrophy due to vasomotor dysfunction,³ and central or parietal atrophy (which is a result of loss of control of trophic influences on the muscle).² Of the above, parietal atrophy has been described in detail.^{2,4,6-15} But it is not generally accepted that any of these theories satisfactorily explain the cause of hemiplegic amyotrophy. In the last several years, the question of lower motor neuron involvement in the hemiplegic patient has been a subject of controversy. Varying results of motor nerve conduction velocity studies and needle electromyogram findings have been reported.¹⁶⁻²⁶

In this paper, the relationship between the clinical findings in hemiplegic patients and hemiplegic amyotrophy will be described, and etiology and mechanism of hemiplegic amyotrophy will be discussed. A following paper will deal with the relationship between clinical data and the maximum motor nerve conduction velocities of ulnar nerves in hemiplegic patients.

MATERIALS AND METHODS

The subjects were 100 patients with varying degrees of hemiparesis as a result of cerebrovascular disease. All patients were examined by the author at Nirayama Rehabilitation Hospital during hospitalization there from January, 1970, to September, 1971. All patients who had such complications as diabetes mellitus or uremia, or who had evidence of peripheral neuropathy, were excluded from this study. Patients who were suspected of having bilateral hemispheric lesions were also excluded.

Clinical diagnosis was made using the following evaluations: onset and course, neurological examination, cerebrospinal fluid findings, cerebral arteriogram, and brain scan. Varying combinations of the above examinations were used for each patient, since not all studies were necessary for every subject. Among these 100 patients, 8 were diagnosed as cerebral hemorrhage, and 4 were diagnosed as ruptured aneurysm and A-V malformation. The other 88 patients had cerebral infarction. The ages of the patients ranged from 40 to 77 years. Eighty patients were men, and twenty patients, women. The duration of lesion from onset of CVD ranged from two months to seven years. Sixty-three patients had lesions of the dominant side, and thirty-seven patients had lesions of the non-dominant side. All patients except one were righthanded. Degree of severity of motor impairment in the upper extremities was divided into three groups by Brunnstrom stages. There were 34 cases in Br. 1-2 stage, 33 cases in Br. 3-4 stage, and 33 cases in Br. 5-6 stage. Degree of muscle tone in the involved upper extremities was evaluated by the author according to resistance to passive stretch. Three groups were formed according to degree of muscle tone. There were 47 patients in the rigidospasticity group, 33 in the normotonicity group, and 20 in the flaccidity group. Degree of deep tendon reflexes in the involved upper extremities was divided into two groups: slightly increased (+1), and increased (+2). The number of cases were 49 and 51, respectively. Cases were also divided into two skin temperature groups: those with no skin temperature difference in both upper extremities, and those with a clinically low skin temperature in the involved extremity. There were 38 patients in the former group, and 62 patients in the latter category. No patients showed an increased skin temperature in the involved extremity at the time of examination. Two other factors examined were aphasia and central sensory involvement in the involved extremity, including face.

Determination of muscle atrophy in this study was done as follows: the maximum circumference of both the involved and uninvolved forearms was

measured to determine the degree of muscle atrophy. As a control group, forty healthy hospital employees, all over 40 years of age, were selected for measurement, also. In this group, measurements revealed that the forearm controlled by the dominant hemisphere had a greater circumference than that of the non-dominant side by 0 to 1 cm. Also, as a preliminary study, the thickness of the skin and underlying subcutaneous tissue of the forearm, when raised between thumb and forefinger, was measured by means of a small caliper. Results showed no significant difference in the involved and uninvolved extremities of each patient. Therefore, using the above measurements as criteria in the patient study, if the dominant arm was on the involved side and smaller than the other by as much as 1 cm, the forearm circumference difference was designated (+1) atrophy; over 1 cm forearm circumference difference was designated (+2) atrophy. However, if the non-dominant arm was on the involved side and smaller than the other by at least 1 to 2 cm in circumference, then this difference was designated (+1) atrophy, and over 2 cm difference, (+2) atrophy. According to these standards, results of the measurements revealed that 31% of the patients had no muscle atrophy, 48% had (+1) atrophy, and 21% had (+2) atrophy. A total of 69 patients (or 69%) had some degree of muscle atrophy in the involved upper extremity.

