

HOW TO INTERPRET NORMAL ELECTROMYOGRAPHIC FINDINGS IN PATIENTS WITH AN ALLEGED HISTORY OF POLIO

Arne Sandberg¹ and Erik Stålberg¹

From the Department of Clinical Neurophysiology, Uppsala University Hospital, Uppsala, Sweden

Objective: In some patients with a history of polio, the electromyography is normal, not showing the typical neurogenic signs. The aim of this study was to explain the normal findings in electromyography, especially in paralytic polio.

Design: Retrospective study.

Subjects/methods: Concentric needle electromyography, macro electromyography (including single fibre electromyography) and neurography were performed in various combinations in 688 patients with an alleged history of polio.

Results: Thirty-five patients with paralytic polio had normal or minimally abnormal neurophysiology. In 6 patients the diagnosis of polio was rejected and was instead found to be other diagnoses. Three patients had a very atypical history. Of the 26 with possible paralytic polio, 17 showed a strong suspicion of previous paralytic polio without any neurophysiological signs of degeneration of the anterior horn cells.

Conclusion: If neurophysiological findings are normal in patients with a history of polio, the original diagnosis may be incorrect. However, the absence of electromyography changes does not entirely exclude a previous history of polio with transient functional loss without degeneration of anterior horn cells vulnerable for later functional impairment.

Key words: polio sequel, electromyography, macro electromyography, single fibre electromyography.

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Correspondence address: Arne Sandberg, Department of Clinical Neurophysiology, Uppsala University Hospital, SE-751 85, Uppsala, Sweden. E-mail: arne.sandberg@nc.uas.lul.se

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INTRODUCTION

In acute paralytic polio the viral attack on spinal anterior horn cells causes dysfunction or cell death, which results in muscle weakness, typically with an asymmetric distribution (1). After the acute period of weakness, patients usually show either a full or partial recovery of strength. This recovery may result from a transient functional disturbance or be due to 2 compensatory mechanisms: re-innervation (collateral sprouting) and muscle fibre hypertrophy.

Polio patients often experience several decades with a relatively stable clinical condition (2).

In many patients new symptoms develop, sometimes progressing rapidly (3). These symptoms may include new weakness, muscular fatigue, atrophy, pain and cold intolerance. If new weakness with or without the above-mentioned symptom(s) develops after 10–15 years of stable condition, and clinical and electromyography (EMG) investigations indicate polio-induced lower motor neurone involvement, the patient is said to have developed post polio syndrome (3).

Patients with prior polio who develop new symptoms are often referred to a clinical neurophysiology laboratory for evaluation. The questions from the clinician may be: Is the neurophysiological investigation compatible with old polio? Are there other reasons than sequel of polio that may explain the newly experienced symptoms? Is the weakness explained by loss of motor neurones or are there other reasons, such as disuse or pain inhibition?

In most cases neurophysiological investigation shows signs of old polio, described in the literature (2, 4–11). This is typically characterized by different degrees of chronic (inactive) neurogenic EMG findings in different muscles, usually with an asymmetric distribution. Less frequently a slight to moderate degree of fibrillation potentials and/or the presence of positive sharp waves are found as signs of denervation. However, in a small number of cases there are no neurophysiological signs at all in these patients. This is not surprising when the patient presents with a vague history of polio. Having a patient with normal neurophysiology is also not surprising when a group of non-paralytic polio (NPP) patients presents with new symptoms that may be central in their origin (12).

In contradiction to this, we have found a group of patients that are presenting new symptoms after paralytic polio with normal neurophysiological findings.

The aim of the present retrospective study was to evaluate and clarify possible mechanisms behind the unexpected findings of normal neurophysiological investigation in patients who had had the diagnosis of status post polio for decades.

The study was accepted by the local ethics committee.

