A 15-year follow-up of neuromuscular function in patients with prior poliomyelitis

Abstract—A population-based cohort of poliomyelitis survivors was established and followed for 15 years (mean time since poliomyelitis was 40 years). Over time, the cohort demonstrated only a modest decline in function as measured by strength measurements, electrophysiologic assessments, and timed functional tasks. There was no association between symptoms of late deterioration and magnitude of decline. Rather, the presence of these symptoms was associated with the magnitude of the residual deficits.

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Progressive neurologic deficits in patients with a remote history of poliomyelitis have been reported for many years.[1] Despite these reports, the natural history of old polio remains unknown. In 1987, we began a population-based cohort study of 50 subjects with a remote history of paralytic poliomyelitis. We have now extended the follow-up period on this cohort to 15 years. Specifically we addressed 1) the stability of strength in this cohort, 2) whether symptoms of progressive weakness are associated with the magnitude of decline with time, and 3) whether symptoms of progressive weakness are associated with the magnitude of baseline residual deficits.

Methods. The demographics of the study population have been previously described.[2,3] Fifty representative subjects were chosen from 298 patients who previously had well-documented paralytic poliomyelitis, living in Olmsted County at the time of the infection. Each subject completed a self-administered questionnaire validated in the previously published studies.[2]

Each subject was assessed by the following outcome measures: manual muscle testing via the Neurologic Disability Scale (NDS) score[4] (table 1) quantitative isometric strength testing (see table 1), electrophysiologic testing for compound muscle action potential amplitudes (bilateral median and peroneal nerves) and motor unit number estimates (bilateral median and peroneal nerves),[5] and a battery of timed functional tasks (time to walk 100 ft, standardized pegboard tests of placement and displacement, pin and collar manipulation, and screw manipulation). All examinations were completed in the same manner at all time points. A single occupational therapist completed all the timed functional tasks at baseline and 5- and 15-year time points. All examiners were blinded to the subjects' other measurement scores and symptom status.

To determine whether there was a decline with time, an analysis of variance for repeated measures was completed for the baseline, 5-year, and 15-year follow-up data. Wilcoxon rank-sum tests were completed to determine whether there was an association between the subjects' symptom status and the magnitude of decline or the magnitude of baseline deficits.

Results. Of the original 50 subjects of the cohort, three subjects died during the 15-year follow-up period. Nine of the remaining 47 refused further participation. Thirty-eight of the original 50 completed the 15-year follow-up. The mean age of the 38 participants was 53 years (range 36 to 71) in 1987 at the inception of the cohort. The mean age at the time of the acute poliomyelitis was 13 (range 1.5 to 36). There were 22 women and 16 men.

The cohort demonstrated a modest decline in function over the 15-year follow-up period. There was a modest change in the NDS scores (mean of 18 at baseline vs 21 at study termination, $p = 0.01$). Additionally there was a drop in summated compound muscle action potentials (CMAPs) and summated motor unit number estimations (MUNEs), vital capacity on pulmonary function testing, and all the timed functional tasks. The data from these measurements are summarized in table 2.

Quantitative isometric strength testing supported this decline in strength. In the upper extremities, virtually all muscle groups declined significantly over time. In the lower extremities, a similar decline in function occurred with time; however, the changes of the measures were not significant.

Twenty-five of the 38 subjects had progressive weakness at the beginning of the study in 1987. The magnitude of change over the 15-year follow-up period was compared between those symptomatic at baseline and those asymptomatic at baseline. There were no significant differences identified in any of the outcome measures between these two groups. There was a mean decline in NDS scores of 2 points for the symptomatic group and a mean decline of 4 points for the asymptomatic group ($p = 0.62$) (figure). Electrophysiology testing demonstrated a mean decline in the summated CMAP of 5.9 mV in the symptomatic group and an 8.9 mV decline in the asymptomatic group ($p = 0.59$). Summated MUNEs demonstrated a mean decline of 139 motor units in the symptomatic group compared with 154 motor units in the asymptomatic group ($p = 0.90$). There were no differences identified in any of the timed function tasks, pulmonary function tests nor the quantitative isometric strength testing.

