Original paper

Speech disfluencies in Parkinson's disease

Zaburzenia płynności mówienia w chorobie Parkinsona

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Słowa kluczowe: choroba Parkinsona, niepłynność mówienia, bariera komunikacyjna.

Abstract

Introduction: Even though speech disfluency is listed in the clinical description of Parkinson's disease (PD), its nature, intensity, symptomatology, and the effect on verbal communication have not hitherto been defined.
Aim of the research: The research paper presents the results of studies aimed at the description of speech disfluencies in PD and the influence on verbal communication.
Material and methods: The tests involved 10 patients from 54 to 72 years of age with documented PD, responsive to L-dopa preparations. The principal method of the study was based on the linguistic analysis of the utterances produced by the people with PD.
Results: The intensity of the speech disfluency observed in the utterances of persons with PD ranged from 6.6% to 23.0%, so it was significantly higher than that which is assumed as acceptable (3–5%); the speaking rate of the examined persons ranged from 0.7 syllabes (syl.)/s to 4.0 syl./s, and only 2 examined persons spoke with a rate considered to be correct (4–6 syl./s). This demonstrates that speech disfluency is a communication barrier in PD.
Conclusions: The absence of differentiation in the speech disfluency (SD) severity between different types of verbal utterances (difference not statistically significant) and a specified hierarchy of SD symptoms indicate that the speech disfluency in PD has an essentially organic background and is generated by cognitive, linguistic, and motor deficits resulting from the damage to the central nervous system. This is also confirmed by the established hierarchy of utterances with respect to the SD intensity, not excluding the simultaneous participation of the emotional factor.

Streszczenie

Wprowadzenie: Mimo że niepłynność mówienia jest wymieniana w opisie klinicznym choroby Parkinsona, dotychczas nie ustalono, jaki jest jej charakter, nasilenie, symptomatologia oraz wpływ na przebieg komunikacji słownej. Wydaje się, że taka charakterystyka jest kluczowa dla optymalnej pracy terapeutycznej.
Cel pracy: Przedstawienie wyników badań, których celem był opis niepłynności mówienia w chorobie Parkinsona.
Materiał i metody: W badaniach wzięło udział 10 pacjentów w wieku 54–72 lat z udokumentowaną chorobą Parkinsona, dobrze reagującą na preparaty L-dopy. Badania prowadzono w trakcie fazy ON. Zasadniczą metodą badań była analiza lingwistyczna wypowiedzi pochodzących od osób z chorobą Parkinsona (łącznie 15 000 sylab).
Wyniki: Natężenie niepłynności mówienia odnotowane w wypowiedziach osób z chorobą Parkinsona zawarło się w przedziale 6,6–23,0%, było więc zdecydowanie większe od przyjętego za dopuszczalne (3–5%). Szybkość mówienia badanych mieściła się w przedziale 0,7–4,0 sylab/s. Tylko 2 badanych mówiło z prędkością uznawaną za prawidłową (4–6 sylab/s).
Wnioski: Rezultaty badania pokazują, że niepłynność mówienia w chorobie Parkinsona stanowi barierę komunikacyjną. Brak zróżnicowania nasilenia niepłynności mówienia w różnych rodzajach wypowiedzi słownych (różnica statystycznie nieistotna) oraz ustalona hierarchia objawów niepłynności mówienia wskazują, że niepłynność mówienia w chorobie Parkinsona ma zasadniczo podłoże organiczne, generują ją deficyty poznawcze, językowe i motoryczne wynikające z uszkodzenia ośrodkowego układu nerwowego. Potwierdza to także ustalona hierarchia rodzajów wypowiedzi ze względu na natężenie niepłynności mówienia, nie wykluczając udziału czynnika emocjonalnego.

Introduction

The universal pattern of the communication process seems simple: a person (transmitter) delivers information (message) in a particular manner (communication channel) to another person (the recipient) [1–3].

The process, outlined so clearly, would be simply perfect were it not for the noise that often hinders its course. The obstacles encountered in the process of communication are defined as noise [1, 2]. Many of them emerge independently from the transmitter. Nevertheless, there are obstacles that depend entirely on the communication process participants; they definitely affect its ultimate outcome, i.e. the mutual understanding, sometimes making it difficult, and sometimes simply impossible. Speech disorders are regarded as communication noise of that type, with speech disfluency being a significant element of the group [4].