MATERIAL AND METHODS

Patients

Patients were referred from various centres of medical rehabilitation and neurology, thus the selection of patients was uncontrolled and the patient material was not considered to represent the general population of polio

patients. The material was obtained from our laboratory since 1989. Patients with negative neurophysiological findings were selected for the study according to specified criteria.

Inclusion criteria. Patients with a clinical diagnosis of "paralytic polio" (verified or strongly suspected) who presented progressive or new symptoms. The electrophysiological findings were normal and the studies must include the limb with (old or new) symptoms including the muscles in which there may be a involvement according to reported functional loss. Patients with minimally abnormal EMG could also be included if there was a reasonable explanation for the findings other than polio.

Exclusion criteria. A case was not included if the diagnosis was very uncertain or if the neurophysiological investigation was incomplete and did not include the area (limb) showing symptoms. A judgement was made that the neurophysiological investigation fulfils criteria of high standard and that a negative finding was not due to poor quality of data. Eight such patients were excluded.

A total of 688 patients with a history of polio were investigated in the laboratory. In this group a number of patients had negative neurophysiological findings and 35 patients remained when inclusion and exclusion criteria have been taken into consideration. An example of a quantitative EMG from the tibialis anterior muscles from a control, a polio patient with typical EMG and from a patient included in the present material are shown in Fig. 1. Initially, the analysis focused on the reliability of the original diagnosis; however, in most cases the diagnosis was difficult to ascertain. When the patients contracted acute polio, no routine antibody tests were available, thus the diagnosis was based on clinical features, epidemiological information and exclusion of other

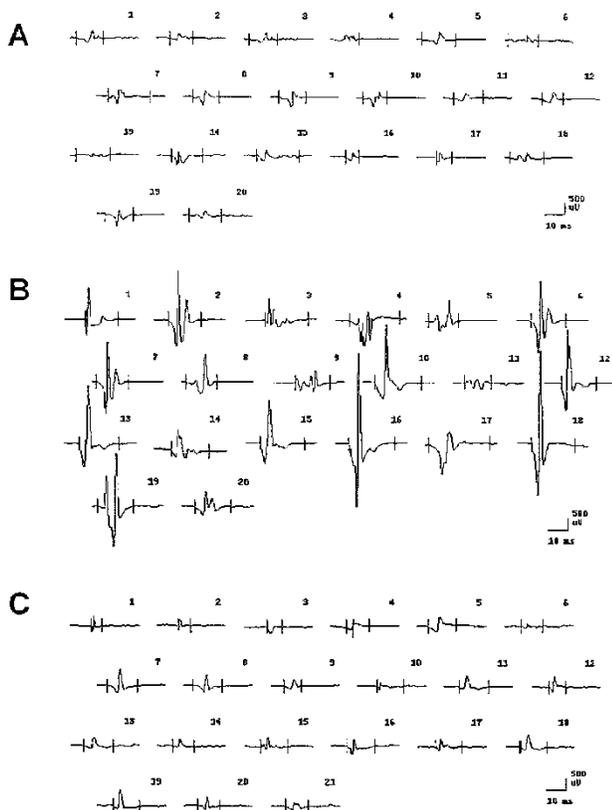


Fig. 1. Multi motor unit potential analysis of electromyography (EMG) with concentric needle electrode in tibialis anterior muscles. A = healthy control; B = typical EMG in a patient with a history of polio; C = from 1 of the patients in the present material, group "paralytic polio".

causes of the symptoms. The present late assessment of the diagnosis was based on scrutinizing patient files (available in 28 cases), contacting the referring physicians and recording the history given by each patient. The material collected was divided into 5 groups:

1. Another diagnosis. The patient had the diagnosis of polio sequel until the present EMG and further retrospective studies reached a different diagnosis.
2. Uncertain diagnosis. The patient was not hospitalized and had no defined acute spell of symptoms.
3. Polio meningitis. Symptoms of meningitis dominated the acute stage.
4. Suspected history of paralytic polio. The patient had acute symptoms, with or without hospitalization.
5. Paralytic polio. The patient had definite weakness, usually for months and recovered slowly; no other reason for the clinical condition has been revealed.

