Journal of Electromyography and Kinesiology 46 (2019) 35–40

Contents lists available at ScienceDirect

Journal of Electromyography and Kinesiology

journal homepage: www.elsevier.com/locate/jelekin

Motor Unit Number Index (MUNIX) as a biomarker of motor unit loss in post-polio syndrome versus needle EMG

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ARTICLE INFO

Keywords:
Post-polio syndrome
Motor unit
Viral infection
Motor unit number index
MUNIX

ABSTRACT

MUNIX method (Motor Unit Number Index) had been not used to assess number of motor neurons in post-polio syndrome in contrary to needle electromyography.

Objectives: To confirm if MUNIX reflects motor unit loss and clinical stage and to assess difference in MUNIX and EMG results between muscles in different stage.

Methods: 132 Muscles (MUNIX) and 96 (EMG) in 12 patients were studied and divided into groups: with normal strength(N), stable weakness and atrophy(S), new weakness and atrophy(W).

Results: In PPS group MUNIX global was 561.36 ± 282.6 (right 6 muscles) and 561.27 ± 281.1 (left) significantly lower than in control group (six muscles 1139.6 ± 164.5) ($p < 0.05$). MUNIX global correlated with MRC global. MUNIX was greater in muscles with normal strength (95–100% of normal values) than in those with stable weakness (48%-0% of normal values) and new weakness (65%-0% of normal values). Respectively to clinical stage of muscle MUP (motor unit potential) amplitude increased to 350% of normal value, from 250% to 110%, and from 300% to 700%. No correlation was found between MUP parameters and MRC values.

Conclusions: MUNIX reflects motor dysfunction and could be a good biomarker for loss of motor neurons in PPS.

1. Introduction

The aetiology of poliomyelitis or Heine-Medin disease is an infection with Poliovirus (an RNA Enterovirus belonging to the Picornaviridae family) which attacks motor neurons. Core symptoms of poliomyelitis include skeletal muscle weakness and wasting that results from death of infected anterior horn cells (Boyer et al., 2010). At least 15 years after poliomyelitis symptoms, new symptoms are described in polio survivors and the diagnostic criteria of post-polio syndrome (PPS) were published by March of Dimes (2001) as below:

1. Prior paralytic poliomyelitis with evidence of motor neuron loss, as confirmed by history of the acute paralytic illness, signs of residual weakness and atrophy of muscles on neuromuscular examination, and signs of nerve damage on electromyography (EMG).
2. A period of partial or complete functional recovery after acute paralytic poliomyelitis, followed by an interval (usually 15 years or more) of stable neuromuscular function.
3. Gradual onset of progressive and persistent new muscle weakness in previously unaffected and healthy muscle or abnormal muscle fatigability (decreased endurance), with or without generalized fatigue, muscle atrophy, or muscle and joint pain and cold intolerance
4. Symptoms that persist for at least a year.
5. Exclusion of other neuromuscular, medical, and orthopedic problems as causes of symptoms (Post Polio Syndrome, March of Dimes, 2001) (March of Dimes, 2001).

In 2006 EFNS guidelines on diagnosis and management of post-polio syndrome was reported (Farbu et al., 2006) and the Halstead's definition of PPS was recommended as diagnostic criteria. The term of PPS was introduced by Halstead and the first criteria of PPS were formulated as (1) confirmed history of polio (2) partially of fairy complete neurological and functional recovery after acute period (3) at least 15 years period of stability (4) two or more of the following health problems occurring after the stable period as extensive fatigue, muscle and/or joint pain, new weakness in muscles previously affected or unaffected , new muscle atrophy, functional loss, cold intolerance (4) no other medical explanation found (Halstead and Rossi, 1985). In revision the criteria Halstead in 1991 added "gradual or abrupt onset of new neurogenic weakness" with or without co-existing symptoms

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https://doi.org/10.1016/j.jelekin.2019.03.006
Received 24 October 2018; Received in revised form 16 February 2019; Accepted 8 March 2019

(Halstead, 1991), what caused that the Halstead 's and March of Dimes criteria are very similar. In addition to the above criteria in Boyer's update article the attention was paid on more rare symptoms as sleep disorders, respiratory disorders, dysphagia, dysarthria, fasciculations and joint deformities. (Boyer et al., 2010)

Depending on the study, 25 to 60% of polio survivors are reported to experience post-polio syndrome (Ragonese et al., 2005; Takemura et al., 2004).

