# Quantitative anal sphincter EMG in multisystem atrophy and 100 controls

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### Abstract

*Objective*—To evaluate data of quantitative anal sphincter EMG in normal controls and to compare them with patients with multiple system atrophy (MSA).

*Methods*—Quantitative anal sphincter EMG were performed on 100 normal controls and 11 patients with MSA to characterise EMG data in these two groups.

*Results*—In the normal controls, there was a trend for increased motor unit potential (MUP) amplitude, duration, area, and polyphasicity with advancing age. Patients with MSA exhibited similar MUP size and fibre density. Significant differences were found only in parameters of the recruitment pattern, which were reduced in MSA, with a diminution in the number of active MUPs during rest.

*Conclusions*—These results may reflect either decreased number of motor cells in Onuf's nucleus without significant consequential reinnervation, or upper motor neuron involvement affecting the anal sphincter in MSA. They further underline the importance of comparative data for age matched controls.

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The involvement of a specific group of cells in the anterior horn of the sacral spinal cord (known as Onuf's nucleus) in multiple system atrophy (MSA)<sup>1 2</sup> may be used as an aid for the diagnosis of the disease. Various studies have found abnormal anal sphincter EMG in patients with MSA, suggesting its utility in establishing the diagnosis in the early stages of the disease.<sup>3-8</sup> However, there is a debate about the criteria for abnormality in this particular EMG examination. Some authors proposed prolonged duration of motor unit potentials (MUPs), exceeding 10 ms, as a sign of denervation.<sup>1</sup> Others suggested the presence of fibrillation potentials as a reliable indicator for the disease.<sup>2</sup> Still others emphasised that the polyphasicity in the anal sphincter (exceeding 60%) or increased fibre density in single fibre EMG (SFEMG) studies (3.0 or above) serve as evidence for MSA.<sup>3</sup> Palace et al<sup>9</sup> described the late components of the sphincter motor units in patients with advanced MSA which might represent the late components of motor units in chronic muscle disease.

The interpretation of EMG of the external anal sphincter is difficult. Firstly, because of the lack of systematic normative EMG data taking into account the effect of aging and sex. Secondly, it is difficult to detect spontaneous activity on the background of the normal continuous activity of this muscle. Quantitative motor unit analysis has rarely been performed in patients with MSA.<sup>10</sup>

In view of the limited data on quantitative sphincter MUP analyses in normal persons and the uncertainty of criteria for abnormality in patients with MSA, we examined 100 normal subjects and analyzed the electrophysiological results. External anal sphincter EMG was performed in 11 patients with MSA and the results compared with 22 matched normal subjects from the control group.

## Methods

One hundred healthy adults (67 women and 33 men), ranging in age from 17 to 89 years (mean 62.5 (SD 15.3) years), who had no uroneurological or proctological disorders, were studied. A complete neurological examination was normal in all subjects.

During EMG examination, the subjects lay on their left side with hips and knees flexed. Their right thighs were grounded electrically. A disposable concentric needle electrode (diameter 0.46 mm) was inserted perpendicularly into the subcutaneous layer of the external anal sphincter muscle about 2 cm form the anal orifice. Deeper insertions were made at the anal orifice at an angle of 30°.11 The EMG activity was measured in four quadrants of the sphincter. By moving the position of the electrode, 20 different motor units were identified. The MUPs were collected and analyzed during relaxation and during activation by standard "multi-MUP analysis" implemented on the EMG system (Keypoint, Dantec Medical, Denmark). Standard filter settings (5 Hz-10 kHz), gain (200 µV/division) and sweep speed (10 ms/division) were used. Amplitude, duration, area, polyphasicity, number of phases, and turns were analyzed during rest with no effort. The following recruitment pattern parameters were also collected: activity (defined as percentage of time with EMG activity), envelope (defined as amplitude with removed outliers), and number of short segments (NSS).12 These data were calculated automatically during effort by the EMG system according to previously published criteria.13 14

Sphincter muscles contain a subpopulation of continuously active MUPs, which fire regularly at rest. The mean number of these MUPs/insertion was calculated. Fibre density was determined by the SFEMG technique using standard single fibre EMG needle electrodes. The examined subjects were asked to fully relax the sphincter and spontaneous activity was then evaluated.

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Table 1 Descriptive statistics of normal controls

Parameters	No	Minimum	Maximum	Mean	SD
MUPs at rest:					
Amplitude (uV)	100	276.0	1006.0	534.5	166.5
Duration (ms)	100	4.0	9.5	6.5	1.4
Area (µV/ms)	100	188.0	976.0	474.9	198.0
Poly (%)	100	0.0	45.0	22.6	11.7
No MUP	22	3.0	5.5	3.5	0.7
FD	90	1.2	2.2	1.7	0.2
Interference patterns:					
Activity (%)	100	4.0	30.0	11.7	6.3
NSS/s	100	56.0	526.0	140.5	77.2
Envelope (µV)	100	343.0	2878.0	1367.2	507.5

MUP=Muscle unit potential; Poly=polyphasic potentials; FD=fibre density; NSS=number of short segments. See text for further explanation.

