

Lack of maintenance of gait pattern as measured by instrumental methods suggests psychogenic gait

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Summary

Fluctuation is a common feature of all psychogenic gait disorder (PGD) patterns. Whether this fluctuation involves only the degree of impairment or whether it affects the gait pattern itself remains an interesting question.

We hypothesize that, on repeated measurements, both normal and abnormal gait may present quantitative differences while maintaining their basic underlying pattern; conversely, in psychogenic gait, the basic pattern appears not to be preserved.

Using an optoelectronic system, data acquired from 19 normal subjects and 66 patients were applied to train a neural network (NN) and subsequently classify gait patterns into four different groups (normal, ataxic, spastic-paraparetic and parkinsonian). Five patients who fulfilled clinical criteria for psychogenic gait and six controls were then prospectively evaluated on two separate occasions, three months apart.

Normal controls and ataxic, parkinsonian or spastic pa-

tients were correctly identified by the NN, and categorized within the corresponding groups at baseline as well as at a three-month follow-up evaluation. NN analysis showed that after three months, no PGD patient preserved the gait pattern detected at baseline, even though this finding was not clinically apparent.

Modification of gait pattern detected by repeated kinematic measurement and NN analysis could suggest the presence of PGD, particularly in difficult-to-diagnose cases.

KEY WORDS: gait analysis, neural network, psychogenic gait

Introduction

Diagnostic agreement in psychogenic movement disorder (PMD) cases is poor, diagnosis being based on video review of phenomenology alone, and strongly relying on clinical interpretation of historical features and diagnostic workup. The limited value of currently available clinical criteria for PMD, and the difficulty characterizing uncertain cases suggests that there is a need for new diagnostic criteria (Morgante et al., 2012), or even perhaps for the development of instrumental evaluations with greater sensitivity and specificity.

Abnormal gait can be an isolated phenomenon in patients with PMDs, or part of a more complex psychogenic condition (Baik and Lang, 2007). Psychogenic gait disorders (PGDs), either isolated or occurring as part of more complex generalized psychogenic syndromes, are not uncommon, accounting for 1.5% to 26% of patients admitted to a neurology unit (Bhatia, 2001; Lempert et al., 1991). Some cases without bizarre features simulating organic disorders remain obscure despite careful workup, and are only clarified during follow up.

Different classifications of PGD have been published, some of which have established PGD categories including ataxic, hemiparetic, spastic, dystonic, truncal myoclonic, stiff-legged, tabetic and camptocormic, with most patients falling into the ataxic, hemiparetic and spastic groups (Keane, 1989; Hayes et al., 1999).

Although the psychogenic nature of gait is in general quickly apparent to an experienced observer, these are often difficult patients, and optimal diagnosis and management require extra time. Imaging studies are easily justified and sometimes informative even when suspicion of an organic disease is low. Diagnosis depends mainly on observation of bizarre motor behavior, discrepancy between obvious dysfunction and normal diagnostic evaluation, and evidence of psychiatric abnormalities. Gait fluctuation is a common feature of all PGD patterns, but whether this fluctuation concerns only the degree of impairment, or whether it affects the characteristics or pattern of gait is an issue that has yet to be addressed applying objective analysis methodology.

In the present study we trained an automated neural network (NN) analysis system to classify gait patterns into four different groups by introducing data from pure clinical cases with unequivocal patterns. In addition to normal gait, we used the most common types of abnormal gait patterns found in neurodegenerative disorders, namely parkinsonian, ataxic and spastic.

We hypothesize that on repeated measurements both normal and abnormal gait may express quantitative differences between measurements while preserving the same pattern, whereas in cases with a psychogenic origin, patients may be unable to maintain the same pattern.

Materials and methods

The protocol was approved by the local IRB and all patients signed an informed consent form prior to study participation.

System hardware for kinematic analysis and walking paradigm

SYSTEM HARDWARE

Controls and patients were supplied with comfortable clothing, and asked to perform tests barefooted. A six-camera optoelectronic ELITE system (BTS Bioengineering, Milan, Italy) was used to determine time and space coordinates of seventeen separate wireless markers. Recordings were conducted in a quiet, artificially illuminated room, in the presence of two investigators and a relative of the patient. Evaluations were conducted on a non-patterned solid light blue treadmill. The room was empty except for the treadmill in order to avoid potential visual cues.