We compared baseline characteristics for all subjects who ever had symptoms of progressive weakness with those who remained symptom free for the duration of the study. The magnitude of the baseline deficits was associated with having late symptomatic progression. The larger the deficit the more likely the subject was symptomatic. The mean baseline NDS score for the symptomatic group was 20 points compared with 4 points for the asymptomatic group ($p = 0.04$; see figure). Electrophysiology testing demonstrated a mean summated CMAP of 24.8 mV for the symptomatic group at baseline compared with 31.8 mV in the asymptomatic group ($p = 0.09$). The mean summated MUNE was 376 motor units in the symptomatic group at baseline compared with 540 motor units in the asymptom-

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Table 1 Muscles examined for the strength testing measures

Muscles included in Neurological Disability Scale score	Muscle groups included in quantitative strength testing
Facial innervated muscles	Elbow flexion
Masseter	Elbow extension
Tongue	Forearm pronation
Neck extensors	Forearm supination
Neck flexors	Hand grip
Supraspinatus	Knee flexion
Infraspinatus	Knee extension
Deltoid	Ankle dorsiflexion
Biceps	Ankle plantarflexion
Triceps	
Brachioradialis	
Wrist flexion	
Wrist extension	
Finger flexion	
Finger extension	
Thenar	
Interossei	
Abdominal	
Iliopsoas	
Quadriceps femoris	
External and internal hamstring	
Anterior tibialis	
Peroneus longus	
Extensor digitorum longus	
Flexor digitorum longus	
Posterior tibialis	
Gastrocnemius	

atic group ($p = 0.04$). The mean time to walk 100 ft was 33.7 seconds in the symptomatic group at baseline compared with 26.2 seconds for the asymptomatic group ($p = 0.03$). No significant differences were identified in other timed tasks or with the quantitative isometric strength testing.

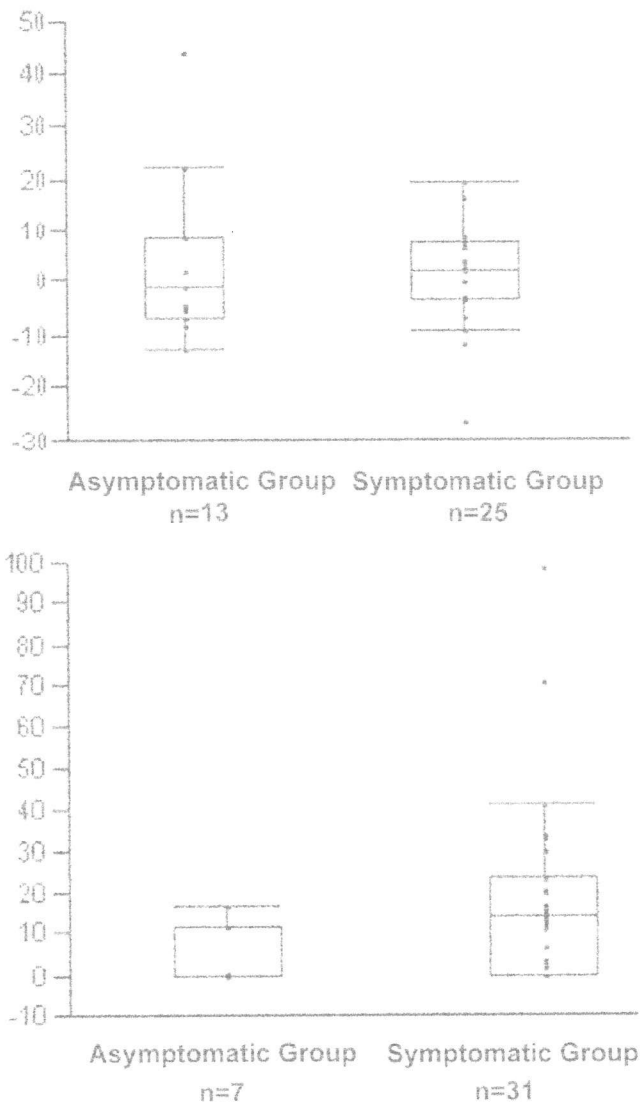

Figure. The upper graph represents the quantile plot of the change in the Neurological Disability Scale (NDS) score by symptom status at baseline ($p = 0.62$). The lower graph represents the quantile plot of the baseline NDS score by symptom status throughout the follow-up period ($p = 0.04$). These graphs demonstrate that no association is identified between the presence of symptoms and the magnitude of change with time; however, there is an association between the presence of symptoms and the magnitude of deficit at the beginning of the study period.

Discussion. The syndrome of progressive weakness late after paralytic poliomyelitis was quite common in our cohort. Thirty-one of the 38 subjects whom we followed for 15 years reported symptoms of progressive weakness during the study period. Only seven subjects remained asymptomatic. The long follow-up period in our population-based cohort has allowed a unique and more accurate assessment of the long-term stability of neuromuscular function in these subjects. Our cohort, as a whole, did demonstrate a modest decline in strength and functional performance with time.

The greatest risk factor for the presence or absence of progressive symptoms was the magnitude of neurologic deficit at baseline. In nearly all our measurements, there was an association between symptomatic progression and the magnitude of deficit at baseline. The larger the residual deficits present at baseline, the more likely the subjects were to be symptomatic. The rate of change over time, however, failed to demonstrate an association with the presence or absence of symptoms at baseline.