Symptoms, age and certainty of diagnosis are given in Table I.

Methods

The majority of EMG recordings were performed on Keypoint EMG equipment (Medtronic, Copenhagen). In some cases Counterpoint (Dantec, Copenhagen) was used.

Concentric needle EMG

Concentric needle EMG (CNEMG) electrodes were used (Medtronic, Copenhagen, Denmark and Medicotest, Copenhagen, Denmark).

Analysis of spontaneous activity at rest was assessed visually from 10 recording sites in each muscle, which typically included 2–3 needle insertions. Spontaneous activity was assessed visually by counting the number of recording sites which showed positive sharp waves or fibrillation potentials. Motor unit potential (MUP) analysis was performed automatically on approximately 20 different MUPs with the program Multi MUP analysis (13). Results were evaluated in relation to reference values used in the department (14). Quantitative analysis was not made in every muscle, however, a visual assessment was considered sufficient for reliable analysis. Instability of individual MUPs, or jiggle (15), was also assessed visually and graded stable, unstable or unstable with blocking. The interference pattern at strong voluntary contraction was either assessed with the inbuilt program (turns/amplitude analysis) or scored visually.

The result was presented as acute or inactive neurogenic changes, myopathic or normal findings in the investigated muscle. In typical patients with polio sequel the MUPs were typically of high amplitude, long duration and relatively stable at consecutive discharges, while the interference pattern was usually reduced.

Macro EMG

The standard macro EMG method was applied (16) using commercially available macro EMG needles (Medelec, Oxford Instruments, Abingdon, GBR and Medtronic, Copenhagen, Denmark). The recording electrode consisted of a modified single fibre EMG (SFEMG) electrode with the cannula insulated with Teflon, except for the distal 15 mm. An SFEMG recording surface was exposed 7.5 mm from the tip. Recording was made on 2 channels of commercially available EMG equipment (Keypoint, Medtronic, Copenhagen, Denmark). For initial recordings the Counterpoint (Dantec, Copenhagen, Denmark) was used. On the first channel, the SFEMG activity was displayed (using the cannula as reference), and used to identify the motor unit and to trigger an averaging procedure. The fibre density (FD) of the triggering single fibre electrode was recorded, and jitter and blocking were assessed visually.

On the second channel, the activity from the cannula (using a remote surface electrode as reference) was averaged until a smooth baseline and a constant macro MUP was obtained. Results were expressed as median values of individual amplitudes from at least 10 recordings. In polio the FD is increased and the macro signal, representing the total electrical size of the motor unit, is increased (17). Reference values included mean values of the median amplitudes from individual control subjects and 95% confidence for amplitude values (16).

Neurography

Neurography was performed according to the standard of the laboratory.

Table I. Characteristics of the patients. Age at acute polio, hospitalization, certainty of diagnosis and symptoms (sequel, new and present) are given