Speech is a higher neural activity based on the interaction of complex neural networks, requiring time-ordered simultaneous and sequential activation of the cerebral cortex, subcortical structures, and peripheral perceptual-executive apparatus. The fulfilment of these conditions ensures a proper course of the process of formulating and delivering statements, and as a result – a fluent verbal utterance.

The structural basis for the linguistic processes is provided by the secondary and tertiary areas of the association cortex in the dominant (usually left) hemisphere of the brain, surrounding the Sylvian fissure: the rear part of the third frontal gyrus, supplementary motor areas, the associative cortex of the temporal lobe, especially in its central and rear parts at the border of the parietal and occipital lobes, as well as the occipital cortex, located toward the front of the calcarine sulcus. Modern neuroimaging techniques have further demonstrated that during linguistic activities that mirror cortical areas of the non-dominant hemisphere are co-activated simultaneously; activity of subcortical nuclei, the thalamus, the cerebellum, and the emotional memory system has been reported as well. Thus, the participation of the non-dominant hemisphere in the process of speech has been confirmed. It is connected primarily with prosodic-emotional features (intonation and speech melody direction, voice tone and modulation, distribution of force and accents) as well as the role of subcortical structures and the cerebellum in the creation of linguistic behaviour [5].

Under physiological conditions there is a fluent transition from linguistic functions to speech performance activities with the cortex (cortical representation for the phonatory-articulatory and respiratory muscles) and corticonuclear tract fibres participating in those activities at the central level, and motor nuclei neurons of the cranial nerves in the brainstem (along with their axons forming peripheral nerves), the neuromuscular synapses, and articulatory and respiratory muscles being involved at the peripheral level. The subcortical parts of the extrapyramidal system and the cerebellum perform basically the function of modelling the course of the speech motor act; however, they also affect the formation of language patterns.

Both at the level of language and the level of performance the speech is affected by the emotional memory system (limbic system), which is manifested primarily in the prosody of speech, as well as in other non-verbal behaviours (facial expressions, gestures, body posture) [6].

To sum up, speech fluency consists of a fluent transition from one element of utterance to another, maintained at an appropriate pace and rhythm, and resulting from the synchronisation of three levels of speech production organisation: content, linguistic form, and phonic substance. Its disturbance leads to impaired speech fluency. Z. Tarkowski (2002) distinguished the following types of disfluency:

– semantic disfluency, characterised by difficulties in a fluent transition from one piece of information to another (with symptoms such as pauses, repetitions, and embolophrasia, the main function of which is giving the speaker the time to think or recall information);
– syntactic disfluency, resulting from the difficulty in a fluent transition from one syntactic structure to another (characterised by revisions and repetition of conjunctions);
– articulatory disfluency generated by problems with a free transition from one articulation to another (characterised by repetitions, prolongations, blockages, dysrhythmias) [7].

Human speech has probably been affected by disfluency from its beginning. Nevertheless, it is a scarcely explored phenomenon. Whereas disfluency is most commonly dealt with in studies focussing on stuttering and cluttering, it is also present in the utterances of the neurologically ill, disturbing verbal communication to a large extent. If its intensity is high, it can even lead to the communication with the person with disease being broken.

Speech disfluency (SD) is listed in the clinical description of Parkinson's disease (PD). Most authors, however (J.D. Henry, J.R. Crawford, A. Goberman, M. Blomgren, S. Budrewicz, K. Słotwiński, R. Podemski), treat it merely as a symptom; hence its nature, intensity, and symptomatology have not hitherto been defined [8–10]. Still, such a characterisation appears to be essential for optimal therapeutic work.

Aim of the research

The aim of the study was to describe speech disfluency in the utterances of people with PD, in particular

its severity, symptomatology, pathomechanism, and the speaking rate.

Material and methods

The tests involved 10 patients (9 men and 1 woman) aged from 54 to 72 years, with documented PD, responsive to L-dopa preparations. All the examined patients had undergone neuroimaging tests (computed tomography – CT or magnetic resonance imaging – MRI) conducted during the ON state.