RESULTS

(1) Muscle Atrophy in Relation to Clinical Diagnosis

Patients were divided into three groups: those with cerebral infarction, those with cerebral hemorrhage, and those with ruptured aneurysm and A-V malformation, as described before. Each group was then subdivided into two more groups: those without muscle atrophy, and those with muscle atrophy, as shown in Table 1. No relationship was found in comparing clinical diagnosis to the absence or presence of muscle atrophy.

Table 1
Muscle atrophy in relation to clinical diagnosis

Muscle atrophy	Cerebral infarction	Cerebral hemorrhage	Miscellaneous	Total no. of patients
—	29	1	1	31
+	59	7	3	69
Total no. of patients	88	8	4	100

(2) Muscle Atrophy in Relation to Age

Patients were divided into three age groups: below 50 years, 50 to 60 years, and over 60 years. Each of these groups was subdivided into two more groups: those without muscle atrophy, and those with muscle atrophy (+1 and +2 atrophy groups combined). These results are shown in Table 2. There was no statistical relationship between the patient's age and the absence or presence of muscle atrophy in the involved forearm.

Table 2
Muscle atrophy in relation to age

Muscle atrophy	Age in years			Total no. of patients
	Below 50	50 to 60	Above 60	
—	7	4	20	31
+	14	13	42	69
Total no. of patients	21	17	62	100

(3) Muscle Atrophy in Relation to Sex

Each group was divided according to sex, and then further divided into groups without and with muscle atrophy. As Table 3 shows, no statistical relationship was found between the sex of a patient and the absence or presence of muscle atrophy.

Table 3
Muscle atrophy in relation to sex

Muscle atrophy	Male	Female	Total no. of patients
—	27	4	31
+	53	16	69
Total no. of patients	80	20	100

(4) Muscle Atrophy in Relation to Duration of Lesion from Onset

Patients were divided into three groups according to duration of lesion: less than 6 months, 6 to 12 months, and more than 12 months. These groups were subdivided into two more groups: those without and those with muscle atrophy. As shown in Table 4, duration of lesion seemed to have no effect on muscle atrophy.

Table 4
Muscle atrophy in relation to duration of lesion from onset

Muscle atrophy	Duration of lesion from onset			Total no. of patients
	Less than 6 months	6 to 12 months	More than 12 months	
—	8	9	14	31
+	21	18	30	69
Total no. of patients	29	27	44	100

(5) Muscle Atrophy in Relation to Side of Occurrence of Lesion

Patients were divided into two groups: those with right hemiparesis and those with left hemiparesis. A significant relationship was noted between side of lesion and absence or presence of muscle atrophy, as shown in Table 5 ($P < 0.001$). In the group with right hemiparesis, muscle atrophy was more frequently noted (84.1%), as compared to the group with left hemiparesis (43.2%). However, the degree of muscle atrophy (+1 or +2) was found to be unaffected by the side of lesion.

Table 5
Muscle atrophy in relation to side of occurrence of lesion

Muscle atrophy	Patients with right hemiparesis	Patient with left hemiparesis	Total no. of patients
—	10	21	31
+	53	16	69
Total no. of patients	63	37	100

$(P < 0.001)$

(6) Muscle Atrophy in Relation to Degree of Motor Function

All patients were divided into three groups, according to Brunnstrom stages, and then further subdivided into two more groups: those without and those with muscle atrophy. There was a significant relationship between degree of motor function and absence or presence of muscle atrophy, as shown in Table 6(a) ($P < 0.01$). Further study revealed that there was significant correlation between absence or presence of muscle atrophy and the comparison of Br. 5-6 group to Br. 3-4 group (Table 6(b), $P < 0.02$), and Br. 5-6 group to Br. 1-2 group (Table 6(c), $P < 0.05$). But no relationship was noted between absence

or presence of muscle atrophy and the comparison of Br. 3-4 group to Br. 1-2 group (Table 6(d)). Patients in Br. 3-4 and Br. 1-2 groups showed higher incidence of muscle atrophy, 78.9% and 76.5%, respectively, as compared with 51.5% in Br. 5-6 group.