Patient	Age acute polio (years)	Duration of acute symptoms	Hospitalized	Sequel	"New" symptoms	Present symptoms					
						Pain	Fatigue	Chronic weakness	Accentuated weakness	New weakness in new area	Atrophy
<i>Other diagnosis</i>											
1	2	No data	Uncertain	No data	Weakness, pain	+				++	
2	1	No data	Yes	Tiredness, atrophy, pain	Hemi-weakness	+	++			+	+
3	8	No data	No	No significant	Hemi-weakness, pain	++				+	
4	10	18 months	Yes	Weakness	Weakness					++	
5	26	6 months	Yes	Uncertain if any	Fatigue		++				
6	25	No data	No data	No data	Weakness					++	
<i>Uncertain polio</i>											
7	6	No data	No	Fatigue, pain	Weakness, postural	++		+			
8	4	No data	Yes	Scoliosis, pain	Fatigue	++	+				
9	1	No data	No	Weakness, sensory symptoms	Weakness			+	++		+
<i>Meningitis</i>											
10	3	6 months	Uncertain	Postural problems	Hemi-weakness					++	
11	18	4 months	Yes	Pain, bladder disturbance	Weakness	+		+	++		
<i>Suspected polio</i>											
12	12	>1 month	Yes	None	Muscle cramps, pain	+					
13	31	3 months	Uncertain	Hemi-weakness	Weakness, pain	+				++	
14	0,7	No data	Uncertain	Hemi-weakness	Hemi-weakness, pain			+	++		
15	11	3 weeks	Uncertain	Restless legs	Weakness, sensory problems					++	
16	6	3 weeks	Yes	No significant	Fatigue		++				
17	6	No data	Uncertain	Weakness, atrophy	Fatigue, pain	++	+	+			+
18	2	No data	Yes	Fatigue, scoliosis	Pain, hemi-weakness	+				++	
<i>Paralytic polio</i>											
19	6	"short"	Yes	Hemi-weakness	Pain, fatigue	++		+			
20	11	No data	Uncertain	None	Pain, hemi-weakness	++				+	
21	3	3 weeks	Uncertain	Pain	Hemi-weakness	++		+			
22	5	2 months	Uncertain	Pain	Weakness	++				+	
23	6	3 months	Uncertain	Cold intolerance	Pain	++					
24	3	No data	Uncertain	Weakness, fatigue	Pronounced weakness, fatigue		+	++	++	+	
25	8	6 months	Yes	Hemi-weakness	Weakness			+	++		
26	13	3 months	Yes	Hemi-weakness, swallowing difficulties	Fatigue, hemi-weakness		+	+	++		
27	10	1 month	Yes	Pain	Fatigue	++					
28	2	No data	Yes	Pain	Pain, postural	++					
29	3	No data	No	Pain, scoliosis	Hemi-weakness, pain, sensory problems	++				+	
30	4	1 week	Yes	None	Weakness	++		+		++	
31	1	6 months	Uncertain	Pain, weakness	Thigh pain	++					
32	6	6 months	Yes	No significant	Pain	++					
33	6	3 months	Yes	None	Fatigue, pain, weakness	+				+	
34	1,5	6 months	Yes	Fatigue	Fatigue		++				
35	9	1 month	Yes	Pain	Pain	++					

+ = present symptom; ++ = dominating present symptom.

Surface electrodes were used for stimulation and recording of median, ulnar, peroneal and tibial nerves. Parameters included compound muscle action potential (CMAP) amplitude, area, latency, proximal-distal change in amplitude and duration, F-wave minimal latency minus M-latency and persistence. Sensory neurography was performed with surface stimulating and recording electrodes. Usually sural, median and ulnar nerves were investigated, but other sensory nerves were sometimes included. Reference values were from the laboratory.

RESULTS

Thirty-five patients were classified into the different groups with the following distribution: another diagnosis (6 patients), uncertain (3 patients), polio meningitis (2 patients), suspected history of polio (7 patients) and paralytic polio (17 patients).

Neurophysiological findings are summarized in Table II. In general, symptom profiles regarding degree of weakness, fatigability, pain and cold intolerance do not differ between the groups.

Individual groups

1. Other diagnoses (patients 1–6):

Two patients (patients 1 and 2 in Table I) had symptoms of unknown cause since childhood. The acute stage was defined by hemi-weakness. The diagnosis was cerebral palsy.

Patient 3 initially suffered from weakness in the legs, especially on the right side; a normal EMG and increased, asymmetric reflexes indicated a central disorder. The diagnosis in patient 3 was a right-sided central paresis of unknown cause with acute onset at the age of 9 years.

