

Gdańsk, 15 November 2014

## SUMMARY OF PROFESSIONAL ACCOMPLISHMENT

### **Basic information:**

Dariusz Wieczorek PhD  
clinical psychology specialist  
position: senior academic  
e-mail: wieczore@gumed.edu.pl

Home address: ul.Pegaza 16, 80-299 Gdańsk, Poland

Workplace address:

Department and Clinic of Rehabilitation, GUMed  
Al. Zwycięstwa 30, 80-219 Gdańsk, Poland

5.11.1982      Masters in Psychology, specialization: clinical psychology (University of Gdańsk, Faculty of Humanities)

### **Professional experience:**

20.11.1982      Wojewódzki Zespół Rehabilitacyjny w Gdańsku (Regional Rehabilitation Unit in Gdańsk) – psychologist  
01.05.1983      Państwowy Szpital Kliniczny nr 1 w Gdańsku, Katedra i Klinika Rehabilitacji GUMed (State Hospital no. 1 in Gdańsk, Department and Clinic of Rehabilitation, GUMed) – psychologist  
22.11.1988      First-degree specialization in clinical psychology (under the supervision of prof. Marta Bogdanowicz)  
18.05.1993      Second-degree specialization in clinical psychology (under the supervision of prof. Gabriela Chojnacka-Szawłowska)  
01.04.1991      Medical Academy in Gdańsk, Department of Rehabilitation, employed as Assistant Lecturer; from 01.11.97 as Assistant Professor, from 01.07.2013 as Senior Academic

### **Main scientific achievement:**

Monograph: " The analysis of selected learning, memory, and executive functions in patients with idiopathic Parkinson's disease." *Annales Academiae Medicae Gedanensis*, VOLUME XLIV, suppl. 17, 2013

## Scientific development

### Summary of main directions of scientific work before the doctorate:

The works conducted before the doctorate focused on child clinical neuropsychology, and then on adult clinical neuropsychology. The research on the consequences of open-heart surgeries (no. 1.13 in the list of publications) explained why the studies assessing the influence of intraoperative factors (perfusion time and aortic cross-clamp time) on the later development of the child yield sometimes paradoxical results of better intellectual development in children with longer perfusion time and operation time. The works resulted in the explanation of this paradox. Longer time of the surgery was caused by the necessity to remove a defect having more profound influence on the cardiopulmonary system. The removal of such a defect resulted in greater improvement of the functioning of the circulatory and respiratory systems, which enabled quicker intellectual development after the surgery.

The study on cerebral palsy in children (no. 1.15 in the list of publications) resulted in the identification of cognitive functioning indicators, on the basis of which clinicians can understand the causes of reading and writing disorders in these children.

Later, due to changing profile of patients in the Department and more and more frequent consultations in the Neurology Clinic of Medical Academy of Gdańsk (currently Medical University of Gdańsk), I dedicated myself to differential diagnosis problems in the field of neurology. In the paper published in *Psychiatria Polska* (no. 1.18), I discussed different mechanisms of complaining about memory disorders in the groups of patients diagnosed with depression and patients diagnosed with memory disorders related to diseases affecting the CNS. In the paper, I have shown that each of the groups refers in their complaints to other symptoms of memory disorders diagnosed in the neuropsychological examination.

In this period, I also focused my attention on the problems related with MMPI questionnaire (most popular multidimensional personality test), which, when used in the examination of a person with an undiagnosed neurological disease, may involve a risk of wrong diagnosis of functional impairment. In the PhD thesis defended on 23.05.1997, entitled "Differential diagnosis of disturbances of organic and functional origin in the light of studies involving the MMPI questionnaire" (Gdańsk: University of Gdańsk, Faculty of social Sciences, Department of Psychology, 1997), I have analyzed the causes of MMPI results with a profile indicating functional impairment in patients with diffuse cerebral injuries. I have

indicated the solution of the problem, proposing a different calculation of MMPI results without items identified as referring to neurological symptoms and with the use of a coefficient compensating the loss of these items in raw results in each scale, if there is a suspicion of a CNS disorder.