A significant relationship was also found between the degree of muscle atrophy and the comparison of Br. 5-6 group to Br. 3-4 and Br. 1-2 groups combined (Table 6(e), ($P < 0.05$)). In Br. 5-6 group, 88.2% of the patients had (+1) atrophy. Out of the 21 patients in the group with (+2) atrophy, 19 (or 90.5%) were in the combined Br. 3-4 and Br. 1-2 groups.

Table 6 (a)
Muscle atrophy in relation to degree of motor function

Muscle atrophy	Brunnstrom stage			Total no. of patients
	Br. 5-6	Br. 3-4	Br. 1-2	
—	16	7	8	31
+	17	26	26	69
Total no. of patients	33	33	34	100
(P < 0.01)				

Table 6 (b)

Muscle atrophy	Br. 5-6 group	Br. 3-4 group	Total no. of patients
—	16	7	23
+	17	26	43
Total no. of patients	33	33	66
(P < 0.02)			

Table 6 (c)

Muscle atrophy	Br. 5-6 group	Br. 1-2 group	Total no. of patients
—	16	8	24
+	17	26	43
Total no. of patients	33	34	67
(P < 0.05)			

Table 6 (d)

Muscle atrophy	Br. 3-4 group	Br. 1-2 group	Total no. of patients
—	7	8	15
+	26	26	52
Total no. of patients	33	34	67

Table 6 (e)

Degree of muscle atrophy	Br. 5-6 group	Br. 3-4 and 1-2 groups, combined	Total no. of patients
+	15	33	48
++	2	19	21
Total no. of patients	17	52	69
(P < 0.05)			

(7) Muscle Atrophy in Relation to Degree of Muscle Tonus

As described previously, patients were divided into three groups: rigidospasticity, normotonicity, and flaccidity. These groups were subdivided into those without muscle atrophy and those with muscle atrophy. Absence or presence of muscle atrophy was found to be significantly related to degree of muscle tonus, as shown in Table 7(a) ($P < 0.01$). Further statistical study showed a significant relationship between absence or presence of muscle atrophy and the comparison of the rigidospasticity group to the normotonicity group (Table 7(b), $P < 0.01$). A similar relationship was found in the flaccidity and normotonicity groups (Table 7(c), $P < 0.05$). No relationship was found between

*Table 7 (a)**Muscle atrophy in relation to degree of muscle tonus*

Muscle atrophy	Rigidospasticity	Normotonicity	Flaccidity	Total no. of patients
—	9	17	5	31
+	38	16	15	69
Total no. of patients	47	33	20	100
(P < 0.01)				

the absence or presence of muscle atrophy and the comparison of the flaccidity and rigidospasticity groups. Among 47 rigidospasticity patients, 38 or (80.9%) had muscle atrophy in the involved forearm. Among 20 flaccidity patients, 15 (or 75%) had muscle atrophy in the involved side.

Table 7 (b)

Muscle atrophy	Rigidospasticity	Normotonicity	Total no. of patients
—	9	17	26
+	38	16	54
Total no. of patients	47	33	80
(P < 0.01)			

Table 7 (c)

Muscle atrophy	Flaccidity	Normotonicity	Total no. of patients
—	5	17	22
+	15	16	31
Total no. of patients	20	33	53
(P < 0.05)			

(8) Muscle Atrophy in Relation to Degree of Deep Tendon Reflexes in the Upper Extremity of the Involved Side

No statistically significant relationship was found between degree of deep tendon reflex and absence or presence of muscle atrophy, as shown in Table 8.