In patient 4 the acute stage included unconsciousness, treatment in a respirator and slow recovery. The patient had a verified stroke 25 years later, at the age of 35 years; thus, it is not unlikely that also the first episode was a stroke. A low thoracic spinal meningioma was diagnosed and treated surgically 26 years after the verified stroke. EMG showed central weakness in symptomatic leg muscles.

In patient 5 the normal EMG and slightly abnormal nerve conduction studies (NCS) initiated further search in patient files where it was clearly noted that the patient actually had acute polyradiculo-myelitis, i.e. Guillain-Barré syndrome (GBS).

Patient 6 had a normal clinical status and normal neurophysiology; however the iso-kinetic force test showed low test values, especially in both biceps; thus findings seem to support an earlier suspicion of a hysterical reaction.

2. Uncertain (patients 7–9):

Patient 7 suffered from stiffness in the acute stage, while present symptoms consist of pain and weakness.

In patient 8 there was unclear data available about the exact location of the acute paralysis; she had scoliosis, which later has been considered to be unrelated to polio. Currently the patient suffers from pain and fatigue.

Patient 9 showed slight atrophic leg muscles on 1 side at the age of 1.5 years. The present complaint was weakness in the leg with atrophy. Normal EMG was recorded from these 3 patients.

Table II. Electromyography (EMG) findings in the different patient groups based on new symptoms

New symptoms	Diagnosis					Paralytic
	Other	Uncertain	Meningitis	Suspected		
Weakness	3 (1 central weakness, 1 myopathy)	2	1 (central weakness and very slight neurogenic changes)	2 (1 NCS prolonged F responses)	5	
Hemi-weakness	2 (1 central weakness)		1 (central weakness)	2 (0)	4 (1 CTS)	
Pain	2 (1 central weakness, 1 myopathy)			5 (1 FD elevated)	9 (2 CTS, 1 central weakness)	
Fatigue	1 (1 polynuropathy)	1		2 (1 FD elevated)	6 (1 CTS)	
Sensory symptoms				1 (1 NCS prolonged F responses)	1	
Muscle cramps		1		1 (0)	1	
Postural symptoms		3	2	7	17	
Patients (EMG/ NCS pathological)	6 (2 central weakness, 1 myopathy, 1 NCS shows polynuropathy)		(2 central weakness and 1 very slight neurogenic changes)	(1 FD elevated, 1 NCS prolonged F responses)	(1 EMG + NCS shows CTS, 1 EMG central weakness, 1 NCS CTS)	

NCS = nerve conduction studies; FD = fibre density; CTS = carpal tunnel syndrome.

3. Meningitis (patients 10–11):

Both patients 10 and 11 had initial clinical signs of meningitis while patient 10 reported weakness in the leg and in respiratory muscles (respirator). The present EMG in leg muscles did not show any loss of neurones. Patient 11 had general weakness with gait problems as the dominating symptom in the acute stage, later back pain and bladder disturbance. The present EMG showed slight neurogenic EMG and was considered unrelated to polio but corresponded to clinical signs of a later developed lumbar radiculopathy. Both patients showed a reduced central drive of motor units in leg muscles, i.e. upper motor neurone sign. Present symptoms consisted of new general weakness in patient 10 and accentuated weakness in the hip and leg muscles in patient 11.

4. Suspected paralytic polio (patients 12–18):

Acute symptomatology was dominated by general weakness in patient 12, hemi-weakness in patients 13 and 14 and weakness in the legs in patients 15 and 16. The dominant acute stage symptom was somewhat unclear for patients 17 and 18. Presently, patients 13, 14, 15 and 18 had new or accentuated weakness, patients 12, 13, 17 and 18 had pain and patients 16 and 17 complained of fatigue.

EMG findings were normal except in one case showing FD elevation in 1 muscle with normal macro MUP amplitude. Patient 15 showed neurographic findings compatible with signs of a slight polyneuropathy based on prolonged F-latencies. However, no patients had neurophysiological findings that could be related to symptoms or clinical signs.