Our electrophysiology results agree with the findings of McComas et al.,[8] in which a loss of motor units was identified over time in a small group of subjects with polio followed for a short period of time. Additionally, our findings are consistent with those of a recent study in which a 6-year follow-up period failed to identify any deterioration and the subjects' perceived health status was only associated with the magnitude of their residual deficit.[7]

How the changes identified in our polio cohort compare with those of a normal aging population remains unknown. We did not include a normal control group at the inception of the cohort. In the absence of a normal control population, the effects of normal aging in this cohort cannot be commented on. The other principal limitation of this study was the

Table 2 Means for all measured variables over time for the cohort (n = 38)

	Baseline mean (SD)	5-year follow-up mean (SD)	15-year follow-up mean (SD)	*p*
Neurological Disability Scale score	18 (5.5)	15 (3.3)	21 (7.5)	0.01
Summated compound muscle action potential amplitudes, mV	26.6 (2.5)	27.8 (2.9)	19.0 (3.7)	<0.001
Motor unit number estimates	407 (67)	331 (61)	226 (64)	<0.001
Pulmonary vital capacity, L	3.97 (0.19)	3.6 (0.18)	2.99 (0.24)	<0.001
Timed 100-ft walk, s	28.5 (4.2)	24.0 (3.8)	29.2 (5.6)	0.01
Object turning, s	159 (17)	162 (9)	179 (18)	0.01
Object displacing, s	144 (8)	144 (5)	197 (8)	0.10
Pins and collars, s	337 (48)	348 (47)	440 (71)	<0.001
Screw manipulation, s	500 (111)	473 (139)	891 (242)	<0.001
Right elbow flexion, kg-cm	400 (65)	341 (49)	305 (41)	<0.001
Left elbow flexion, kg-cm	428 (52)	352 (35)	323 (42)	<0.0001
Right elbow extension, kg-cm	273 (45)	187 (30)	196 (35)	<0.0001
Left elbow extension, kg-cm	263 (52)	173 (32)	201 (33)	<0.0001
Right supination, kg-cm	53 (7)	41 (6)	42 (7)	<0.001
Left supination, kg-cm	52 (8)	45 (5)	41 (8)	<0.0001
Right grip, kg-cm	36 (4)	28 (3)	24 (4)	<0.0001
Left grip, kg-cm	32 (4)	26 (3)	23 (4)	<0.0001
Right pronation, kg-cm	47 (7)	51 (7)	52 (8)	0.08
Left pronation, kg-cm	46 (8)	43 (6)	47 (6)	0.29
Right knee flexion, ft-lb	40 (9)	41 (6)	38 (9)	0.74
Left knee flexion, ft-lb	41 (8)	37 (5)	35 (9)	0.15
Right knee extension, ft-lb	61 (11)	57 (8)	56 (13)	0.60
Left knee extension, ft-lb	66 (11)	59 (7)	61 (11)	0.10
Right foot dorsiflexion, ft-lb	16 (5)	14 (4)	15 (4)	0.36
Left foot dorsiflexion, ft-lb	14 (4)	11 (3)	11 (3)	0.08
Right foot plantarflexion, ft-lb	41 (10)	32 (7)	40 (9)	0.67
Left foot plantarflexion, ft-lb	38 (6)	29 (9)	40 (10)	0.34

loss to follow-up of nine (18%) of the subjects. It is not unexpected with such a long follow-up time to lose a small number as we did. There was no apparent difference between the nine subjects who withdrew consent and the remaining cohort. Nonetheless, the dropout rate, while acceptable, is not ideal for this study design.

References

1. Mulder DW, Rosenbaum RA, Layton DD Jr. Late Progression of poliomyelitis or forme fruste amyotrophic lateral sclerosis. Mayo Clin Proc 1972; 47:756–764.
2. Windebank AJ, Litchy WJ, Daube JR, Kurland LT, Codd MB, Iverson R. Late effects of paralytic poliomyelitis in Olmsted County, Minnesota. Neurology 1991;41:501–507.
3. Windebank AJ, Litchy WJ, Daube JR, Iverson R. Lack of progression of neurological deficit in survivors of paralytic polio: a 5-year prospective population-based study. Neurology 1996;46:80–84.
4. Dyck PJ, Kratz KM, Lehman KA, et al. The Rochester diabetic neuropathy study: design, criteria for types of neuropathy, selection bias, and reproducibility of neuropathic tests. Neurology 1991;41:799–807.
5. Daube JR. Estimating the number of motor units in a muscle. J Clin Neurophysiol 1995;12:585–594.
6. McComas AJ, Quartly C, Griggs RC. Early and late losses of motor units after poliomyelitis. Brain 1997;120:1415–1421.
7. Nollet F, Beelen A, Twisk JW, Lankhorst GJ, Visser M. Perceived health and physical functioning in postpoliomyelitis syndrome: a 6-year prospective follow-up study. Arch Phys Med Rehabil 2003;84:1048–1056.