The principal method of the research was the linguistic analysis of the utterances produced by persons with PD. The corpus of verbal expression was collected based on the performance of classic tests such as: *repeating text*, *reading text*, *picture story telling*, *guided conversation*, *and free conversation*. The linguistic analysis covered the text of the first three hundred syllables of each test (a total of 1500 syllables (syl.) for each examined person).

The speech fluency intensity was expressed by the ratio of the number of disfluent syllables to the total number of syllables, multiplied by 100.

The speech rate (SR) for each examined person was evaluated based on the ratio of the total number of syllables to the time of their utterance.

Standardisation of results

1. Each empirical result was converted into the T1 scale, according to the formula:
   $y = 10z + 50$,
   where:
   z – standardized result, expressed with the formula:

$$Zi = \frac{x_i - x_{śr}}{S} \quad [11]$$

   with:
   x_i – i^{th} empirical result,
   $x_{śr}$ – mean empirical result, whereas:

$$s = \sqrt{\frac{1}{n}\sum(x_i - x_{sr})^2}$$

   – standard deviation.
2. The results from point 1 were added up for each examined patient.
3. Relational indicator (W_r) was calculated:

$$W_r = \frac{y_{i,j}}{\Sigma y_i}$$ for each i^{th} and j^{th} factor.

4. The mean W_r result was calculated for the appropriate parameter.

The general result for the speech discontinuity was established as follows:

a. Average empirical results were summed.
b. The results obtained in step 1 were converted into the T1 scale in accordance with the procedure in point a.
c. Results from point b were summed.
d. The relational W_r was calculated dividing the results from point b by the results from point c.

The hierarchy of the types of utterances with regard to the intensity of SD and a hierarchy of the intensification of the SD symptoms were established as in step 4.

In order to examine the significance between the values of speech disfluency intensity in particular types of utterance, the structure indicator was applied, expressed by the formula:

$$t = \frac{\left| \frac{m_1}{n_1} - \frac{m_2}{n_2} \right|}{\sqrt{\frac{p \times q}{n}}}$$

where:
m_1, m_2 – numbers defined as m_1 and m_2, respectively,
n_1, n_2 – numbers defined as n_1 and n_2, respectively

$$\rho = \frac{m_1 + m_2}{n_1 + n_2}$$ – mean structure indicator

$$q = 1 - p$$

$$n = \frac{n_1 x\, n_2}{n_1 + n_2}$$ – pseudorandomness

The null hypothesis was rejected if, at the confidence level of $\alpha = 0.05$, the calculated t-value was less than the critical value read from the tables. In this case, the value was 1.96.

In order to establish the statistical relation between the rate of speech and the values of the speech disfluency intensity, the correlation coefficient was calculated, expressed with the formula:

$$r = \frac{\sum_{i=1}^{n} x_i \cdot y_i}{\sqrt{\sum_{i=1}^{n} x_1^2 \cdot \sum_{i=1}^{n} y_i^2}}$$

where:
x_i, y_i – deviations from relevant means.

The null hypothesis for the correlation coefficient r was verified, applying the formula:

$$t = r \cdot \sqrt{\frac{N-2}{1-r^2}}$$

where:
N – the number of pairs of the relevant results.

The relation was statistically significant if, at the level of statistical significance of $\alpha = 0.05$, the value of the correlation coefficient was higher than or equal to 0.6.

Results

Intensity of speech disfluency in the utterances of persons with Parkinson's disease

The lowest intensity – up to 10% – was observed in patients with the shortest duration of disease (up to 5 years), and the highest (exceeding 20%) was characteristic of the speech of those patients who had had the disease diagnosed ten years before, with the neuroimaging tests demonstrating cortical or corticobasal atrophy (Table 1).

Speech disfluency intensity in different types of verbal utterances

The lowest intensity of SD – from 2.0% to 9.0% – was observed during the test: *repetition of text*, whereas during the test: *reading of text* it ranged from 5.0% to 18.0%. During *telling a picture story* the intensity of SD ranged from 7.0% to 31.0%, during the test *guided conversation* – from 9.0% to 34.0%, and during the test *free conversation* – from 10.0% to 32.0% (Table 2).