Several hypotheses were suggested, among them persistence of viral genetic material able to interfere with and deregulate the immune system response. Inflammatory changes found in the spinal cord may suggest persistent polio infection, autoimmune attack and increased vulnerability of damaged neurons (Pezeshkpour and Dalakas, 1987).

It is assumed that in the spinal cord damaged during acute polio, with destruction of a certain number of cells, another pool of preserved overworked motor neurons dies gradually after many years (Wiechers and Hubbell, 1981). In the acute phase of the disease, degeneration of motor neurons leads to denervation of muscle fibers followed by terminal axon sprouting to muscle fibers primarily orphaned by the loss of their original motor neurons.

The motor neurons are overloaded as they functionally and metabolically serve an excessive number of muscle fibers as a result of their reinnervation. Weakness of new muscles is probably a consequence of an imbalance between denervation and re-innervation of muscle fibers (Baj et al., 2015; Wiechers and Hubbell, 1981).

Decompensation of secondary re-innervation could be due to both processes, death of overworked motor neurons and physiological loss due to aging (confirmed in pathological (Emeryk et al., 1990) and also electrophysiological studies (MUNE) (Doherty et al., 1993; Doherty and Brown, 1993; Gawel and Kuzma-Kozakiewicz, 2016), which could be more intense in polio survivors.

Quantitative EMG study may estimate parametrically the change in the architecture of a motor unit in post-polio patients. Re-innervation of denervated muscle fibers through sprouting from the remaining motor axons results in an enlarged motor unit territory of the surviving motor neurons (Buchthal and Kamieniecka, 1982; Lange, 1995). These abnormalities are demonstrated by the finding of a markedly increased motor unit potential (MUP) amplitude, its prolonged duration, and increased area by quantitative EMG studies (Emeryk et al., 1990; Lange, 1995) but, it is impossible to assess the actual loss of motor units.

One of the newest electrophysiological methods, which had not been used in PPS yet, reflecting the degree of motor unit loss is the Motor Unit Number Index (MUNIX) method, a non-invasive tool which can be applied to both distal and proximal muscle. A mathematical model based on the compound motor action potential was applied and the surface EMG interference pattern (SIP) was calculated based on a graded effort. In the final step, the CMAP and SIP data are imported to an analysis software written by Nandedkar. For each SIP, its area and power are measured, and together with CMAP area and power, the "ideal case motor unit count" (ICMUC) is computed. The number and size of motor units recruited at different force levels is reflected by the ICMUC versus SIP area (Nandedkar et al., 1988).

MUNIX has never been used to assess the loss of motor units in the muscles affected by the postpolio process, however, there have been a single study using the MUNE method (Sorenson et al., 2006), which is used mostly for the muscles of the hands and does not test proximal muscles, as it is possible in MUNIX, giving the opportunity of a comprehensive assessment.

The aim of the study was: (1) to assess the degree of motor unit loss in MUNIX, in the muscles with different clinical stages (with normal strength (N), with stable weakness (S) or new weakness (W) (2) to assess the value of the MUNIX method in terms of reflecting clinical stage in patients with post-polio syndrome (3) to assess the degree of changes in MUP parameters in routine needle EMG regarding muscles with different clinical stages (4) to compare the abnormalities in EMG and MUNIX in muscles with different clinical stages.

2. Methods

The study group consisted of 12 patients with PPS at the mean age of 63 ± 8 years, (6 M- 50%). All patients fulfilled the March of Dimes criteria for PPS (March of Dimes, 2001). Patients with neurological abnormalities such as a history of neuropathy, advanced discopathy, or injuries of upper or lower extremities were excluded.

The mean duration of the new symptoms (PPS) was 6.5 yrs (2–20 yrs). The protocol of the study was approved by the Ethics Committee at the Medical University of Warsaw. (The approvement No KB/189/2016).