WALKING PARADIGM

Subjects performed five trials, walking ten meters along a straight line. Patients and controls were allowed three to five minutes' rest between tests; patients were instructed to walk at a comfortable, self-paced speed, similar to one they might use at home during unhurried everyday activities. Cycles corresponding to the first and last step were excluded from the analysis.

Variables included in the neural network

All data obtained were processed using software created specifically for this study by calculating the spatiotemporal position of the corporeal center of mass (COM: geometric point corresponding to ideal location for body weight application) as well as the spatiotemporal position of the center of pressure (COP: point of dynamic support). By positioning the markers and applying previously calculated variables, absolute and normalized values were determined according to subject height and weight for an additional 42 gait kinematic variables.

Development and characterization of a neural network

To classify gait disorders on the basis of kinematic parameters and using a NN, it is first necessary to deter-

mine relevant kinematic variables critical to each of the conditions to be tested, then normalize value differences and variations depending on measurements, subject gender, height or weight, and finally analyze magnitude and deviations of critical variables between cases studied and normal controls, as well as typical pathologies.

In order to determine whether the variables selected were truly meaningful for characterizing kinematics of gait patterns, a cluster analysis was performed prior to the implementation of the network. Cluster analysis is a multivariate statistical analysis technique applied to divide a set of objects into groups (clusters) in which the profiles of objects in the same group are very similar (property of internal cohesion) and very different from those from different clusters (external isolation). Essentially, cluster analysis considers each subject as a point, in a space of n dimensions; the coordinates of the point in that n -dimensional space are the values of the variables chosen. For this study, an algorithm grouping subjects according to the n -dimensional Euclidean distance between them was implemented using an ad hoc computer program. We were then able to measure the 42 variables mentioned in the previous section, excluding non-statistically significant differences between gait patterns. Nevertheless, the number of possible combinations was still in the order of 200 million. Therefore we calculated the different possible combinations generating clusters, and assigned a score for each combination of variables according to the clinical classification groups, which resulted in a promising set of 16 variables (Table I).

An artificial NN is a computer algorithm that allows the classification of elements in different groups from a series of input variables. This consists of an input layer (the number of nodes in this layer is equal to the number of input variables), an output layer and an undetermined number of intermediate layers (or hidden layers). The number of nodes in the output layer is chosen according to the responses expected of the network, which in this case was four, equal to the number of gait patterns selected. Values of variables in the input layer were subjected to a threshold function, a nonlinear sigmoid type function ($f(x) = 1 / (1 + \exp(-x))$), which determined the intensity needed to transfer to the next layer. Values were transferred entirely to each of the nodes of the next layer, where weighted summation of these values was performed, i.e., they were subjected to a linear hyperplane-type function.

The network used was perceptron, with one hidden layer and supervised learning with a back propagation algorithm. Network training was performed using a group of subjects whose gait pattern had been established clinically. Initially the network was programmed with random weights per node. Introducing variable values from these subjects in the input layer, we assumed the values of all four nodes in the output layer were 1 for the node of the subject's gait pattern, and 0 for the others. Normally this condition is not met, causing the algorithm to transfer the values obtained in the output to the input, in order to adjust appropriately for node weight (feedback) based on a calculated error. If the variables chosen were correct, then for a given number of feedback loop outputs (in the order of tens of thousands), all cases would be sufficiently close to expected results. When this occurred the network was said to be trained, with values of weights considered the result of such training,

a result later verified in an independent group of subjects with known gait patterns.

Gait patterns included for neural network training

In order to feed the NN we assessed gait in 19 normal subjects (9 males and 10 females; mean age 54.2 years), in 24 presenting spastic gait (11 males and 13 females; mean age 56.7 years; 10 due to stroke and 14 to cervical myelopathy), in 10 with pure ataxic gait (6 males and 4 females; mean age 61.3 years; with acquired progressive ataxia), and in 32 subjects with parkinsonian gait due to Parkinson’s disease (all with stable response to L-dopa without dyskinesias; 18 males and 14 females; mean age 60.4 years). All patients were under prospective follow-up at the neurology outpatient clinic. Two neurologists separately confirmed specific gait patterns in each subject, both clinically, as well as after ancillary workup including: brain and spine MRI scans, motor and somatosensory evoked potentials, and peripheral nerve conduction velocity. Complete orthopedic evaluation ruled out hip, knee or foot pathology. Patients with a history of hip or knee replacement, or MMSE scores below 26 were excluded.