The contribution in other papers is described in the enclosed list of publications. Here, I have listed those works, in which I had substantial impact on the research concept, the research itself and text preparation.

### **Post-doctorate scientific work**

In relation to my clinical work in Department and Institute of Rehabilitation, involving mainly therapy of the patients with focal brain injuries, I became interested in neuropsychological mechanisms of hemispatial neglect (also known as hemi-inattention or unilateral visual inattention). It is a group of disorders of diversified mechanisms, often wrongly treated as inseparable syndrome, which was reflected in the diagnostic method used then in clinical practice.

At the beginnings of the research, the methods applied in Poland were usually "paper and pencil" methods, such as finding objects from among distractors and finding the centre of objects, supplemented with copying designs and drawing from memory tests. One of the methods most quoted in literature was Behavioral Inattention Test – a method of examination which initially contained tasks involving finding objects from among distractors, copying designs and drawing from imagination, as well as finding the centre of the objects. The tasks used in it were partly redundant, engaging very similar cognitive mechanisms (*Halligan, P.W., Cockburn, J., Wilson, B. A. /1991/. The behavioral assessment of visual neglect. Neuropsychological Rehabilitation, 1, 5-32*). Later, the method was developed to incorporate the growing knowledge about the complexity of hemispatial neglect mechanisms, mainly by adding tasks which involve the patient's interaction with typical objects. The test did not contain tasks related to personal space (the patient's own body).

During the therapy of patients with focal brain injuries, the disorders related to the representation of the patient's own body on the side affected by paresis have detrimental impact on the return of the patient's motor function, as they limit the possibilities of cooperation with the physiotherapist. That is why it is important to diagnose them early, especially on rehabilitation wards. This aspect can be examined with another, rarely used scale (*Zoccolotti P., Antonucci G. i Judica A. /1992/. Psychometric characteristics of two semi-structured scales for the functional evaluation of hemi-inattention in extrapersonal and*

*personal space. Neuropsychological Rehabilitation, 2, 179-191*), which was, however, difficult to apply in clinical practice.

In the paper no. 1.21 in the publication list (*Wieczorek D., Jodzio K.: Proposal of a comprehensive diagnostic evaluation of symptoms of hemispacial neglect in patients with brain damage. Stud. Psychol., 2002, vol. 40, s. 151-171.*), it has been proposed to merge the ideas from both methods and develop them by adding new tasks, in which the assessment of hemispacial neglect is conducted in a manner joining psychometric and clinical-experimental approaches to neuropsychological diagnosis. The psychometric properties of the constructed scale was analysed. The study was conducted with the participation of 39 patients suffering from right-side focal brain injuries. Scale reliability was satisfactory (Cronbach's alpha = 0.81). Similarly to the aforementioned paper by Zoccolotti et al. (2002), two groups of items were identified: symptoms related to personal and extrapersonal space. The scale significantly correlated to the external criterion in the form of standard screening hemispacial neglect assessment methods (crossing out letters and line crossing). The strengths and weaknesses of the scale in clinical practice was also discussed.

Similarly to some previous works, the research was conducted in cooperation with prof. Krzysztof Jodzio, and the cooperation resulted in another publication.

One of the most frequent symptoms of hemispacial neglect encountered by neuropsychologists conducting symptomatic therapy for patients with this disorder is "neglect dyslexia". The dominating type of reading errors is omitting the words on the left side of the text and paralexias within words (usually substituting, omitting or adding sounds at the beginning of the word). The patient does not process information at the beginning of the word and recognizes a shorter word ("kin" instead of "skin"), changes the beginning ("change" instead of "range") or adds an additional element ("flight" instead of "light"). The fact that the wrong recognition almost always results in reading out a work **existing** in a language suggested that visual word representations are intact, and the problem is related to input perception mechanisms.