Before every MUNIX measurement, a manual muscle testing according to the Medical Research Council Scale for Manual Muscle Testing (MRC) was performed in each investigated muscle (0-complete paralysis, 1-minimal contraction, 2-active movement with gravity eliminating 3-weak contraction against gravidity, 4-active movement against gravidity and resistance, 5-normal strength). Overall, we studied 132 muscles, divided into three groups: with normal strength (N); (n = 57), with stable weakness and atrophy (S); (n = 39), and with new weakness and atrophy (W), (n = 36). We defined muscles with "stable weakness and atrophy" as those that were originally weak at the time of the polio infection, and clinically its clinical features have not changed, so presently its neuromuscular function is stable and the weakness and atrophy are the same as after acute period of the infection. We defined muscles with "new weakness and atrophy" as muscles that were originally weak at the time of the infection, then recovered fully clinically, and subsequently weakened after an period of recovery.

We performed MUNIX measurements in the abductor pollicis brevis (APB), abductor digiti minimi (ADM), biceps brachii (BB), tibial anterior (TA), extensor digitorum brevis (EDB), and abductor hallucis (AH) muscles. MUNIX was recorded on both sides of the body. The global MUNIX score (6 muscles at one side) was calculated for the examined muscles and MUNIX score was calculated for every single muscle. We used the Keypoint Classic Natus Apparatus. If CMAP amplitude was lower than 0.5 mV, a given muscle was not used for MUNIX calculation, and MUNIX was rated as zero (12 muscles).

The needle electromyography (EMG) was performed in 96 muscles. MUPs were recorded using concentric needle electrodes, with Keypoint Classic Natus Apparatus, with the band pass of 20 Hz–10 kHz. MUPs parameters were obtained in FDI, BB, VL, and TA muscles on both sides. MUP parameters including amplitude, duration, and size index (SI) were analyzed (Buchthal and Kamieniecka,1982; Nandedkar et al., 1988; Sonoo and Stalberg, 1993; Stålberg et al., 1996; Dumitru et al., 1997). At least 20 MUPs at minimal voluntary activation were recorded. The MUP amplitude from the highest positive to the lowest negative deflection of the curve was measured. The MUP duration from its first deflection from the baseline to its final return was analyzed, and start- and end-points of the deflections were selected manually. SI, was calculated automatically as $2\log(amplitude) + \frac{area}{amplitude}$ for each single MUP (Sonoo and Stalberg, 1993). This index minimizes the influence of the position of the electrodes, which markedly affects the value of the amplitude, and it is seemed to be more important than simple parameters, such as amplitude or duration (Hałsmanowa-Petrusewicz, 2008). The EMG results were compared with the normal values adopted by the Neurophysiological Unit at the Department of Neurology, Medical University of Warsaw.

Wherever statistical significance was estimated using p it was based on Student's t test. We have used Student's t for both the estimate of the significance of Spearman paired ranks correlation as well as for comparison of means.

The data: MUNIX of each muscle : ADM, APB, BB, TA, AH, EDB; MUNIX global- a sum for 6 muscles of one side- separately for left and right side; MRC of each muscle: ADM, APB, BB, TA, AH, EDB, left and right; MRC global-a sum for the 6 muscles of one side, separately for left and right side; MUP parameters as amplitude, duration, SI of 4 tested

muscles as FID, BB, VL, TA of both sides) were collected and analysed using statistical methods to answer the four questions listed above as the aims of the study. In what follows consecutive steps of analysis are marked as aim 1 to 4.

To assess the degree of motor unit loss in MUNIX, in the muscles with different clinical stages (aim no 1) (with normal strength N, with stable weakness S or new weakness W) values of global MUNIX index and MUNIX for individual muscles were calculated and compared with the normal value.

To verify a hypotheses that MUNIX reflects motor unit loss and clinical stage (aim no 2) the relationship between MUNIX and MRC was analysed. Data for a given muscle (MUNIX, MRC) in all examined patients were collected and a preliminary correlation was examined using Pearson and Spearman correlation coefficients. For each of these coefficients, an error of its determination was calculated. Significance of a preliminary correlation was verified using the Student *t*-test. It turned out that the criterion based on the Spearman correlation coefficient is more restrictive hence it was used for further data analyses. For the muscles where the Spearman coefficient indicated a possible correlation, a graph of MRC vs MUNIX was made and examined.