Application of the neural network for PGD diagnosis

PATIENTS

Patients were prospectively selected from our movement disorders clinic during 2010. Five patients fulfilled clinical criteria for psychogenic gait and underwent ex-

tensive workup to rule out other underlying physical conditions. The diagnostic classification of Fahn and Williams (1988) was followed, requiring confirmation of abrupt onset, inconsistent, incongruent gait pattern, and prior history of minor injury.

Psychogenic features were assessed in these patients using techniques such as suggestion, repeated attempts at triggering abnormal movement, distraction, and administration of placebo in one case. All patients presented a history of prior psychiatric illness and fluctuation of impairment. None had additional psychogenic neurological symptoms other than psychogenic gait. Although consensus on final diagnosis was always reached, it took several visits to confirm. Table II (over) shows demographics and clinical characteristics of all five patients.

A group of six controls, not belonging to the group used to feed the NN, were prospectively selected from the movement disorders clinic: one male patient 60 years old suffering from pure ataxic gait, one male 62 years old with Parkinson’s disease, one female 67 years old with spastic gait, one normal control 65 years old, and one female 63 years old with myopathy. The myopathic patient was also analyzed to show that NN can exclude other gait patterns (data not shown).

METHODS

Patients and controls were evaluated prospectively using the system hardware and walking paradigm previously described, on two separate occasions three months apart, in accordance with the usual evaluation period at our clinic. Data obtained were then incorporat-

Table I - Values of the variables used in training the network to classify the four gait patterns analyzed

	Gait Pattern (mean ± SD)			
	Normal	Spastic	Ataxic	Parkinsonian
Stance time (**)	.68 ± .02	.77 ± .05	.77 ± .09	.72 ± .04
Swing time (**)	.91 ± .05	.74 ± .10	.74 ± .17	.83 ± .09
Double stance time (**)	.23 ± .06	.43 ± .12	.44 ± .21	.32 ± .11
Cycle length (*)	.81 ± .08	.56 ± .13	.57 ± .20	.64 ± .15
Average swing speed (*)	.78 ± .09	.54 ± .12	.61 ± .18	.63 ± .14
Maximum swing speed (*)	.79 ± .09	.56 ± .11	.64 ± .17	.64 ± .14
Average gait speed (*)	.72 ± .11	.41 ± .14	.48 ± .21	.54 ± .16
Step width (*)	.43 ± .11	.46 ± .10	.71 ± .20	.47 ± .14
Swing time (*)	.77 ± .03	.77 ± .08	.69 ± .09	.75 ± .06
Average longitudinal position of CM (*)	.41 ± .16	.43 ± .23	.27 ± .24	.31 ± .23
Average vertical position of CM (*)	.75 ± .10	.77 ± .11	.84 ± .11	.80 ± .12
Vertical excursion of CM (*)	.54 ± .10	.43 ± .11	.47 ± .12	.48 ± .17
Maximum position of CM (*)	.54 ± .15	.35 ± .09	.36 ± .16	.38 ± .15
Lateral excursion of CM (*)	.36 ± .09	.37 ± .09	.58 ± .17	.40 ± .08
Foot rotation/ Knee flex.-ext.	.25 ± .13	.24 ± .15	.45 ± .28	.19 ± .14
Knee flex.-ext./ Pelvis rotation	.48 ± .13	.45 ± .10	.41 ± .13	.57 ± .16

Values of the variables used in training the network and their average values and standard deviation in the four gait patterns analyzed. The average values of the variables in each column represent the position of the centroid of each gait pattern in the 16 D space. Abbreviations and symbols: (*)=as a percentage of the gait cycle; (**)=normalized according to height and weight; (***)=calculated relative to the center of pressure; CM=center of mass.

Table II - Demographic and clinical characteristics of patients with psychogenic gait disorders

	Patient 1	Patient 2	Patient 3	Patient 4	Patient 5
Age (years)	52	62	45	62	63
Sex	F	M	F	M	F
Clinical pattern at basal evaluation	ataxic	paraparetic	parkinsonian	ataxic	ataxic
Clinical pattern at 3-mth evaluation	ataxic	paraparetic	parkinsonian	ataxic	ataxic
Neurologist's impression of change	no change	no change	slight change	no change	slight change
Fluctuation of impairment	yes	yes	yes	yes	yes
Duration (months)	12	3	36	12	40
Type of onset	insidious	abrupt	abrupt	abrupt	abrupt
Psychiatric illness	anxiety	anxiety	depression	depression	depression
Spontaneous remissions	no	yes	no	no	no
Paroxysmal symptoms	no	no	no	yes	yes
Distractibility	no	yes	no	yes	no