On the other hand, it has been known that there are forms of hemispacial neglect in which the main deficit affects the recognition of left side of the space, or objects in space (here regardless of their location). These forms of hemispacial neglect cannot be explained with input perception mechanism disorders. Therefore, if the disorder may affect the representation of objects, a question was raised in what scope it affects visual word representations. It could be expected that people with left-side neglect affecting the

representation of objects will have problems with the recognition of known word patterns in the text – contrary to the majority of people with neglect dyslexia, who misread the words but recognize the visual material as words existing in a given language. The answer to this question is presented in the paper no. **1.23** in the list of publications (*Wieczorek D., Jodzio K.: Reading as a process of word reconstruction: a short review and empirical evidence from neglect dyslexia. Acta Neuropsychol., 2004, vol. 2, nr 2, s. 162-175*).

In the study, the results of two groups of patients with hemispatial neglect were analyzed. The first group was characterized by a deficit in left-side spatial representation of objects (it was measured by a test involving drawing a clock from memory), the second one experienced the symptoms of hemispatial neglect, but had no problems with the representation of the left side of the clock. The groups did not differ in the intensification of other hemispatial neglect symptoms.

It turned out that the patients with hemispatial neglect visible in object representation tend to create more neologisms while reading, reading out nonsense letter sequences instead of Polish words. At the same time, the groups did not differ as far as text errors (word omissions) are concerned. The intensity of hemispatial neglect in both group was comparable. It was concluded that the problem consists in distorted left-side component of visual word representations in this patients, which makes it more difficult to reconstruct the stimulating material on the basis of known representations acquired in the process of learning to read. The results were a new, indirect evidence for the assumption that reading is not only simple perception of the stimuli, but, to a great extent, also their reconstruction.

In this period I participated in some interdisciplinary studies in other clinics (e.g. item 1.20).

In the years 2002-2007, I cooperated with Extrapiramical System Disease Clinic in Gdańsk, there I examined patients with Parkinson's disease, dementia with Lewy bodies and progressive supranuclear palsy. The papers which were created during this cooperation combined neurological knowledge on extrapyramidal system disorders, neuroimaging and neuropsychology and were prepared by a team of people specialized in these three branches of knowledge. I cooperated here as a co-author, neuropsychologist, with a specialist in neurology and neuroimaging, and was responsible for the following tasks: developing neuropsychological examination methods, presenting the study methods of cognitive functioning in the paper, analyzing the relationships between the neuropsychological and neuroimaging examination and describing them in the paper, interpreting the results of neuropsychological examinations, participating in the formulation of conclusions, if the

results of the neuropsychological examinations were important in their formulation. The papers were published in journals with **IF 1.28**: (*item 1.1 in the list of publications, 3rd author*), published as a letter to editor: **IF 3.2** (*item 10.4, 2nd author*), as well as: *item 2.4* (3rd author), *item.1.27* (2nd author) and *item 1.25* (3rd author). For the contribution to the aforementioned works, in 2007 I was awarded with II degree Team Scientific Award granted by the Rector of Medical Academy of Gdańsk (currently Medical University of Gdańsk) for research into "cognitive and psychical disorders in chosen extrapyramidal system disorders".

In one of these papers (*item 1.1, see information in the list of publications*), we have managed to show that patients with Parkinson's disease who exhibit dementia in neuropsychological tests have lower cerebral perfusion in the parietal-temporal areas of the left hemisphere, visible in SPECT imaging, than patients who do not suffer from dementia. Hypoperfusion of the left temporal area and increased perfusion of the thalamus has turned out to be an indicator observable in SPECT, which may suggest dementia in the course of Parkinson's disease.

In this period, I conducted also strictly neuropsychological research devoted to praxial disorders in idiopathic Parkinson's disease (PD). In the previous research into this problem, the most popular theoretical frameworks were various modifications of the ideas related to Liepmann's proposal, reconstructed and presented as a new theoretical system by Heilman (*Heilman K.M. Apraxia. W: K.M.Heilman i E.Valenstein (red:) Apraxia 1985, Oxford University Press, New York, 1985 s.131-150*), and operationalized in the paper by Rohi and Heimann (*Rothi, L.J. & Heilman, K.M. Apraxia: the neuropsychology of action. 1997: Psychology, Press, Hove, 1997*). Some of this research indicated the presence of ideomotor apraxia in almost one third of the patients with PD (*Leiguarda RC, Pramstaller PP, Merello M, Strakstein S, Lees AJ, Marsden CD. Apraxia in Parkinson's disease, progressive supranuclear palsy, multiple system atrophy and neuroleptic-induced parkinsonism. Brain 1997; 120: 75-90*). The presence of praxial disorders in PD was sparsely confirmed in some other papers, but remained contrary to clinical experience. My own research was aimed at explaining possible mechanisms of apraxia in PD.