Table 8

Muscle atrophy in relation to degree of deep tendon reflexes

Muscle atrophy	Deep tendon reflexes		Total no. of patients
	+	++	
—	19	12	31
+	30	39	69
Total no. of patients	49	51	100

(9) Muscle Atrophy in Relation to Skin Temperature Differences in the Involved and Uninvolved Extremities

Patients were divided into two groups as described before: those without skin temperature differences and those with skin temperature differences. These two groups were further subdivided into two more groups: those without muscle atrophy and those with muscle atrophy (+1 and +2 groups combined). There was a statistically significant relationship between absence or presence of muscle atrophy and the comparison of absence or presence of skin temperature differences in both extremities, as shown in Table 9. Among 62 patients with lower skin temperature in the involved side as compared to the uninvolved side, 88.7% had muscle atrophy in the impaired extremity. On the other hand, out of the 38 patients with no skin temperature differences, 36.8% had muscle atrophy.

Table 9
Muscle atrophy in relation to skin temperature differences
in the upper extremities

Muscle atrophy	Skin temperature differences		Total no. of patients
	—	+	
—	24	7	31
+	14	55	69
Total no. of patients	38	62	100
(P < 0.001)			

(10) Muscle Atrophy in Relation to Central Sensory Involvement in the Involved Extremity

All patients were divided into two groups: those without central sensory involvement and those with central sensory involvement. These two groups were similarly subdivided into two more groups: those without muscle atrophy and those with muscle atrophy. A statistical significance was found between absence or presence of muscle atrophy and absence or presence of central sensory involvement, as shown in Table 10 ($P < 0.001$). Evidence of muscle atrophy was high in the group with central sensory involvement, as compared to the group without central sensory involvement; 84.5% in the former, and 47.6% in the latter.

Table 10
Muscle atrophy in relation to sensory involvement
in the involved extremity

Muscle atrophy	Sensory involvement		Total no. of patients
	—	+	
—	22	9	31
+	20	49	69
Total no. of patients	42	58	100
(P < 0.001)			

(11) Muscle Atrophy in Relation to Aphasia

All patients were divided into two groups: those without aphasia and those with aphasia. These two groups were then subdivided into two more groups: those without muscle atrophy and those with muscle atrophy. There was a statistically significant relationship between these groups, as shown in Table 11 (P<0.01). In the group with aphasia, 85.7% had muscle atrophy; in the group without muscle atrophy, 80.6% were in the group without aphasia.

χ^2 test was used in the analyses of all preceding data.

Table 11
Muscle atrophy in relation to aphasia

Muscle atrophy	Aphasia		Total no. of patients
	—	+	
—	25	6	31
+	33	36	69
Total no. of patients	58	42	100
(P < 0.01)			

COMMENT

Many reports have been written concerning the histological and electrophysiological aspects of hemiplegic amyotrophy.^{12,13,15,16,17} Other reports have dealt with precise clinical and pathological descriptions of parietal atrophy.^{8-10,12,14,15} But there seem to be no reports dealing specifically with the relationships examined in this study between hemiplegic amyotrophy and clini-

cal data in a large group. The results of this study are summarized below.

(1) Hemiplegic amyotrophy was found in a total of 69 patients out of the 100 patients examined in this study.

(2) Patient's age, sex, and duration of lesion from onset were not related to the absence or presence of hemiplegic amyotrophy.

(3) Hemiplegic amyotrophy was more frequently observed in groups with lesions of the dominant hemisphere, and also in groups with aphasia.

(4) Hemiplegic amyotrophy was seen in patients with rigidospasticity, and also in those with flaccidity in the involved side. There was no relationship noted between hemiplegic amyotrophy and degree of deep tendon reflexes. There was a significant relationship between the severity of motor impairment and the absence or presence of muscle atrophy.

(5) Hemiplegic amyotrophy was seen more frequently in patients with central sensory involvement in the involved side.

(6) There was a significant relationship between hemiplegic amyotrophy and a low skin temperature in the involved side.