To assess the degree of changes in MUP parameters in routine needle EMG regarding muscles with different clinical stages (aim no 3 and 4) mean values of MUP parameters were calculated for individual muscle and presented as a percentage of the normal value for a given muscle.

The graphs for MUP parameters as a function of clinical stages for various cases were obtained from subsets of the measured data grouped into clinical stages. For each patient, muscles for a given case (normal-N, stable weakness /with atrophy-S, or new weakness /with atrophy-W) were selected. For each muscle, normal values of MUNIX were taken from our previous study (Gawel and Kuzma-Kozakiewicz, 2016), and the measured values were divided by the reference value (%). Because it was very difficult to present the total results from different muscles, for which there are different normal ranges in MUP parameters and MUNIX, it was decided to present the results as values in percentages and to compare the values in percentages. The data (MUNIX of each muscle, MUNIX global, MRC of each muscle, MRC global, MUP parameters as amplitude, duration, SI) obtained were grouped according to the clinical stage (N, S, W) and the mean value was calculated for each clinical stage. The percentage values are labeled as Amplitude%, Duration%, SI% and MUNIX%.

3. Results

3.1. MUNIX global and MUNIX for individual muscles (aim no 1)

In the PPS group, the MUNIX global was 561.36 ± 282.6 for the right side and 561.27 ± 281.1 for the left side, significant lower -compared to mean MUNIX global 1139.6 ± 164.5 in healthy volunteers measured as in PPS patients in 6 muscles of one side ($p < 0.05$).

A significant differences between the MUNIX values for every examined muscle (APB, ADM, BB, TA, EDB, AH) in study groups and controls were found ($p < 0.05$) except for BB Table 1.

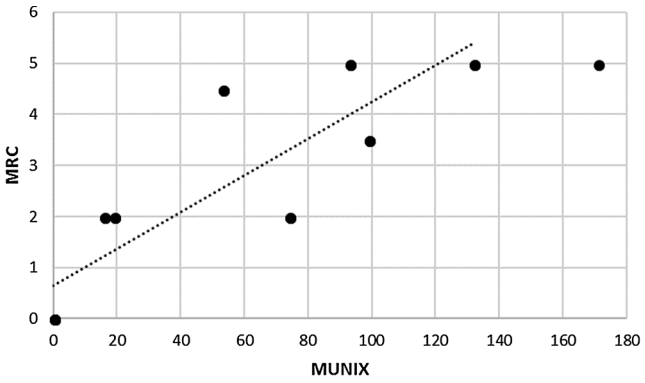

Fig. 1. Dependence between MUNIX in the TA (right) and the MRC score. TA, tibialis anterior; MRC, Medical Research Council, MUNIX, motor unit number index.

3.2. Correlation between MUNIX and MRC (aim no 2)

The MUNIX global value correlated with the MRC global for 6 muscles (ABP, ADM, BB, TA, AH, EDB of one side) and separately, the MUNIX value for every muscle was correlated with MRC each of the following muscle, ADM, EDB, TA, AH ($p < 0.05$) but not for APB, BB for right hand side, and with EDB, TA, AH but not for APB, ADM, BB for left hand side. In [Figs. 1, 2] the data for MRC are plotted as a function of MUNIX for TA (right) and AH (right). We have observed that although the linear correlation between MUNIX and MRC is significant ($p < 0.05$), a better description of the dependence of MRC on MUNIX is such that at small values of MUNIX ($\lesssim 100 - 150$), MRC increases with the increase in MUNIX, while above a certain value of MUNIX, MRC stops to increase. We have fitted the data on the rising part of the dependence of MRC on MUNIX with a straight line. The results are presented in [Figs. 1, 2].