Although the conducted tests varied with regard to SD intensity, in all the cases the difference between the values of SD intensity in particular types of utterance did not prove to be statistically significant. The highest value – 0.3769 – was observed for the difference in SD intensity between *telling a story* and *free conversation* (Table 3).

Table 1. Speech disfluency intensity in the examined patients' utterances

Examined patient	Mean percentage of speech disfluency	Patient's age	Time from the moment of diagnosis [years]	Topography of the central nervous system damage*	Unified Parkinson's Disease Rating Scale
1	19.8	66	9	Brain tissue without lesions. Ventricular system unexpanded	32
2	18.0	72	10	Visible cortical atrophy within the frontal lobe and temporal lobe on the left. The asymmetry of lateral ventricles wider on the right	45
3	10.0	54	4	Unexpanded symmetrical ventricular system, without any signs of proliferative process	19
4	13.0	70	7	Brain tissue without lesions, isolated calcifications within vascular plexuses of both lateral ventricles	41
5	6.6	61	3	Vascular lesions not manifested, symmetric ventricular system, after the administration of contrast no foci of pathological enhancement were observed	14
6	8.2	67	5	Symmetrical expansion of ventricles	26
7	19.6	68	8	Asymmetry of ventricles – wider on the left, features of trace cortical atrophy	37
8	21.0	71	13	Cortical atrophy within both frontal lobes	49
9	11.8	59	6	Symmetrical, unexpanded ventricular system without lesions within the brain tissue, calcification within the falx cerebri	36
10	23.0	72	12	Generalised cortico-subcortical atrophy	43

**Based on the medical documentation made available by the patients.*

Table 2. Speech disfluency intensity in different types of speech utterance (expressed as percentage)

Examined patient	Repetition	Reading	Telling a story	Guided conversation	Free conversation
1	4	13	28	22	32
2	8	15	20	22	25
3	2	5	13	15	15
4	4	10	19	14	18
5	3	4	7	9	10
6	5	13	28	23	29
7	4	6	11	10	10
8	7	18	31	25	28
9	3	7	16	16	17
10	9	13	29	34	30
Mean value	4.9	10.4	20.2	19	21.4
Standard deviation	2.330951	4.718757	8.469553	7.644897	8.382521

Table 3. Statistical difference between the values of SD intensity in different types of utterance

	Repetition	Reading	Telling a story	Guided conversation	Free conversation
Repetition	–	0.00281	0.000114	9.29E-05	5.68445E-05
Reading	0.00281	–	0.003209	0.004248	0.001377
Telling a story			–	0.371662	0.376906
Guided conversation				–	0.256039

Table 4. Hierarchical ranking of utterances with regard to SD intensity

Repetition	Reading	Telling a story	Guided conversation	Free conversation
0.200807	0.199797	0.199712	0.199957	0.199726
1	3	5	2	4

The standardisation of results permitted also a hierarchical ranking of different types of utterance with regard to SD intensity. *Text repetition* was ranked in the first position, with *guided conversation*, *reading*, *free conversation*, and *telling a story* in further positions, respectively (Table 4).

Symptomatology

In total, 2592 manifestations of speech disfluency were observed in the utterances of the examined patients. One syllable uttered non-fluently corresponded to 1.13 manifestations of SD. Six patients showed all the SD symptoms, the speech of 2 patients was not affected by *blockage of sounds*, and the speech of 2 other patients showed no signs of *blockage of sounds* or *revision*.

Repetition accounted for nearly half, i.e. 43.06%, of all the recorded manifestations. *Pauses* accounted for 26.74% and *sound prolongation* for 20.49%. *Embolophrasia* and *revisions* were encountered less frequently (5.21% and 3.12%, respectively). *Blockage of sounds* appeared least frequently – 1.38% (Table 5).

Based on the statistical analysis, the hierarchical ranking of SD manifestations was established with regard to their frequency. *Repetition* and *pauses* were placed in the first and second positions, respectively. *Sound prolongation* came third, with *embolophrasia* and *revisions* following in positions 4 and 5, respectively. *Blockage of sounds* occupied the sixth position (Table 6).