Muscle atrophy is often seen in the hemiplegic patient who has suffered from cerebrovascular disease. Several hypotheses in the literature attempt to explain the etiology of muscle atrophy in the hemiplegic patient with cerebral lesions, but none so far have proved to be entirely satisfactory.^{1,2,3} Muscle atrophy resulting from lesions in the parietal lobe, where the trophic center of the muscle is said to be, is called central atrophy by some authors.^{5,12,15} Disuse atrophy or inactivity atrophy comes from inactivity or immobilization of the involved muscle as a result of severely involved motor function and/or long duration of lesion from onset. As seen in this study, the duration of lesion from onset had no bearing on the development of muscle atrophy. Some patients developed muscle atrophy in the involved extremities (especially the upper extremities) very soon after stroke episodes. Critchly¹³ has described hemiplegic amyotrophy as appearing from several days to two weeks after onset of the cerebral lesion. Rhein⁵ observed patients who were completely confined to bed and chair, and used neither healthy nor involved extremities, but who still showed more exaggerated atrophy in the involved side.

In this study, 72.4% of patients with less than 6 months duration of lesion already had muscle atrophy in the involved upper extremities. Some of them were receiving physical therapy at this time, also. In the group of patients with over 12 months duration of lesion, 68.2% still had muscle atrophy in the involved side, even though almost all patients had been receiving physical therapy for at least six months. These facts suggest that hemiplegic amyotrophy may not

be reversible if due to a central origin; but further study is needed before any definite conclusion can be reached.

The author's clinical analyses showed muscle atrophy to be related to the following: lesions of the dominant hemisphere, aphasia, rigidospasticity and flaccidity, and central sensory impairment, thus suggesting that the cause of muscle atrophy is not merely disuse alone, but rather is a result of central influences. Some other authors have also disputed the disuse theory of hemiplegic amyotrophy.^{5,12,13,14}

The positive relationship of hemiplegic amyotrophy to the degree of motor impairment has had various explanations, of which four will be presented here. The first is the disuse theory, which the author does not adhere to for the reasons described above. The second is the compression or traction theory, which explains amyotrophy as resulting from denervation of lower motor neurons; but this theory was discarded by the author for the same reasons as previously mentioned. Also, the patients in this study were in a rehabilitation hospital, receiving proper care and range of motion exercises, thus ruling out any opportunity for nerve compression. As cited by Fenichel *et al*,¹² "Experimentally, muscle wasting due to limb immobilization is difficult to produce. Casting of an extremity in experimental animals rarely produces significant loss in muscle bulk, unless pressure complications occur."

The third theory is that of vasomotor dysfunction resulting from disability or inactivity of the muscle in the involved extremity.^{27,29} The author holds that this muscle disability leads to decreased blood flow to the vasa-nervosum, followed by lower motor neuron denervation, thus further inducing neurogenic myopathy. This muscle disability also leads to decreased blood flow to the vasa-musculosum, followed in turn by ischemic myopathy.^{3,11}

Decreased circulation in the involved extremities of hemiplegic patients has been suggested by several researchers.²⁷⁻³⁰ But thus far, it has not been explained whether vasomotor dysfunction is a result of a lesion in the vasomotor center of the brain, or whether it is merely a result of hemiplegic motor dysfunction. In this study, 62 hemiplegic patients (or 62%) had low skin temperatures in the involved extremities. Among them, 88.7% had muscle atrophy in the impaired extremities. Some patients also had other skin changes, such as discoloration, abnormally soft texture, edema, and increased or decreased perspiration, all of which are considered to be results of autonomic nervous system dysfunction.

From the author's examinations, a high correlation was found between severity of motor impairment and low skin temperature in the involved side.

Out of 67 patients in the moderate and severe motor dysfunction groups, (Br. 3-4 and Br. 1-2 groups combined), 50 patients, or 73.1%, had low skin temperatures in the involved extremities. On the other hand, in the Br. 5-6 group, 14 out of 33 patients, or 42.4%, had a lower skin temperature in the involved side. Since the vasomotor center may be in close proximity to the motor center of the brain,²⁸ there is a possibility that a lesion involving one area might also involve the other. These speculations are still being questioned.