In the paper listed under the number 1.30 (*Wieczorek D., Sławek J., Białkowska M., Dziadkiewicz A., Sitek E.: Sequence learning and multi-step activity impairment in Parkinson's disease. Acta Neuropsychol., 2011, vol. 9, nr 3, s. 303-311.*), the presence of praxial disorders in PD has been analyzed. The study was conducted with indicators related to Luria's classification *Luria AR. Higher cortical functions in man. New York: Basic Books, 1966*). The problems of patients with PD were revealed in the multi-stage task and in the field

of oral praxia, however, the influence of bradykinesia and other motor disorders on the results could not be excluded in this case. It was also shown that patients with PD (in comparison with control subject) have problems consisting in higher number of mistakes in a task in which patients had to learn a sequence of moves corresponding to conflicting motor programs. The mistakes could not be explained exclusively by motor slowness. In the interpretation of the results, an influence of procedural memory disorders on complex motor activities of PD patients was revealed.

Furthermore, I cooperated (usually participating in the development of paper concept and examination methodology, and consultation of the results analysis) with the team of prof. Jarosław Sławek conducting research into the self-awareness of the symptoms in extrapyramidal diseases, on several papers published in journals with IF: item 1.4; (IF=1.368, **3rd author**), item 1.6; (IF=1.518, **3rd author**), item 1.7; (IF=2.759, **3rd author**). The participation in these works was a continuation of my interest in the problem of the self-awareness of the nature of the symptoms in patients with CNS-related diseases, which was initiated in some motifs of my doctoral dissertation and research conducted simultaneously with the doctoral research (cf. the aforementioned item 1.15 in the list of publications). Currently I am preparing one more paper related to this topic.

The works have revealed, among others, lower awareness of chorea in patients with Huntington's disease in comparison to the awareness of dyskinesia in patients with Parkinson's disease in spite of comparative cognitive condition. For my contribution to these studies I was again awarded with II degree Team Scientific Award of the Rector of the Medical University of Gdańsk.

I had also some influence on the methodology of spatial functions examination in some papers concerning the first Polish family with FTDP-17 gene mutation, which made use of my experiences from the research on hemispatial neglect and earlier research into personality, conducted for the doctoral dissertation, item 10.3 of the list (IF=1.559), item 2.6, item 1.5 (IF=3.692). For my contribution to these studies I was awarded with II degree Team Scientific Award of the Rector of the Medical University of Gdańsk for the third time.

One of the recently published works (item 1.33 in the list of publications), *Memory impairment in Dementia with Lewy Bodies relative Alzheimer's Disease with Dementia, Acta Neuropsychologica, vol.11, 3, 2013 s.289-297*, is devoted to the search for differences in clinical image of dementia in Alzheimer's disease (AD), dementia with Lewy bodies (DLB) and dementia in Parkinson's disease (PDD). The groups were equalized in the field of depth of

global cognitive functioning disorders. The PDD group was characterized by better maintenance of semantic memory resources acquired before the onset of the disease in comparison to the AD group. In the AD group, rate of forgetting was higher than in the PDD and DLB groups, however the patients from this group were better in tasks assessing the speed of simple motor activities. No significant differences in the studied cognitive activities have been detected between the PDD and DLB groups. However, the groups were differentiated by much quicker advancement of dementia in patients with DLB in comparison to the patients with PDD.

Among the overview works, one which may be beneficial to clinical practice is recently published paper (item 3.7 in the list) "Mild disturbances of cognitive functions and dementia in Parkinson's disease" (*Przegląd Neurologiczny* 2013, volume 9, no 3 pp.96-104). It is an attempt wider dissemination of knowledge about current criteria for diagnosis of the diagnostic categories listed in the title among the clinicians. It seems that the publication may contribute to unification of MCI and PDD in patients with PD in various treatment centres.