Table 5. Speech disfluency manifestations in the utterances of the examined patients

Patient	Sound prolongation	Blockage of sounds	Embolophrasia	Pauses	Revisions	Repetition
1	63	0	15	102	6	123
2	75	6	21	69	9	108
3	30	3	9	39	9	60
4	33	12	15	48	6	99
5	24	0	6	30	3	39
6	51	0	3	87	0	168
7	18	0	9	48	0	48
8	66	3	21	90	21	150
9	54	6	9	42	6	90
10	117	6	27	138	21	231
Total	531	36	135	693	81	1116
Mean value	53.1	3.6	13.5	69.3	8.1	111.6
Standard deviation	9.84378	1.316561	2.54951	11.46444	2.496664	19.76979

Table 6. Frequency of speech disfluency manifestations in PD – hierarchical ranking

Manifestation	Sound prolongation	Blockage of sounds	Embolophrasia	Pauses	Revisions	Repetition (sounds, syllables, fragments of phrases)
Scale T1	52.53651	39.85396	42.39047	56.6871	41.0069	67.52499
Hierarchy	0.175122	0.132847	0.141302	0.18895	0.13669	0.225083
	3	6	4	2	5	1

Table 7. Rate of speech of the examined patients

Patient	Speech disfluency intensity	Number of syllables uttered non-fluently	Number of speech disfluency manifestations	Average pace of speech (syl./s)
1	19.8	297	309	1.9
2	18.0	270	288	1.3
3	10.0	150	150	2.3
4	13.0	195	213	2.1
5	6.6	99	102	4.0
6	19.6	294	309	1.2
7	8.2	123	123	3.7
8	21.8	327	351	1.1
9	11.8	177	207	2.3
10	23.0	345	540	0.7

Table 8. Statistical relationship between the pace of speech and the number of syllables uttered non-fluently and the number of speech disfluency manifestations

PD	Correlation coefficient value		
	Very certain relationship	Substantial relationship	Significant relationship
Rate of speech/number of non-fluent syllables	–	–0.85085	–
Rate of speech/number od SD manifestations	–	–0.74087	–

Rate of speech

The rate of speech of the examined patients ranged from 0.7 syl./s to 4.0 syl./s. Only 2 examined patients spoke at a rate regarded as normal, i.e. 3–4 syl./s (Table 7).

As expected, there was a statistically significant negative relationship both between the rate of speech and the number of non-fluently uttered syllables, and the number of SD manifestations. In both cases the relationship was substantial (Table 8).

Discussion

Even though speech disfluency is frequently listed in the clinical picture of PD, knowledge on this phenomenon is scarce. It has not hitherto been established what its intensity, pathomechanism, and symptomatology are, whereas such information seems to be a key condition for effective therapeutic work.

Speech disfluency is a universal phenomenon and as such does not constitute a barrier to communication. Nearly every language user speaks at times in a disfluent manner. When 3–5% of words or syllables are uttered non-fluently, it is perceived as a normal phenomenon. Such an "acceptable" disfluency occurs sporadically and is easy to overcome [12]; however, when its intensity becomes higher, it hinders the process of communication [13]. Speech disfluency intensity recorded in the utterances of patients with PD ranged from 6.6% to 23%, indicating that speech disfluency constitutes a communication barrier in PD.

Speech disfluency is a complex phenomenon. In general, one can distinguish two kinds of SD: organic and functional type. There is a fundamental difference between them. The first is a consequence of the damaged central nervous system (CNS), responsible for the planning and implementation of the verbal utterance. It has a stabilised (consistent) character, with the dynamics and picture connected primarily with the course of the disease [9, 19]. Functional disfluency, on the other hand, is determined primarily by an emotional factor. Therefore it is situationally variable and often transient. This kind of disfluency is typically manifested by *blockages* [7].

Verbal utterances are burdened with communication stress to a varying degree, the largest being characteristic of those requiring direct contact with the interlocutor, those in which there is a change of roles: transmitter – receiver, those in which the contact between interlocutors is a conversational ping-pong match; hence – due to its emotional background – the intensification of the functional SD varies in different types of verbal utterances, being determined by the kind of utterance [14, 15]. The difference between the values of SD intensity in different types of utterance, i.e. *text repetition*, *reading*, *picture story telling*, *guided conversation*, and *free conversation* established in the previous studies was not statistically significant (the highest value being 0.3769). This indicates the organic character of SD in PD.