5. Paralytic polio (patients 19–35):

In the acute stage the dominating symptom was weakness; in 8 (patients 19–26) hemi-weakness, in 5 (patients 27–31) general weakness; in 3 (patients 32, 33 and 34) weakness in the legs and in 1 (patient 35) weakness in the back. In addition, patient 26 suffered from prominent dysphagia. At present 11 patients had pain, 4 had weakness and 2 had fatigue as the dominating symptom. Patients 19 and 20 also showed neurophysiological signs of carpal tunnel syndrome. Patient 35 showed EMG signs of reduced central activation but no signs of re-innervation or other neurogenic signs were seen. This was interpreted as a sign of possible central involvement. She had paralytic polio in the acute stage.

If the material of all 35 patients is analysed with regard to symptoms in Table II, it can be seen that the few abnormal neurophysiological findings were scattered among all groups of symptoms.

When SFEMG was included, no signs of pathological jitter or blocking were revealed.

The neurography investigations, when performed, were normal except in those where polyneuropathy or carpal tunnel syndrome had been indicated. The M-response amplitude was normal except in the few cases of clear carpal tunnel syndrome, understood as a coincidental finding in this study.

DISCUSSION

A total of 688 patients with a history of polio were investigated. All patients, except those selected for this study had clear neurogenic EMG findings, indicating re-innervation in some muscles. Often the involvement is quite asymmetric, as reported in the literature (18). A patient with clear abnormality in 1 muscle may show completely normal EMG findings in the contra-lateral or even neighbouring muscles. Usually the degree of abnormality parallels the symptomatology and also the initial involvement of anterior horn cells (AHC).

In this study, 35 patients were referred as status post polio and were found to have negative or remarkably meagre neurophysiological changes. The reason for this discrepancy needs further analysis; however, a few explanations have evolved during the detailed examination of documents and patients:

1. In 6 patients referred as polio survivors, clinical signs of central paresis (4 cases), a demyelinating polyneuropathy compatible with GBS (1 case) and hysterical reaction (1 case) were found. These patients will not be further discussed. It should be noted that these patients had until now been considered as having polio sequelae, thus a certain sensitivity from the physician is required to tell the patient the new situation and a mental strength from the patient to adapt to this.
2. In 3 cases the initial diagnosis of polio was very uncertain although the patients lived with the diagnosis of status post polio for more than 40 years. If the diagnosis was not polio, we are not surprised that the EMGs have been normal and the group needs no further discussion.
3. Two patients were given the diagnosis of polio in the acute stage with main symptoms of meningitis and muscular weakness. One was in a respirator. The EMG showed central involvement with difficulties in activating motor units with a high discharge rate at maximal effort. In 1 case the EMG showed very slight neurogenic changes, possibly from radiculopathy. Reflexes were reported as brisk in 1 of them, which is atypical in patients with a history of polio in whom reduced reflexes are frequently found. These cases illustrate the importance of analysing not only the MUP changes, but also the EMG pattern at strong voluntary effort.
4. Seven patients were suspected of having paralytic polio with acute illness and weakness. No other explanation however has evolved and no findings contradict that they have suffered from polio.
5. In 17 patients we cannot exclude but rather strongly suspect that the patient really had paralytic polio, although the neurophysiological investigation was unable to detect changes in EMG.

In all we have 17, probably 24 or possibly even 26 patients with paralytic polio but with negative or almost negative neurophysiology. Among the 24 patients with the highest likelihood of really having had acute polio, 16 patients had chronic or new weakness. The clinical picture is dominated by

pain (12 patients), new or accentuated weakness (8 patients) and fatigue (3 patients). In only 1 case (in the suspected group) atrophy of leg muscles was found.

What are the possible explanations for the negative neurophysiological findings in patients with weakness and/or fatigue after a history of paralytic polio? EMG is used as an adequate indicator of lower motor neurone involvement (dysfunction or degeneration) but also of central involvement causing reduced activation pattern.