The fourth theory suggests that the lesion is either small and localized in the precentral gyrus, internal capsule, or brain stem, or that it is a lesion located elsewhere which becomes widespread, thus involving one of these areas. An upper motor neuron lesion affects the anterior horn cells and/or lower motor neurons, leading secondarily to muscle atrophy.¹ A widespread lesion, involving singly or in combination the somatosensory system, the vasomotor system, the central trophic system, and the central system of muscle tonus, is able to involve the muscle by way of lower motor neurons, also.

The significant relationship of muscle atrophy to rigidospasticity and flaccidity suggests a lesion involving either singly or in combination the extrapyramidal system, including the frontal cortex and/or subcortex, basal ganglia, internal capsule, or brain stem. Thus, the upper motor system, vasomotor system, central trophic system of the muscle, or central trophic system of the peripheral nerves might be involved as well, by way of the extrapyramidal tract.

Botez³¹ in a recent study of muscle atrophy and cerebral dominance found a prevalence of hemiplegic amyotrophy in patients with lesions of the non-dominant hemisphere. However, in this study, the fact that hemiplegic amyotrophy was significantly seen more often in lesions of the dominant hemisphere and in association with aphasia suggests that the central trophic system of the muscle might possibly be in the dominant hemisphere, and also that lesions may more often involve the cortex and subcortex of the frontal and/or temporal lobes.

The relationship of central sensory involvement to hemiplegic amyotrophy, which appeared in this study, has been described by some authors, who suggest a strong relationship between parietal lobe lesions and hemiplegic amyotrophy.^{8,9,10,13} Silverstein holds that this relationship exists on the strength of his clinical observations that atrophy was always accompanied by sensory loss, and never seen in its absence. In this study, a higher incidence of muscle atrophy was found in the group with central sensory involvement than in the group without central sensory involvement. However, the latter group to date still shows muscle atrophy in 20 out of 42 patients. Even in the group with central sensory involvement, the lesions seemed to be located in various parts of the

brain and not necessarily in the parietal lobe. Kennard⁶ and Peele⁷ have reported finding atrophy in only some experimental animals with parietal lobe lesions.

The relationship between low skin temperature and hemiplegic amyotrophy has been explained in part by immobilization of the involved extremity, as described before. But the author holds, as some others do,^{3,11} that a lesion in the vasomotor system of the brain induces vasomotor changes in the contralateral extremity, and thus induces deprivation atrophy. The author also suggests that the vasomotor system is in function closely related to the central trophic system of the muscle and/or peripheral nerves, which may be topographically near the paracentral area down to the internal capsule. Involvement of either one of the above areas may to a greater or lesser degree induce hemiplegic amyotrophy.

SUMMARY

From the results of this study, the author considers the conclusions listed below to be most relevant.

(1) Sixty-nine per cent of the 100 hemiplegic patients in this study had some degree of muscle atrophy in the involved upper extremities.

(2) Hemiplegic amyotrophy is muscle atrophy resulting from a central origin.

(3) Important factors in the etiology of hemiplegic amyotrophy are the interrelationships of vasomotor system dysfunctions and disturbances of the central trophic system of the muscle and/or peripheral nerves.

(4) The locations of the above-mentioned systems have not been exactly determined, but the author believes that these areas play an important role in inducing hemiplegic amyotrophy as does the parietal lobe.

(5) Hemispheric dominance is an important factor in inducing hemiplegic amyotrophy.

(6) The relationship of hemiplegic amyotrophy to severity of motor impairment suggests that (in addition to the possible close proximity of the vasomotor and central trophic systems to the pyramidal tract) lower motor neurons, involved by upper motor neurons, may possibly induce denervation atrophy.

To approach the problem of peripheral motor neuron impairment, a follow-up paper will deal with the author's examinations of hemiplegic patients by means of measuring motor nerve conduction velocities of ulnar nerves, the results of which will be compared with the patients' clinical data.

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