Abbreviations: Values of the variables used in training the network and their average values and standard deviation in the four gait patterns analyzed. The average values of the variables in each column represent the position of the centroid of each gait pattern in the 16 D space. Abbreviations and symbols: (*)=as a percentage of the gait cycle; (**)=normalized according to height and weight; (***)=calculated relative to the center of pressure; CM=center of mass.

ed into the NN. Clinical evaluation of patients and of the control group was performed at baseline and after three months' follow-up by the same two neurologists. Gait pattern was classified during each visit and rated at follow-up as unchanged, slightly changed or presenting a different pattern altogether from baseline. Neurologists were blind to NN classification on both occasions; however, at the follow-up visit they were not blind to the initial clinical diagnosis.

Results

Neural network

The NN developed categorized four quite different gait patterns: normal, ataxic, parkinsonian and spastic-paraparetic, based on the combination of 16 variables. Each group presented a centroid (a point calculated on the basis of the average of the 16 coordinates) and a radius (equivalent to twice the average distance from the centroid). The groups were found to be distributed without overlapping in a 16-dimension space (Fig. 1). The values for each variable in the different groups are detailed in Table I.

Controls

The normal control, as well as the controls with ataxic, parkinsonian or spastic gait was adequately identified by the NN at baseline evaluation. At the three-month follow-up visit, the normal control as well the patients with defined gait patterns remained in the same groups. The myopathic patient did not fit any of the NN-defined gait patterns, as expected (Fig. 2).

Psychogenic patients

None of the PGD patients showed changes in gait pattern on clinical evaluations performed after an interval of

three months by the same two neurologists; however, kinematic evaluation and NN analysis indicated the opposite, namely that no PGD patient, at the three month follow-up visit, was able to maintain the same gait pattern detected during the first evaluation. Patient 1 was classified by the NN as ataxic during the baseline evaluation but as paraparetic at three months; patient 2 was classified by the NN as paraparetic at baseline, but as ataxic after three months; patient 3 was classified by the NN as parkinsonian initially but as ataxic after three months; patient 4 was first classified by the NN as ataxic but after three months as parkinsonian, while patient 5 was initially classified by the NN as ataxic, but as paraparetic three months later.

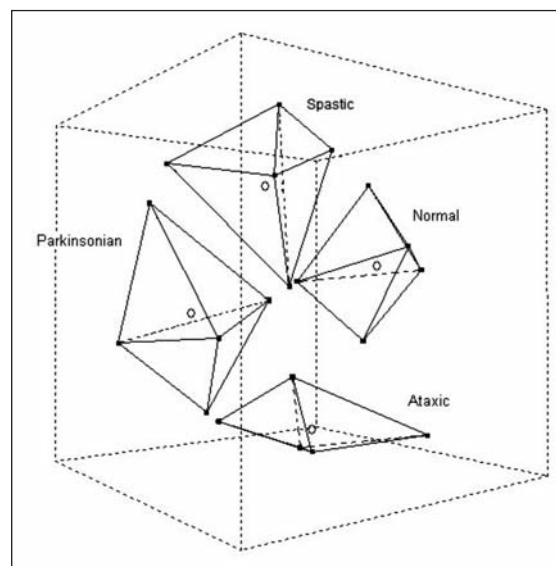


Figure 1 - Schematic representation of the clusters in a 3D graphic (the true clusters are distributed in a 16-dimension space).