Table 2 EMG parameters by sex (n=100)

Parameters	Sex	No	Mean	SD	p Value*
Amplitude (µV)	F M	67 33	526.5 550.8	151.9 194.4	0.494
Duration (ms)	F M	67 33	6.5 6.5	1.3 1.4	0.591
Area (µV/ms)	F M	67 33	474.3 476.1	200.1 196.9	0.968
Poly (%)	F M	67 33	23.3 21.4	10.8 13.4	0.458
Activity (%)	F M	66 32	11.2 12.5	6.4 6.0	0.355
NSS/s	F M	66 32	136.0 149.3	85.1 58.0	0.427
Envelope (µV)	F M	66 32	1327.2 1449.6	475.3 576.4	0.265
No MUP (at rest)	F M	10 5	3.3 4.0	0.4 1.0	0.218
FD	F M	40 29	1.6 1.7	0.2 0.2	0.468

\*By t test.

MUP=Muscle unit potential; Poly=polyphasic potentials, FD=fibre density, NSS=number of short segments.

Eleven patients with MSA (seven women and four men; mean age 66 (SD) 10.4 years) were examined. Mean disease duration was 5.5 (SD 3) years. Ten patients were diagnosed as having MSA with prominent parkinsonism (nine with possible MSA and one with probable MSA). One patient with probable MSA had a prominent cerebellar syndrome. Eight patients had autonomic disturbances (orthostatic hypotension and urinary incontinence). The clinical diagnoses were made according to the MSA standardised diagnostic criteria.15 No pathological confirmation of the diagnosis was made. The results of these 11 patients with MSA were compared with those of 22 control subjects matched by age and sex.

#### Statistical methods

Electromyographic parameters by age were analyzed using a polynomial trend curve to capture the parameter change by age. The normative presentation was followed by a case-control study. The controls were drawn from the normative cohort and matched with the study patients by age and sex. The means were then compared by t test.

## Results

The quantitative EMG data at rest and during recruitment of the normal control group are summarised in table 1. There was no significant effect of sex on any parameter (table 2). In the group of 67 women, four were nulliparous and 63 of varying parity (mean vaginal delivery 2.4 (SD 1.0)).<sup>1-5</sup> On regression analysis, parity did not significantly affect the MUP and IP parameters. The control study population was divided into age groups according to their natural distribution (table 3). The results are reported in quintiles of age. The parameter of age did not significantly affect the MUP data.

Comparing the quantitative EMG values for the sphincter of patients with MSA with the matched controls, there was no statistically significant difference between the groups in amplitudes, durations, areas, polyphasicity, and fibre densities. A significant difference was found only in the parameters of the recruitment pattern. The activities (as defined above) of all patients were significantly decreased. The envelope amplitudes were decreased, as well as NSS (fig 1). Another parameter that was significantly reduced in the patients was the number of active motor units during rest (table 4). No fibrillation potentials were detected in any patient. The late components found were calculated within the duration of the MUPs.

Despite non-significant mean differences in MUP size between the patients and matched controls while considering individual MUPs, there were three patients with MSA who showed more than 10% of MUPs with duration above 10 ms (outliers). No outliers were seen in the control group.

## Discussion

In the present study we examined the external anal sphincter muscle in normal subjects and patients with MSA. There was no difference in the various EMG parameters between sexes, nor was there any effect of parity or age on MUP/IP analysis. These results resemble those

Table 3 Normative database of sphincter EMG by age (n=100)\*

Age	Amplitude (µV)	Duration (ms)	Area (μV/ms)	Poly (%)	No of MUPs†	FD	Interference patterns		
							Activity (%)	NSS	Envelope (µV)
<49	535.0 (106.0)	6.1 (1.1)	480.0 (118.0)	19.5 (13.0)	4.4 (1.0)	1.6 (0.3)	11.2 (5.9)	123.6 (50.0)	1266.0 (348.0)
49-62	557.6 (143.8)	6.9 (1.3)	521.3 (186.0)	26.4 (10.0)	4.2 (1.0)	1.6 (0.14)	13.5 (8.1)	148.8 (92.7)	1374.9 (461.0)
63–68	545.7 (186.6)	6.3 (1.4)	481.4 (232.3)	21.5 (11.0)	3.3 (0.4)	1.6 (0.2)	11.5 (5.4)	139.8 (60.6)	1445.3 (558.5)
69–75	554.2 (184.3)	6.5 (1.5)	482.2 (208.4)	23.0 (11.1)	3.6 (0.8)	1.6 (0.19)	10.7 (6.2)	122.7 (58.7)	1364.6 (613.3)
>75	551.3 (177.1)	6.5 (1.2)	504.8 (206.2)	22.9 (12.1)	3.2 (0.2)	1.6 (0.1)	11.7 (5.8)	169.4 (109.7)	1390.0 (548.7)

\*There was no significant age effect, although a tendency towards lower amplitude, duration, area, and polyphasicity was seen in the younger age group. †Number of MUPs/insertion during rest. Values are mean (SD).