In Fig. 3, a combined plot of an average MRC for a given MUNIX range is shown for all data for the cases with a significant correlation. For all the muscles with a significant correlation between MUNIX and MRC, average values for MRC were calculated for MUNIX intervals with the width of 30. For the first bin, $0 \leq MUNIX < 30$. Altogether, there were 77 data points. From these data, those for which MUNIX was in the range corresponding to the first bin were averaged and assigned coordinates $(0, \langle MRC\,(0 \leq MUNIX < 30)\rangle)$. The same was repeated for consecutive bins. For the data for which $MUNIX_{30}$ was smaller than 150, a straight line has been fitted. It illustrates that for smaller MUNIX values, MRC increases with the increase in MUNIX, while for larger MUNIX values MRC remains constant.

3.3. Relationship between MUP parameters and MRC (aim no 3)

In addition, the relationship between MRC and motor unit potential parameters (amplitude, duration, size index) obtained in muscles during a slight voluntary effort was analyzed. Only one of the parameters, the percentage of polyphasic potentials in the right BB, correlated inversely with MRC ($p < 0.05$). No correlations were found between all other MUP parameters and MRC values in all other muscles.

In muscles with normal strength, as well as with stable weakness

Table 1
MUNIX values in PPS patients and controls.

MUNIX	MUNIX megascore	APB*	ADM*	BB	TA*	EDB*	AH*
PPS Patients Right	561.4 ± 282.6	88.8 ± 60.5	98.2 ± 52.1	178.5 ± 67.9	59.7 ± 56.2	25.6 ± 14.1	110.5 ± 104.2
PPS Patients Left	561.3 ± 281.1	91.5 ± 48.4	114.9 ± 56.2	165.5 ± 87.9	53.8 ± 71.3	19.6 ± 12.2	115.9 ± 76.5
Healthy controls	1139.6 ± 164.5	170.7 ± 37.8	168.9 ± 53.1	165.0 ± 56.0	126.0 ± 31.0	132.9 ± 109.6	375.4 ± 193.3

*$p < 0.05$ (the correlation between MUNIX and MRC), MUSCLES: APB – abductor pollicis brevis, ADM – abductor digiti minimi, BB – biceps brachii, TA – tibialis anterior, EDB – extensor digitorum brevis, AH – adductor hallucis.

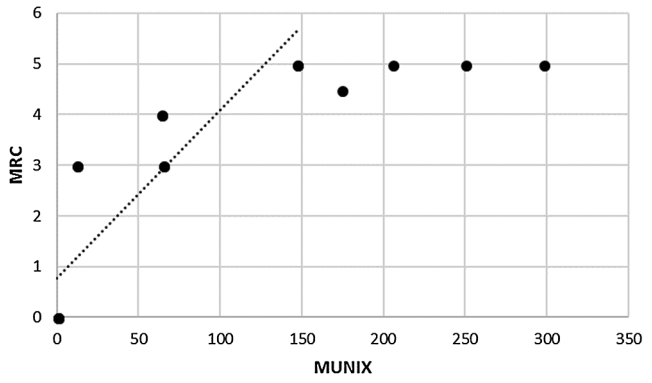

Fig. 2. Dependence between MUNIX in the AH (right) and the MRC. AH, abductor hallucis; MRC, Medical Research Council; MUNIX, motor unit number index.

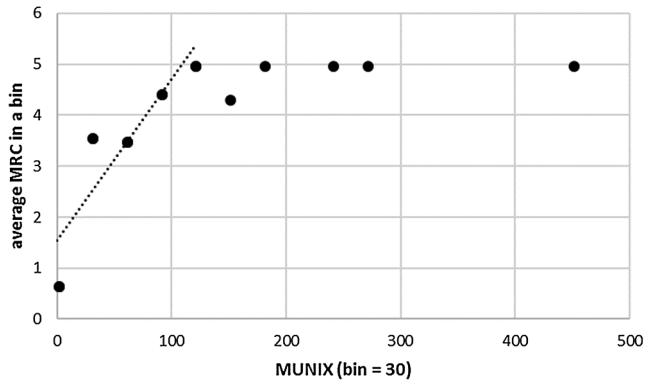

Fig. 3. Dependence of the average MRC on MUNIX. MUNIX values grouped into bins with the width of 30. Average MRC calculated for each bin with data. A straight line is drawn for data with $MUNIX_{30}$ smaller than 150.

and atrophy, and also with new weakness and atrophy the relation between MUP parameters and MRC was nor found in opposite to the correlation between MUNIX and MRC ($p < 0.05$)