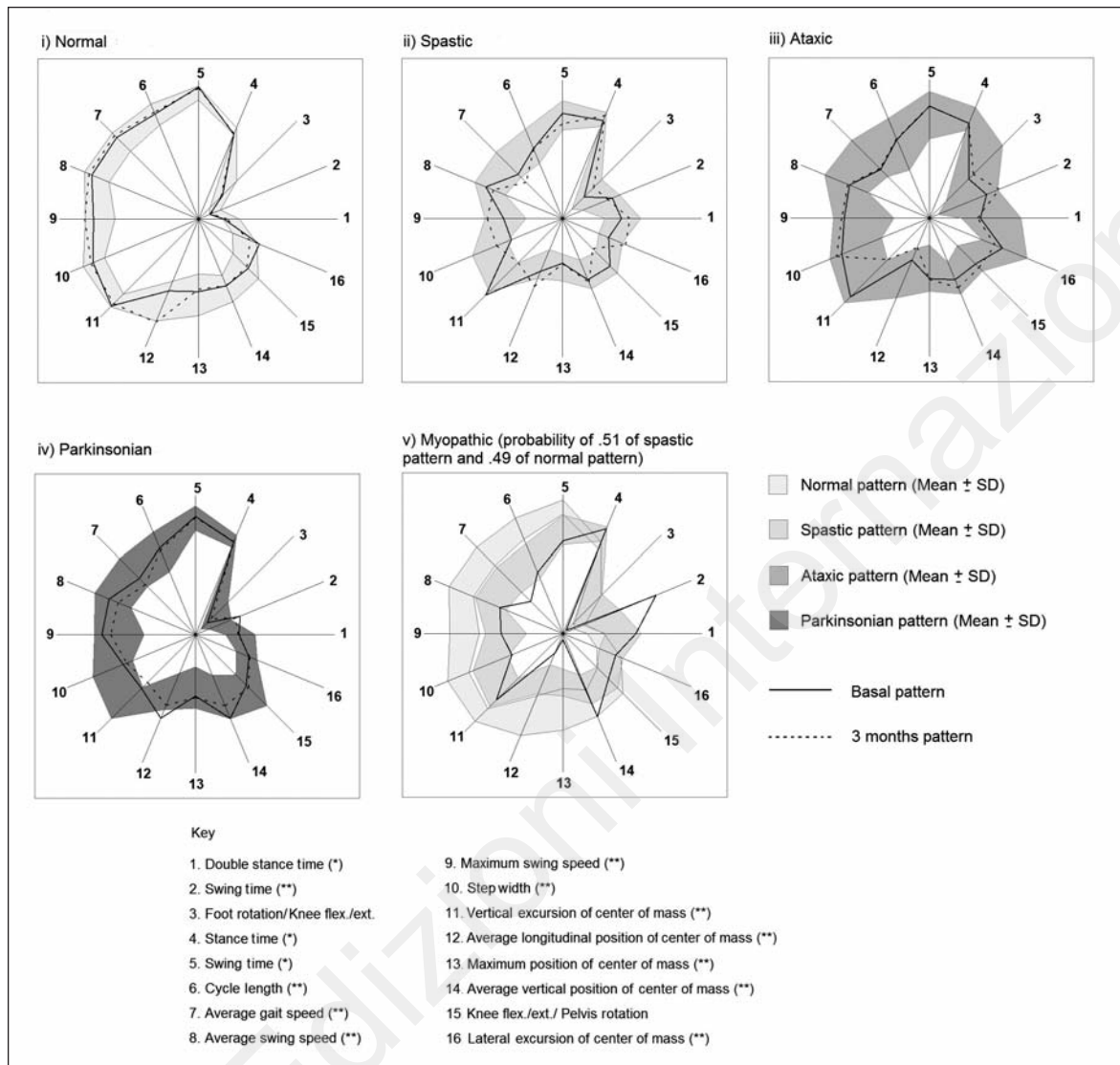


Figure 2. Normal control and patients with defined gait patterns measured three months apart. The radial lines represent the 16 variables. The gray areas represent the different groups showing the average value and standard deviation recorded for each variable.

Figure 3 (over) depicts changes in gait patterns between first and second evaluation, while clinical and demographic characteristics are given in Table II. The likelihood of belonging to a given group, calculated by the network for normal controls, patients with organic disorders, and psychogenic patients, is shown in Table III (over).

Discussion

A diagnosis of PMD should not be an exclusion diagnosis. Careful clinical assessment is critical, and imaging or electrophysiology studies may provide important insights and diagnostic confirmation, even though some cases remain challenging, with current assessments failing to provide the necessary clarification (Gupta and Lang, 2009). At present, there is only one scale available to describe, assess and quantify PMDs

(Fahn and Williams, 1988), and no tool exists for diagnosing PGD.