**While conducting the aforementioned studies, I worked also on the monograph, which I would like to indicate as my main scientific accomplishment.**

**It is a habilitation dissertation published as a supplement to "Annales Academiae Medicae Gedanensis" VOLUME XLIV, supplement 17, 2013, entitled: "The analysis of selected learning, memory, and executive functions in patients with idiopathic Parkinson's disease."** It contains results (not published earlier) of research into cognitive functions in Parkinson's disease, correlated to the neurological condition of the patients and cerebral perfusion data (relative perfusion assessed with the use of SPECT).

Learning and memory processes, with executive function involvement, play a crucial role in situations in which the individual has to adapt to new conditions. These cognitive processes are impaired in Parkinson's disease (PD) - in some patients even at the time of diagnosis. Of note, being affected with PD, which progressively impairs motor function, leads to a complex of new and changeable conditions. This, in turn, implicates being faced with new and challenging tasks.

Research on learning and memory processes in PD is quite extensive. However, there are still some controversial issues.



(1) It is debatable if learning impairment, verbal learning deficit in particular, is specific, or if the observed deterioration of learning is secondary to executive function impairment.

(2) The patients' problems in learning visuospatial material have not been fully explained. Assessment of visuospatial learning in PD is often biased by the use of methods with significant motor component. The motor symptomatology of Parkinson's disease may lead to interpretative confusion. Moreover, executive impairment and / or deficits in processing visuospatial material itself may also contribute to impaired visuospatial learning.

(3) Previous studies on procedural learning rely mainly on speed and reaction time, which negatively affects their validity. In most studies learning of motor procedures has been analysed, whereas very few studies have focused on cognitive procedural learning. In those few studies indexes of procedural learning were partially dependent on the executive component associated with planning. Indirect data may suggest a possible dissociation in PD: more impaired motor procedural learning than cognitive procedural learning.

(4) Studies that demonstrate the impaired access to semantic memory resources, are usually biased by executive factor. Verbal fluency is deficient in PD. However, it remains unclear, whether there is a true semantic impairment, or the self-generated strategies of searching through these resources are predominantly affected.

It was assumed that in order to illustrate the clear-cut association of cognitive impairment with Parkinson's disease the demonstration of differences between the patient groups and healthy individuals is not sufficient. Apart from that, it is crucial to show the association of cognitive functions with neurological measures of disease severity and to demonstrate the worsening of a given cognitive function in the course of PD in association with progression of abnormalities within central nervous system (CNS).

Cognitive function was assessed in 58 patients with idiopathic Parkinson's disease: average age 68.75 years (SD=7.08; min.=51, max.=81), average disease duration 8.78 years (2-27), H-Y score – median=2.5 (1.5-5), years of education 11.28 (4-20). The patients' performance was compared against 36 healthy controls matched in terms of age and education. The average age of the controls equaled 67.61 years (SD=8.17, 52-80), the mean duration of education was 12.33 years (4-23). Follow-up neuropsychological examination after three years was performed in 24 patients. In 21 individuals follow-up neuroimaging was also available. The average age at follow-up was 72.92 (8.91), average disease duration equaled 9.96 (SD=3.53, 5-17) and median H-Y stage 3 (2-4).

Indices of analysed variables were chosen so as to exclude the influence of performance speed and motor slowing on the results. None of the learning tasks had time constraints or was dependent on performance speed.

Only performance errors were analysed. Similar approach was used in the selection of other indices. Cognitive assessment addressed: verbal learning (AVLT with recognition and delayed recall trials), visuospatial learning (DUM/ DCS test with additional recognition and delayed recall trials), motor procedural learning (modification of “fist-edge-palm” task), cognitive procedural learning (recall of cognitive procedure learned during *Tower of Toronto* performance after 90 min.) and semantic memory. Semantic memory resources were assessed with the use of Vocabulary and Information subtests from Wechsler Adult Intelligence Scale-Revised / Polish edition (WAIS-R/ PL), while the ability to generate search and retrieval strategies was assessed through semantic and phonemic fluency trials with minimal time constraints. Additional variables, closely related to the main variables, were also analysed: planning performance (*Tower of Toronto*- ToT-number of moves), cognitive control- number of rule violations), ability to initiate and sustain alternating or conflicting motor programs (rhythm sequences, multiple loops drawing, Luria’s alternating graphomotor design), bradykinesia (Finger Tapping Test- the only timed measure used to assess the performance speed), mood (Beck Depression Inventory, BDI) and global cognitive function (Mini-Mental State Examination, MMSE).