The organic nature of SD in PD is also indicated by the established hierarchy of the types of utterance with regard to disfluency, not excluding the emotional background. The classification concerning SD starts with *text repetition*, despite the fact that this type of utterance is not particularly difficult communication-wise. An important factor conditioning its fluent performance is the preserved working memory. Numerous authors (J.L. Cummings, L.S Forno, E. Osiejuk-Łojek, J. Sławek, D. Wieczorek), on the other hand, point out the presence of selective deficits of cognitive functions – including memory – in idiopathic parkinsonism [16–21]. Thus, it can be assumed that it is the reduction of the working memory that is responsible for the intensity of SD in this kind of utterance. The reduced concentration of attention present in PD, as pointed out by the above-mentioned authors, should be regarded as an additional factor. In addition, the high degree of speech disfluency observed in an attempt at *text repetition* could be attributed to the deficits in the plasticity of behaviour, which are also manifested as disorders while recreating verbal stimuli [18, 19]. The preserved working memory and the concentration of attention are also necessary for the performance of the test: *guided conversation* (in the second position in the hierarchical ranking), this type of utterance; however, is accompanied by communication stress, so the high degree of SD may have an emotional background as well.

The speech disfluency in PD is connected with the damage to the CNS and, consequently, cognitive, linguistic, and motor deficits, as indicated by its symp-

tomatology. In the hierarchical ranking of the speech disfluency manifestations, *repetition* definitely took the first position frequency-wise (0.225083). Words or fragments of phrases, and less frequently syllables and sounds, were the most frequently repeated elements. The presence of the above-mentioned symptom can be related to the problem that patients with PD have with planning the utterance and with the deficits in the plasticity of behaviour, manifested, for example, in perseverations [20]. The disorder of cerebral inhibition process should also be regarded as a significant aetiological factor, resulting from *frontal-subcortical deafferentation, as confirmed by neurophysiological tests, indicating a reduced level of dopamine in the prefrontal areas of the brain* [22]. *Repetition of sounds*, in turn, could be the reflection of tremor in speech [9, 10]. The next place on the list of manifestations was taken by *pauses* (0.18895). They stemmed primarily from the dysfunction of memory processes – both the short-term and the long-term ones.

This symptom was also generated by the difficulty in performance – resulting from the increased muscle tension and stiffness [17, 23]. *Pauses* at the beginning of an utterance can be explained by the problems that the patients have with movement initialisation; in the middle of the utterance this problem could have been the reflection of the so-called freezing manifested in speech [17]. *Pauses* should also undoubtedly be attributed to the states of depressed mood and pessimism of persons with PD [24]. Similarly to *repetitions* and *pauses*, *embolophrasia* (0.141302) demonstrated difficulty in planning the utterance and the memory dysfunction; they gave the examined patients time to gather their thoughts and think. *Revisions* represented a small percentage of the total number of symptoms (0.13669). As in the average speaker, they stemmed from syntactic disfluencies [7]. The last position is occupied by *blockages* (0.132847), which are a dominant symptom in the case of functional disfluency.

Rate of speech is an integral part of speech fluency. The transmitter speaks fluently if he/she utters approximately 120 words per minute and maintains a regular pace of speech [25]. The rate of speech of the examined patients ranged from 0.7 syl/s to 4.0 syl./s. Only 2 patients spoke at a rate considered as correct. Such a slow rate of speech resulted both from the intensity of SD and from the number of demonstrations [26]. In both cases a statistically significant negative relationship was observed; it was a substantial relationship.

Conclusions

Speech disfluency in PD has an essentially organic background with cognitive, linguistic, and motor deficits resulting from the damaged CNS being responsible for its intensity and symptomatology. Even though the effect of the emotional factor on the picture of SD in PD cannot be entirely excluded, it is of marginal character. Speech disfluency in PD constitutes a communication barrier: for the communication to be successful it is not enough to speak correctly and logically, it is equally important that the utterances are fluent and swift.

Conflict of interest

The authors declare no conflict of interest.

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