We found that unlike patients with gait disturbance of organic origin, patients with PGD failed to maintain the same gait pattern after repeated evaluations, which could represent an important finding for PGD diagnosis. Different types of bizarre psychogenic gait patterns have been described including buckling of the knee, astasia-abasia, waddling, tightrope gait, excessive retropulsion, walking on ice and penguin gait (Lempert et al., 1991; Keane, 1989). In general, careful patient examination by trained observers readily clarifies cases; however, the fact remains that even for the expert, a substantial proportion of PGD patterns may mimic organic disorders and remain a diagnostic challenge. Keane (1989) found that 24 of 60 cases with PGD presented ataxic, paraparetic and trembling slowness patterns, mimicking gait disturbances associated with the most common neurodegenerative disorders. It is on this last group of pa-

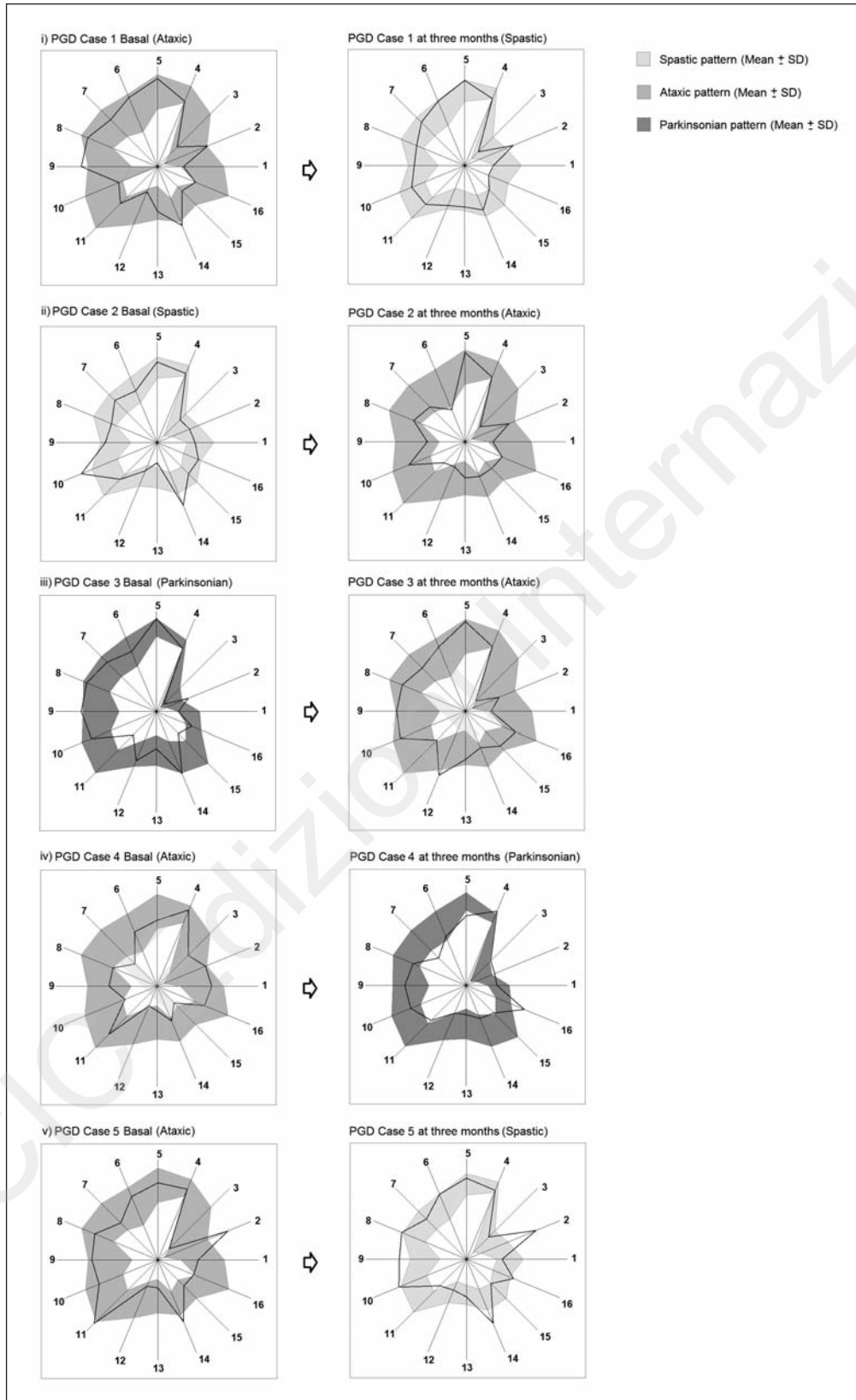


Figure 3. Psychogenic patients measured three months apart. The radial lines represent the 16 variables. The gray areas depict the different groups showing the average value and standard deviation recorded for each variable. It is possible to observe the change in gait pattern on the second measurement.