Additionally, in order to control the impact of disease severity neurological assessment results were also used: Unified Parkinson’s Disease Rating Scale – part II (UPDRS II), UPDRS-III, UPDRS-IV, H-Y Scale and Schwab-England Scale (S-E).

Brain function was assessed through perfusion assessment in 64 regions of interests and perfusion within larger areas (frontal, parietal, temporal, occipital and perirolandic) with  $^{99m}\text{Tc}$ -ECD SPECT.

Analysis of verbal learning performance indices, the forgetting index (controlling for executive function impact) in particular, demonstrated the greater loss of previously learned material in PD than in healthy controls. It was evidenced that memory impairment is specific and cannot be attributed to executive dysfunction. Verbal learning performance was associated with disease / symptom severity ratings (UPDRS, H-Y, S-E) as well as left temporal and left parietal perfusion. However, the follow-up examination after 3 years has not shown significant deterioration of verbal learning.

PD patients scored lower than the controls on visuospatial learning task. However, the difference was not significant when forgetting rates were considered (rates with minimal

contribution of executive functions). Additional analyses demonstrated the association of visuospatial learning with procedural memory and executive functions. Visuospatial learning performance was related to left parietal perfusion. Moreover, visuospatial learning rates were also associated with neurological measures of disease and symptom severity. Some of them showed decrease at 3 year follow-up.

Semantic memory performance was associated with premorbid education level (years of education) and global cognitive status (MMSE). However, it did not differentiate PD patients from the controls. It was also unrelated to symptom severity and remained stable at 3-year follow-up. Additional analyses demonstrated that vast cognitive reserve (defined as high score on semantic memory measures) was associated with lower vulnerability to depressed mood that is often related to the progression of motor symptoms.

Motor procedural learning impairment was demonstrated with the use of the untimed task in which only performance errors were analysed and performance speed did not influence the scoring. Moreover, cognitive procedural learning deficit was shown with the use of a measure controlling for executive impairment. There was no dissociation between motor and cognitive procedural learning. Motor procedural learning was associated with symptom severity and deteriorated at 3-year follow-up.

Additional analyses demonstrated impairment of cognitive control at 3-year follow-up, which manifested through impulsive and inadequate reactions, when intentional self-corrections of ineffective reactions was needed (rule violations in ToT).

There were significant differences between the baseline and follow-up brain perfusion assessment. Increased hypoperfusion was noted mainly in the parietal and occipital lobes bilaterally and to a lesser extent in left prefrontal dorsolateral cortex.

Furthermore, the obtained results were interpreted with reference to main components of Supervisory Attentional System model by Norman and Shallice and procedural learning model by Fitts and Anderson, which implies declarative phase of this type of learning. The obtained results may be interpreted as deficient processes enabling modifications of behaviour schemes. Research has shown that PD patients may experience difficulties at each stage of behaviour scheme modification.

(1) Firstly, intentional correction of automatic, albeit ineffective reaction, is impaired, which is supported by deficient scores on cognitive control measures. When routine behaviour modification is required, the patients are less efficient in using intentional control mechanisms. In other words, instead of generating more effective behaviour scheme, they are prone to impulsive reactions.

(2) Secondly, at the stage of subsequent modification of activity scheme, when intentional mechanisms predominate and learning is based on declarative memory mechanisms, there may also be some difficulties, as mechanisms of declarative learning are less effective in PD patients, independent of the modality.

(3) Thirdly, at the stage when automatization of previously developed solutions (thanks to declarative learning mechanisms) is needed, PD patients are also impaired, which was evidenced by deficient cognitive and motor procedural learning scores.