The negative neurophysiological result may just be the result of incomplete neurophysiological investigation, i.e. wrong or insufficient number of symptomatic muscles studied. We have made sure that this is not a likely cause. In all patients symptomatic limbs and muscles have been investigated, sometimes quite extensively (quantitative MUP analysis, SFEMG, macro EMG in various combinations).

Another explanation of the negative neurophysiological findings is insensitivity of the neurophysiological methods to detect MUP changes after degeneration of a small number of neurones or central causes. In a simulation study (19) it was shown that mean values of conventional EMG parameters (duration, area, turns) and FD in SFEMG do not become definitely abnormal until a stage when more than 30–40% of neurones are lost followed by re-innervation. In interpretation of EMG in this study, individual MUP values outside a given limit or outliers have also been assessed. In this way the sensitivity is increased (20) and a value of 20% loss of neurones is not an unlikely lower limit. Among our patients with significant weakness, it was reasonable to assume a loss that exceeded 20–40% of functioning neurones alone explained the presented weakness. However, no neurophysiological changes were seen in these patients. Other possibilities must be considered. In our cases, the normal MUP could be due to persistent apraxia of the motor neurone or reduced re-innervation capacity. In both situations the MUP would be normal but the interference pattern would indicate an abnormality with reduction at full effort at least with more than 20% loss of neurones (21), which was not the case in these patients. Therefore the possibility was considered that the polio virus had affected the anterior horn cells functionally in the acute stage but with a minor degree of cell death. Such functional defect has been shown reversible even with up to 90% of abnormal motor neurones in the acute stage (22). During recovery of acute polio, patients showed an asymptotic recovery, i.e. initially fast and later more slow recovery (1). It seems likely that the early phase of recovery is due to the release of conduction impairment, which occurs before re-innervation or other compensatory mechanisms have become effective. In most of the patients in this study the functional defect did not seem to be a significant degeneration of AHCs.

Since we were unable to verify a significant lower motor neurone involvement, additional causes for muscle weakness should also be considered.

A possible cause is central involvement leading to improper motor performance. This should include central motor path-

ways, reflex support for muscle performance including the gamma-loop and pain inhibition. Bruno et al. (23) presented a hypothesis that the fatigue in post polio has a central aetiology; damage of reticular activating system and mono-aminergic systems based on magnetic resonance imaging studies. Therefore, weakness could also have this cause. Although we did not find an obviously reduced central drive in our study, we cannot exclude this mechanism. Reflexes are sometimes reduced out of proportion to weakness, which will be the subject of future studies. Pain inhibition is also a potential cause of muscle weakness that is difficult to detect during an EMG investigation, but reported in 16 of 24 patients. However, no patients had severe symptoms and all were able to produce a good contraction for the short test of EMG interference pattern. Thus, a discrepancy between test results (EMG and clinical findings at force testing) and reported subjective symptoms of weakness may be due to the different conditions during a test and the long-term use of muscles in daily life. The muscular atrophy seen in the patient with suspected polio is most likely due to disuse since the clinical picture is dominated by pain.

Muscular causes which must be considered include disturbed neuromuscular transmission, metabolic changes or defect contractility properties. These are not easy to detect since it has been difficult to pinpoint the cause of weakness and fatigability even in patients with obvious loss of lower motor neurones and fatigue (24, 25).

Regarding junctional defects, intermittent impulse blocking has been reported to contribute less than 6% of the recorded weakness (26). In the present study, impulse blocking was not present.