MUP=muscle unit potential; Poly=polyphasic potentials; FD=fibre density; NSS=number of short segments.

The number of patients in each group was 20.



Figure 1 Interference pattern data and number of MUPs at rest in 11 patients with MSA versus 22 peer matched controls. (A) Mean envelope with removed outliers ( $\mu$ V). (B) Percentage activity (percentage of time with EMG activity). (C) number of short segments (NSS). (D) Number of MUPs at rest. For further details see text.

of Podnar *et al.*<sup>16 17 19</sup> On the other hand, the mean values of MUP parameters (amplitude duration area) were slightly greater than the values in one of these studies, but the IP parameters were similar to their data.<sup>17</sup> The difference could emanate from a higher activation during analysis, recruiting larger units, although we sampled MUPs at a level of 3–5 MUPs activation, similarly to their technique.

Comparing patients with MSA with controls, the most obvious parameter for distinction between the two groups was the number of firing MUPs at rest and recruitment pattern data—that is, the activity, NSS, and envelope during effort. These values were found to be abnormal in every patient in respect to mean values of the control group, although it is a relatively small group of patients with MSA.

Some studies suggested that either the detection of spontaneous activity,<sup>4</sup> polyphasicity above 30%,<sup>5</sup> or MUP duration above 10 ms,<sup>3</sup> are indicators of motor neuron degeneration in Onuf's nucleus. By contrast, our results show no significant differences in these parameters between the patients with MSA and controls. Employing the technique used by us, which detects 3–5 MUPs/insertion site, rise time is not taken into account, and hence units that are not very close to the needle electrode

Table 4 EMG parameters: cases versus controls

	Cases (n=11)		Controls (n=22)			
Parameters	М	SD	M	SD	p Value	
MUPs at rest:						
Amplitude (µV)	473.5	133.9	521.4	159.8	0.324	
Duration (ms)	7.0	1.6	6.8	1.5	0.744	
Area (µV/ms)	447.3	165.7	524.9	189.6	0.239	
Poly (%)	23.1	11.8	26.1	11.1	0.484	
No MUP	2.1	0.9	3.0	0.1	0.019	
FD	1.9	0.8	1.6	0.2	0.234	
Interference patterns:						
Activity (%)	1.1	1.0	12.7	6.9	< 0.0001	
NSS/s	18.1	13.0	141.7	66.0	< 0.0001	
Envelope (µV)	574.4	341.0	1395.5	486.4	< 0.0001	

MUP=Muscle unit potential; Poly=polyphasic potentials; FD=fibre density; NSS=number of short segments.

are recorded as well. This multi-MUP analysis was found to be practical and useful for obtaining normative data, being precise and easily performed, as noted by Podnar and Vodusštek.<sup>11</sup> However, it does not explain the difference between previous results and ours. If we consider the presence of more than 10% outliers (duration exceeding 10 ms) which was not detected in any of the controls, then three of 11 patients with MSA would be considered as having some neurogenic changes. Therefore, in addition to our other findings, some "classic EMG abnormalities" were also present in certain patients.

The most important finding in our study was the poor recruitment values, which seem to be characteristic of the pathological process in patients with MSA. To the best of our knowledge, recruitment has not been systematically investigated previously. The reduced number of firing units at rest and decreased activity parameters with effort may reflect the few remaining motor cells in Onuf's nucleus. The non-detection of large MUPs and the non-increased fibre density in our patients imply that consequential reinnervation has not occurred.

Another plausible explanation for the poor recruitment with no weighty reinnervation process may be upper motor neuron involvement. In limb muscles, reduced recruitment without rapidly firing units may ensue either from voluntary inactivity or from upper motor neuron affection. Because voluntary inactivity in our patients seems highly unlikely, and the nature of the firing units during rest is not influenced by voluntary activation, upper motor neuron involvement seems reasonable. In fact, upper neuron involvement in patients with MSA has already been described. Urodynamic studies showed urethral sphincter hyperactivity<sup>16 18</sup> suggesting this mechanism. However, upper motor neuron control of the bladder and anal sphincter is not well delineated and its involvement and role in MSA is not adequately known.

In a recent study of anal sphincter EMG<sup>10</sup> comparing patients with Parkinson's disease and MSA, no significant difference in the MUP parameters and in the presence of spontaneous activity between the groups was found. Thus, MUP analysis does not seem to have an important value in distinguishing between MSA and other extrapyramidal diseases. Recruitment was not investigated in patients with Parkinson's disease and other extrapyramidal syndromes. Thus, it is not clear whether studying recruitment is helpful in detecting patients with MSA among other extrapyramidal diseases.

In conclusion, external anal sphincter EMG may detect abnormality in patients with MSA. The reduced interference activity and reduced number of MUPs at rest are the main EMG abnormalities in MSA. It is not clear whether these EMG findings are specific for MSA and their diagnostic value should be further studied, particularly in comparison with Parkinson's disease. Our normative data obtained by a rapid practical technique from a large control group may serve as a basis for other EMG studies of the anal sphincter.

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