3.4. Changes of MUP parameters and MUNIX in different subgroups of muscles (aim no 4)

It was found that for all examined muscles, analyzed together (N + S + W) in relation to the clinical stage (from MRC 5 to 0), MUP amplitude increased from 400% to 800% of the normal value, SI increased from 200% to 350%, and duration increased from 100% to 150% [Fig. 4A]. Depending on the clinical stage (from MRC 5 to 0), MUNIX decreased from 80% to 5% of the values in healthy controls [Fig. 4B]. In muscle with preserved strength (N) (MRC 5) without any new post-polio symptoms, MUP amplitude increased to 350% of the normal value, SI and duration was about 100%, and MUNIX was about 95–100%. In muscle with stable weakness and atrophy (S) analyzed in relation to the clinical stage, MUP amplitude increased from 250% to 1100% of the normal value, SI increased from 200% to 400%, and duration increased from 110% to 180%. [Fig. 5A]. Depending on the clinical stage (from MRC 5 to 0), MUNIX decreased from 48% to 0% of the values in healthy controls [Fig. 5B].

In muscle with new weakness and atrophy analyzed in relation to the clinical stage, MUP amplitude varied from 300% to 700% of the normal value, SI increased from 250% to 300%, and duration increased from 100% to 150%. MUNIX decreased from 65% to 0% of the values in healthy controls, depending on the clinical stage (from MRC 5 to 0).

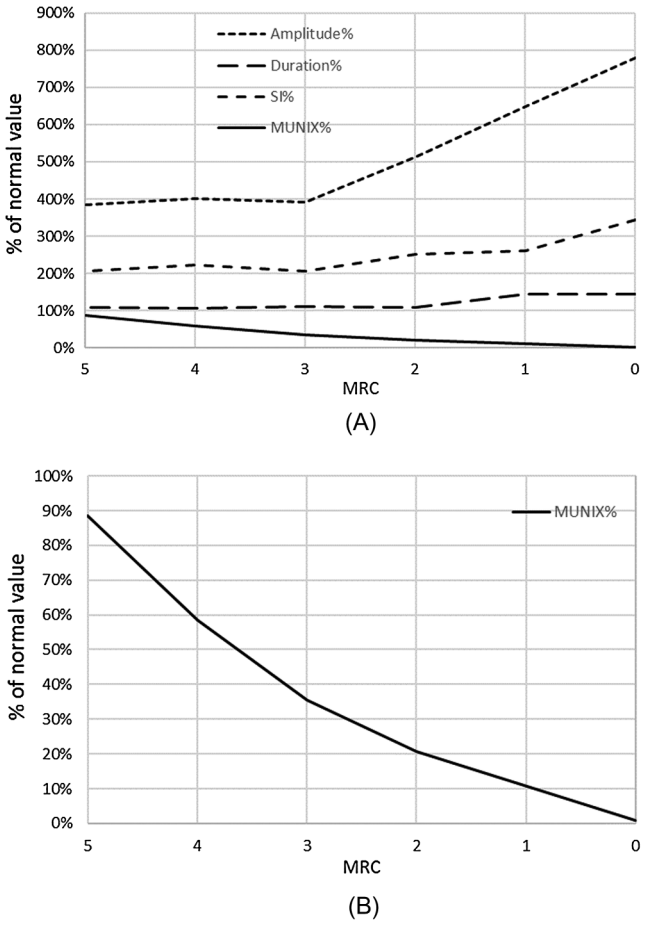

Fig. 4. A MUP parameters in relation to the clinical stage in all examined muscles; SI- Size Index. 4B MUNIX % values in relation to the clinical stage in all examined muscles. One hundred percent is regarded a normal value. MRC, Medical Research Council.

4. Discussion

Reorganization of the motor unit as a result of coexisting processes of denervation and reinnervation initially allows full compensation but finally leads to decompensation of muscle fiber innervation. In the initial disease stage, MUP parameter values are increased because of efficient reinnervation of muscle fibers by collateral axonal sprouting (for amplitude up to 1000% of the normal value, for duration up to 40–50% of the normal value) (Emeryk- Szajewska et al., 2003). Reinnervation by sprouts, leading to temporal dispersion of a single fiber potential, is reflected by long-duration MUPs and an increased fiber density. However, the most impressive evidence for extensive reinnervation was obtained by another EMG method, Macro-EMG study by Lange, where the amplitude of 200% was found (Lange, 1995).