Metabolic factors have been discussed mainly in relation to fatigue in post polio (27–29). Borg and Henriksson (30) discussed low capillary density and decreased oxidative and glycolytic enzyme potentials might be important factors for the development of muscle weakness, fatigue and muscle pain; this should not lead to EMG changes. Nordgren et al. (31) found a decreased concentration of muscle creatine phosphate in nearly 50% of the studied muscles, indicating a disturbed muscle energy metabolism in a high percentage of patients with post polio muscular dysfunction, however, this was not correlated to the post polio symptoms. A study regarding muscle fatigue in polio that included force measurements and repetitive nerve stimulation was performed by Stibrant-Sunnerhagen et al. (25). Fatigue developed similarly for both controls and patients during activity with the only deviation from normal being a slow recovery of force and EMG after exhaustion, compatible with the results by Rodriguez and Agre (27). Changes in contractile properties should cause weakness without EMG changes.

Larsson et al. (32) investigated contractile and firing rate properties in prior polio. They found a difference in these parameters when comparing the findings with those in normal subjects. However, the authors commented that their polio patients suffered from a loss of a large proportion of the motor neurone pool in the investigation, therefore this result may not

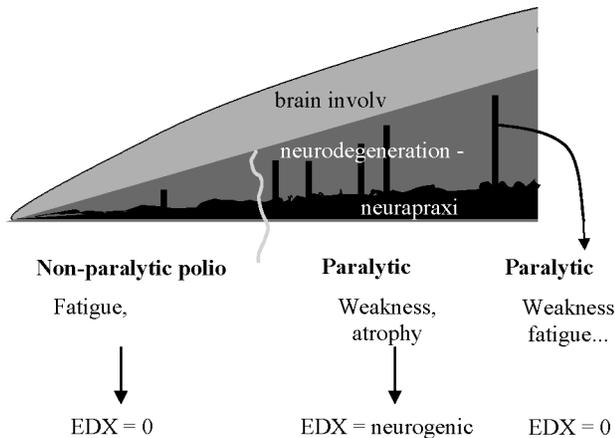


Fig. 2. Spectrum of different types of involvement in acute polio. In mild cases (non-paralytic) there is a different degree of central involvement combined with a small or negligible degeneration of anterior horn cells (AHC). Electromyography (EMG) shows no sign of AHC involvement. In paralytic cases there is commonly signs of different degrees of re-innervation according to AHC involvement. A small number of the paralytic cases are showing a normal EMG investigation despite a rather pronounced symptomatology both in the acute and post polio phase, not indicating any AHC death. In the acute phase this could be due to apraxia. Possible mechanisms for the late expressed symptoms are discussed in the text. The curved line indicates the border between the non-paralytic and the paralytic cases. EDX = electrodiagnostic examination

be relevant in our group of patients that has no signs of AHC loss.

Unable to find a single reason for the combination of weakness (and fatigue) and normal EMG in some patients with a history of paralytic polio, we express the hypothesis that there is a spectrum of degree of involvement, from apraxia to degeneration of central neurones and of AHCs in the acute phase of polio (Fig. 2). Acute central involvement often shows restitution, but may remain as symptoms that are part of the symptomatology in late polio, e.g. pain, fatigue, cold intolerance and possibly central weakness. Regarding AHCs it can be shown that a proportion of the neurones usually degenerate but some may recover after a transient functional loss (apraxic reaction). In our large group of patients passing the EMG laboratory we may have found a small group where the virus has not caused a significant degree of loss of AHCs. The initially inflicted but recovered neurones may later in life have a reduced functional safety margin, resulting in weakness and fatigue. One can speculate that trans-synaptic changes or a vulnerability to metabolic changes in the recovered motor neurones give rise to the presented symptoms (22, 33).

CONCLUSION

Our findings suggest that absence of EMG signs of lower motor neurone dysfunction is unusual in cases of polio. Usually other diagnostic alternatives to the present symptoms should be identified when EMG is normal. However, negative neurophysi-

ology may occur in some cases with a history of paralytic polio. In those, the initial weakness may have been due mainly to central involvement, e.g. meningitis or to a functional impairment of the lower motor neurone, not causing denervation/re-innervation. The dysfunction of central or lower motor neurones may remain and give rise to muscular weakness and fatigue not detectable using electrophysiological methods.

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