Table III - Probabilities of belonging to different gait patterns

Patient	Pattern							
	Basal			3 months				
	Normal	Spastic	Ataxic	Park.	Normal	Spastic	Ataxic	Park.
Normal control	0.93	0.00	0.06	0.00	1.00	0.00	0.00	0.00
Spastic	0.00	0.97	0.00	0.02	0.00	0.99	0.00	0.00
Ataxic	0.00	0.00	0.99	0.00	0.02	0.08	0.89	0.00
Parkinsonian	0.00	0.00	0.00	0.99	0.00	0.00	0.00	0.99
Myopathic	0.00	0.48	0.51	0.00	–	–	–	–
PGD Case 1	0.00	0.00	0.91	0.08	0.09	0.72	0.17	0.00
PGD Case 2	0.00	1.00	0.00	0.00	0.00	0.00	0.99	0.00
PGD Case 3	0.00	0.00	0.00	0.99	0.00	0.00	0.99	0.00
PGD Case 4	0.04	0.00	0.85	0.09	0.00	0.00	0.16	0.83
PGD Case 5	0.00	0.00	0.96	0.04	0.00	0.99	0.00	0.00

Probabilities of belonging to different gait patterns given by the neural network to the normal control, the patients with different clinical gait patterns and the psychogenic patients. Note that for one subject with a myopathic pattern the network failed to provide a conclusive answer.

tients, which constitutes about 10% of all PGD cases (Lempert et al., 1991) that we focused our analysis.

Fluctuation of gait may occur in neurological disorders, as in the case of Parkinson's disease, normal pressure hydrocephalus, myasthenia gravis, episodic ataxias or ion channel myopathies, among others. However, since modification of gait implies fluctuation of the degree of impairment, not of the pattern of gait, fluctuations are difficult to capture during short video recordings or during consultations as they may not be apparent from simple clinical observation, and can only be detected using an instrumental intervention with greater diagnostic sensitivity, such as the one presented here (Morgante et al., 2012).

Many authors have attempted to identify walking patterns using electromyography or kinematic analysis (Mulroy et al., 2003; O'Byrne et al., 1998; Kinsella and Moran, 2008; Wong et al., 2004; Olney and Richards, 1996). Neural network analysis (NNA) is a tool widely used in biomedical research, including neuroscience, but few papers have shown it to be useful in distinguishing gait patterns (Holzreiter and Köhle, 1993; Lafuente et al., 1998), and so far its utility in psychogenic gait diagnosis has not been tested.

Neural network analysis uses artificial NNs, inspired by natural NNs (Rosenblatt, 1958; Widrow and Lehr, 1990), offering an alternative to conventional signal and data processing algorithms based on linear models. Since NNA manages complex non-linear association data, it functions especially well in cases such as those investigated in this study. Non-linear mapping of complex data is often required in the field of neurology, and NNA provides an excellent framework for this type of analysis (Freeman and Skapura, 1991).

On this occasion it was applied to movement pattern recognition and gait analysis, and provided a method for identifying patients at risk of, or presenting non-functional gait disorders (Begg et al., 2006).

Neural networks can be trained to detect all existing gait patterns, however because our interest is in movement disorders in particular, we decided to incorporate into

the network only those abnormal gait patterns (such as paraparetic, ataxic and parkinsonian gait) that are observed in the majority of degenerative central nervous system disorders, and excluded those linked to orthopedic, peripheral nerve or muscle pathology. Furthermore, the exclusion of other difficult overlaps, such as patients who are both dystonic-myoclonic and stiff-legged and who should be taken into account, could be considered a limitation of the current study.

The decision to use a perceptron (supervised training) NNA system for this purpose creates the possibility that the universe analyzed is not entirely occupied by the patterns described, and that some patients might not qualify and therefore go undetected. Bizarre PGD would certainly fall into this category of undefined patterns. Therefore, the idea behind the present study was not to study easy-to-diagnose bizarre gait patterns, but cases with hard-to-define clinical characteristics where the algorithm might prove useful. In conclusion, in certain difficult-to-diagnose PGD cases, absence of specific gait pattern preservation on repeated evaluations conducted using more sensitive instrumental methods may help identify patients not detected through clinical observation.

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