The results of 3-year follow-up suggest that in PD patients, especially those with long disease duration, the cognitive profile is characterized mainly by progressive executive dysfunction per se and progressive impairment of cognitive functions in which the executive component is crucial, such as learning and searching through semantic memory resources.

Those deficits are associated with cerebral perfusion deficits. In contrary to the most popular assumption, hypoperfusion is mostly pronounced in posterior areas: occipital and parietal. This pattern of results may be interpreted with reference to a potential breakdown in the compensatory mechanisms created in the disease course.

### **Summary of scientific activity and goals for further work**

At present I am the author or co-author of papers which together (including the papers included in the list of publications) have 466.75 points of the State Committee for Scientific Research, and total impact factor of 23.649. They were mainly interdisciplinary works in which I cooperated with specialists in medicine dedicated to neurology and neuroimaging. Number of citations: 100 according to Web of Science database and 135 according to Scopus. H index of these papers is 6 and 7 (according to Web of Science and Scopus, respectively, as of 20.09.2014). My contribution in the works was sufficient for being awarded three times with II degree Team Scientific Award of the Rector of the Medical University of Gdańsk (in 2007 and 2012-twice). I am the author of one scientific monograph.

General aims always present in my scientific work almost always included two elements: (1) usefulness for clinical practice, (2) using clinical neuropsychology as a science supporting medical sciences.

My scientific interests concerned two groups of topics: hemispatial neglect and extrapyramidal system disorders. and I intend to continue my pursuits in these fields. As for the first area, I am currently preparing research aimed at checking the efficiency of the

proposed method of reading therapy in hemispatial neglect. As far as extrapyramidal system disorders are concerned, I intend to devote more interest to probabilistic learning in patients with PD, relationships between the efficiency of this type of learning with the use of positive and negative reinforcements and dopaminergic therapy, as well as long-term and deferred influence of these phenomena on the patients' personality traits, their relationship with the tendencies to avoid novel stimuli or impulse control problems. Moreover, I participate in the preparations of interdisciplinary papers on progressive supranuclear palsy (PSP) aimed at assessing the differences between the profiles of cognitive disorders in PSP and PD.

### **Didactic activity**

The main place of my teaching activity has always been the Medical University of Gdańsk, where I have conducted classes with the students of medicine, dentistry, physiotherapy, nursing, as well as doctoral courses, above all in the fields of clinical neuropsychology, clinical psychology and methodology of scientific research. I have also conducted clinical neuropsychology classes at the University of Gdańsk (lectures in neuropsychology at the doctor studies of the Faculty of Social Sciences in the academic years 1999/2000 and 2000/2001) and at the Higher School of Social Sciences (SWPS, 2007-2013). I conduct clinical neuropsychology classes on post-graduate studies "Clinical psychology" organized at GUMed and at Studies on Applied Behavioral Analysis (at SWPS, since 2012). This year, I have also cooperated with the Medical University of Warsaw, conducting lectures on neuropsychology for Clinical Psychology Vocational School. Apart from the academic world, I cooperate with Psychological Counseling Centre for Children, Adults and Families in Gdańsk, preparing and conducting courses on clinical neuropsychology. In my didactic activity, I have been the supervisor of four MA theses and seven BA theses. Furthermore, I participate in postgraduate education, supervising specializations in clinical psychology (one finished, four in progress). I am also the supervisor of specialization trainings in neuropsychology.

### **Clinical work**

From the beginning of my career, I have cooperated with Department and Clinic of Rehabilitation at GUMed, where I diagnose and treat patients with CNS-related diseases. Clinical practice is a very important component of my professional activity, and I try to organize my scientific activity around the problems resulting from the practice. I have performed neuropsychological consultations in almost all units and clinics of the Medical University of Gdańsk. In diagnostically difficult cases, I conduct also neuropsychological

examinations as a court expert. I have passed the exams for first and second degree of specialization in clinical psychology. I have the access to one of the biggest databases of cognitive function examination methods in the region, established for neuropsychological diagnosis purposes. I constantly improve my skills related to the therapy of cognitive function disorders.

*Dariusz Wieworek*