In the study by Lange (Lange, 1995), all the examined muscles were divided into groups with normal strength, with stable weakness and atrophy, and with new weakness and atrophy. The most marked reinnervation was found in muscles with normal strength and with stable weakness, in contrast to those with new weakness in which the MUP parameters were not markedly increased (Lange, 1995). In the study by Emeryk et al., a decreased value of MUP amplitude was noted in muscle with marked weakness (Emeryk et al., 1990).

Regarding the most important issues, the results of the present study are in accordance with the previous studies. In muscles with preserved strength (N), MUP amplitude increased to 350%, with duration and SI in the normal range, compared to the study by Lange, where in muscle with normal strength, macroamplitude was increased to 180%, as were

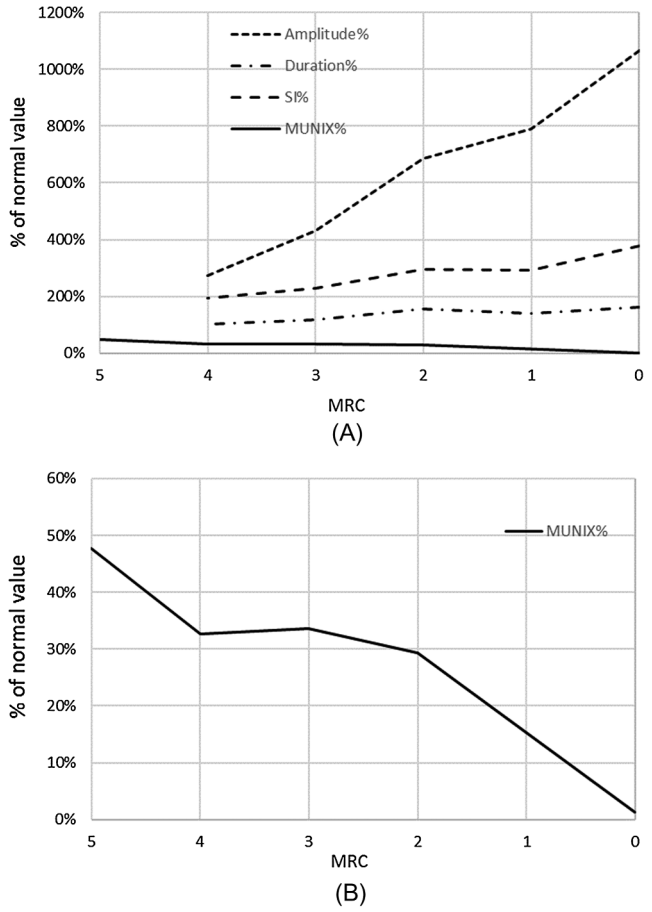

Fig. 5. A MUP parameters in relation to the clinical stage in muscles with stable weakness and atrophy; SI- Size Index. 5B MUNIX % values in relation to the clinical stage. One hundred percent is regarded a normal value. MRC, Medical Research Council.

MUP duration and amplitude (220%). The unexpected were normal values of MUNIX in contrary to increased values of MUP parameters in muscle with preserved strength. We suppose that it was due to primary imperceptible loss of motor neurons leading to slight re-innervation with compensation although the whole number of motor units remained within the normal range (95–100%).

The most marked changes suggesting long-lasting chronic re-innervation were found in muscles with stable weakness (MUP amplitude increased to 1110%, duration to180%, SI to 400%), which is compatible with the study by Lange who found that in muscles with partial recovery, the MUP amplitude increased to 900%, and duration to 300% (Lange, 1995). The loss of motor units reflected by MUNIX was up to 48%.

It was interesting, that less changes in MUP parameters values were found in muscles with “new” weakness” (amplitude was increased to 700%, duration to 150%, SI to 300%) than in muscles with “stable” weakness. Our suggestion was that in the case of muscle with new weakness the process of reinervation has been lasting for a shorter time (mean 6.5 years) than in the muscles with stable weakness (mean 61 years), such a significant reinervation reflecting in the values of MUP parameters has not been obtained. However, till now it is not clear if new symptoms are associated with denervation-reinervation processes which are very slow, but last permanently for many years and start just after polio or start after a several years after primary disorder and last for a short time.

The MRC in BB was higher than in other muscles especially in muscles in lower extremities. For example mean MRC in BB (right and left) was 4.375, mean MRC in VL (right and left) was 2,46 so it was significant difference. Moreover in most patients the paresis was more marked in lower extremities, and the patients used wheelchair or elbow crutches so presumably the higher MUNIX in BB than in controls could be the result of overworking and higher CMAP amplitude due to increased mass of muscle.

A larger loss of motor units revealed by MUNIX method was found in muscles with new weakness compared to muscles with stable weakness (up to 65% versus 48%). Probably, a part of the motor units is lost in the first stage of the disease, followed by compensation due to secondary reinnervation (muscles with stable weakness), and then, in the next stage of the disease, further loss of motor neurons occurs (muscles with new weakness) which is confirmed by MUNIX.

Till now, the MUNIX method was of interest to estimate the number of surviving motor neurons to study disease progression in amyotrophic lateral sclerosis (ALS) (Nandedkar et al., 2010; Neuwirth et al., 2010). In ALS patients in the study by Neuwirth, the MUNIX global at the first examination was reduced by 70% compared to healthy volunteers, indicating that ALS patients have already lost a large number of motoneurons at the time of the diagnosis. It is obvious that motor neuron deficit is the reason for clinical symptoms in polio and post-polio syndrome, and it was confirmed electrophysiologically in our study, which revealed a decrease in MUNIX global by 50% in all patients.

By means of electrophysiological tests (MUNIX and quantitative needle EMG), it is possible to define separately a loss of motor neurons and reinnervation of muscle fibers, which may give a deeper insight to the underlying degenerative process in post-polio syndrome.

To date, only single studies analyzed motor unit loss in post-polio syndrome using motor unit number estimation method (MUNE) (McComas et al., 1997; Sorenson et al., 2006), but not MUNIX. The study by Sorenson demonstrated that in post-polio patients, MUNE counts from the hand and foot muscles are associated with the overall strength of the individual muscle, and it was performed on both median nerves with recording over thenar muscles and on both peroneal nerves with recording over the extensor digitorum brevis (Sorenson et al., 2006). MUNIX method could be used in both distal and proximal muscles not only in distal muscles as the most of MUNE methods. The limitation of MUNE method used in Sorenson’s study was the variability of the data and poor reproducibility, when the high reproducibility of MUNIX was well documented (Neuwirth et al., 2016; Neuwirth et al., 2010). Our study confirms that MUNIX is a valuable method that reflects the loss of motor neurons, correlating with the clinical muscle assessment using the MRC scale in every examined muscle except biceps brachii. It could be explained by the fact that technically, BB is a more difficult muscle for MUNIX tests than others due to a relatively greater mass of muscle causing that CMAP with supramaximal amplitude, decisive for the MUNIX method, is difficult to obtain. In our study, we found no correlation between MUP parameters and the MRC score, which confirms that in contrast to MUNIX, the MUP parameters are in incomplete reflection of clinical muscle dysfunction. Contrary to routine EMG, MUNIX is a good indicator of clinical muscle dysfunction and the MUNIX global seems to be the most useful indicator.

Our study confirms that the MUNIX method is a sensitive tool reflecting motor dysfunction in post-polio syndrome and gives a new insight to the underlying process in post-polio syndrome, supplementing routine needle electromyography. MUNIX directly assesses the loss of the lower motor neurons and the number of motor units is changed dependently on stage of clinical stage - it is normal or slightly decreased in muscles with preserved strength, evidently decreased in muscle with stable strength and most markedly decreased in muscles with new weakness.

Conflict of interest

None of the authors has any conflict of interest to disclose

Financial disclosure statement

Nothing to declare.

Ethical publication statement

We confirm that we have read the Journal's position on issues involved in ethical publication and affirm that this report is consistent with those guidelines.

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